Unstable or Insufficiently Activated Internal Models and Feedback-Biased Motor Control as Sources of Dysfluency: A Theoretical Model of Stuttering

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E ven 2,500 years after the earliest written references to stuttering, the basic mechanisms underlying this disorder of speech fluency remain unknown. Admittedly, some of the original (i.e., before the 20th century) attempts at understanding the disorder were rather naïve, and treatments based on those

ABSTRACT: This article presents a theoretical perspective on stuttering based on numerous findings regarding speech and nonspeech neuromotor control in individuals who stutter in combination with recent empirical data and theoretical models from the literature on the neuroscience of motor control. Specifically, this perspective on stuttering relies heavily on recent work regarding feedforward and feedback control schemes; the formation, consolidation, and updating of inverse and forward internal models of the motor systems; and cortical, subcortical, and cerebellar activation patterns during speech and nonspeech motor tasks. Against this background, we propose that stuttering may result when producing speech (a) with unstable or insufficiently activated internal models or (b) with a motor strategy that is weighted too much toward afferent feedback control. We discuss how these two hypotheses can account for the specific dysfluencies that form the primary charac-teristics of stuttering, and we suggest that the hypotheses are compatible with several of the phenomena associated with the disorder (e.g., age of onset, fluency-enhancing conditions, treatment effects). For one of the hypotheses, we also describe a computer simulation implemented in the DIVA (directions into velocities of articulators) model—a neural network model of the central control of speech movements.

KEY WORDS: stuttering, sensorimotor control, internal model, feedback, neuroscience

early speculations ranged from Mercurialis' advice against bathing the head of stuttering children and Dieffenbach's tongue surgery to Bacon's more enjoyable recommendation to warm up the tongue with a moderate amount of wine (for reviews of historical positions on the nature and treatment of stuttering during this time period, see Bloodstein, 1993; Jonas, 1976; Rieber & Wollock, 1977; Silverman, 1996; Van Riper, 1970). Since the 1920s, however, a more rigorous and scientific approach has been used to study both clinical and theoretical aspects of stuttering. As a result, the current literature contains numerous descriptions of much improved treatment procedures, a wide variety of theoretical speculations, and empirical data regarding numerous psychological and physiological characteristics of individuals who stutter (for reviews of positions on the nature and treatment of stuttering throughout the 20th century, see Bloodstein, 1995; Boberg & Kully, 1989; Ingham, 1984; Silverman, 1996; Van Riper, 1973). In fact, so many clinicians and researchers have devoted their attention to the problem of stuttering that it has been suggested that more has been written about stuttering than about any other speech disorder (Stromsta, 1986; Van Riper, 1982).

Despite these continued efforts to gain a better understanding of the mechanisms underlying stuttering, the cause(s) of the disorder remain(s) unknown. Depending on the *Zeitgeist*, many theories of stuttering have been either physiological (e.g., Orton, 1927; Travis, 1931; Zimmermann, 1980) or psychological (e.g., Brutten & Shoemaker, 1967; Flanagan, Goldiamond, & Azrin, 1959; Johnson, 1942; Sheehan, 1958; Wischner, 1950) in nature. Primarily since the 1980s, others have suggested that stuttering may result from an interaction of predisposing physiological factors and precipitating environmental factors, or that stuttering may have multiple etiologies (e.g., Bloodstein, 1993; Conture, 2001; Guitar, 1998). However, support for such multifactorial models has been based more on the inability to attribute stuttering to one single cause than on direct evidence for the involvement of multiple factors. Consequently, this topic continues to stimulate lively discussions. Onslow and colleagues have argued that "it is illogical to argue that a disorder with multifactorial symptomatology must have a multifactorial cause" (Onslow, O'Brian, & Harrison, 1997, p. 236) and further that "the phenomenology of stuttering, particularly at onset, is more consistent with a unitary explanation than a multicausal one" (Packman, Onslow, & Attanasio, 1997, p. 92).

At this time, there is insufficient evidence to argue strongly for or against either of these opposing perspectives. Although it is clear that many environmental, psychological–emotional, and linguistic variables may have an influence on the development of stuttering, it does not necessarily follow that these variables play a role in either the distal (related to etiology) or the proximal (related to individual moments of stuttering) cause(s) of stuttering. At any rate, given that the primary characteristics of stuttering consist of repeated or prolonged articulatory and phonatory actions that result in sound and syllable repetitions, audible and inaudible sound prolongations, and broken words, stuttering ultimately presents itself as a disruption of the speech motor system. That is, stuttering is characterized by disruptions of the respiratory, phonatory, and/or articulatory movements—or the coordination of these movements—required for fluent speech production. For this reason, we believe that gaining insight into the mechanisms responsible for the disorder requires workers in this area to achieve an in-depth understanding of the neural processes and sensorimotor mechanisms involved in the control, coordination, and breakdown of speech movements in individuals who stutter. Only with a clearer picture regarding the possible involvement of aberrant motor control processes will there be a strong foundation to develop and test hypotheses as to which other factors (e.g., environmental, psychological-emotional, linguistic) may affect speech production in young children and play a role in the fluency disruptions in the speech of those children who develop a chronic stuttering problem.

In the present work, our goal is to contribute to an understanding of the neural processes and sensorimotor mechanisms involved in stuttering by offering for empirical testing two specific hypotheses about the possible sources of fluency breakdown in individuals who stutter (see also Max, 2004; Max, Gracco, Guenther, Ghosh, & Wallace, in press). Both hypotheses offer suggestions primarily for the proximal sources of stuttered dysfluencies (i.e., sound and syllable repetitions, audible and inaudible sound prolongations, and broken words). Thus, they propose explanations for what causes a moment of stuttering when an individual who stutters is speaking. However, limited preliminary speculations as to some potential distal causes (Why does a given individual have the disorder?) are offered where appropriate.

Specifically, we propose here two hypotheses that are, in our opinion, consistent with the extant literature on stuttering, and that are based on recent insights into the neuroscience of both speech and nonspeech motor control. Information about stuttering that was taken into consideration to develop the hypotheses includes primarily findings regarding stuttering individuals' movement characteristics during both dysfluent and fluent speech as well as during nonspeech tasks and findings regarding conditions that have a fluency-enhancing effect on stuttering individuals' speech. These findings were then interpreted in light of experimental data and theoretical notions regarding a variety of other topics, including (a) control schemes that allow the central nervous system (CNS) to take the multiple and complex central command-to-motor output transformations into account during movement planning, (b) feedforward and feedback neural mechanisms and substrates involved in sensorimotor control and learning, (c) activation of cortical and subcortical brain areas during speech production, and (d) neural network models of motor control, in particular, the modeling of speech movements in the directions into velocities of articulators (DIVA) model (Guenther, 1994; Guenther & Ghosh, 2003). Importantly, development of the hypotheses was strongly constrained by the requirement that the hypotheses be consistent with well-known facts associated with the disorder (e.g., types of dysfluencies, typical age of onset). Briefly, the hypotheses suggest that possible sources for stuttered speech dysfluencies may lie

in (a) unstable or insufficiently activated internal representations of the transformations that occur when central motor commands are converted into the acoustic end product of speech, or (b) a motor control strategy that relies too much on afferent signals that are associated with considerable time lags.

We will first summarize and review the pertinent literature on stuttering and draw from it some important conclusions regarding the level at which the speech mechanism breaks down—regarding the distinction between basic breakdowns on the one hand and compensatory or preferred motor strategies on the other hand. Subsequently, we will discuss selected findings from the movement neuroscience literature, with an emphasis on recent evidence for internal representations of the command-to-output and output-to-command transformations (i.e., internal models) and on the neural substrates involved in various aspects of speech and nonspeech sensorimotor control and learning. Based on this evidence, we will describe a general model of a biologically plausible control scheme in which internal models allow the combined use of feedforward and feedback controllers for the organization of goal-directed movements. Next, this model will serve as the global theoretical context within which we will formulate our hypotheses regarding the sensorimotor mechanisms underlying stuttering. In the subsequent section, we will provide an overview of the DIVA model and discuss computer simulations of our proposed hypotheses implemented in this neural network model of speech movements. Last, we will show that the formulated hypotheses are consistent with several phenomena known to be associated with the disorder.

REVIEW OF SELECTED TOPICS IN THE STUTTERING LITERATURE

In this section, we summarize some data from the stuttering literature that are most important for the hypotheses that we present later in this article. A more detailed review of this literature is available elsewhere (Max, 2004).

Speech Movements

Numerous studies using reaction time paradigms have shown that individuals who stutter, as a group, are slower than nonstuttering individuals in initiating phonation and articulation. In addition, several acoustic studies have shown longer voice onset times (VOTs), stop gap (SG) durations, vowel durations, and consonant–vowel transition durations in people who stutter (PWS) versus people who do not stutter, although these differences were sometimes limited to certain conditions of phonetic context or articulatory complexity (see Bloodstein, 1995, for reviews).

Using kinematic analyses to compare PWS and nonstuttering speakers, Zimmermann (1980) and Caruso, Abbs, and Gracco (1988) found that PWS showed longer movement durations and longer temporal intervals between articulatory and phonatory events than did nonstuttering speakers. Findings reported by McClean, Kroll, and Loftus (1990), however, suggested that such differences may be limited to PWS who had recently received treatment for their speech disorder. We therefore analyzed kinematic articulatory data from nonstuttering individuals and stuttering individuals who had not received treatment for at least 8 years (Max, Caruso, & Gracco, 2003). Between-group differences were found in several measures of lip and jaw closing movements, and showed most consistently longer total movement durations and longer durations from movement onset to peak velocity in the group who stutter.

In further analyses of the same data set, we examined specific aspects of relative timing by determining the order of intra-gestural kinematic events across different articulators (Max, Gracco, & Caruso, in press). It had been shown previously that normally fluent speakers typically organize upper lip (UL), lower lip (LL), and jaw (J) movement onsets and peak velocities associated with bilabial closing for the first /p/ in *sapapple* in the order UL-LL-J (Caruso, Abbs, et al., 1988; De Nil, 1995; Gracco, 1994). It had further been shown by Caruso, Abbs, et al. and De Nil for the same articulators, sounds, and contexts and by Alfonso (1991) for different articulators, sounds, and contexts, that individuals who stutter differed from individuals who do not stutter in the preferred sequencing patterns. However, other studies with either the same or different sounds in a different phonetic context had failed to confirm such an articulatory sequencing difference between PWS and nonstuttering speakers (De Nil, 1995; Jäncke, Kaiser, Bauer, & Kalveram, 1995; McClean et al., 1990). Our own results for /p, b/ in different contexts yielded highly similar sequencing patterns for the two groups (Max, Gracco, & Caruso, in press).

In a different approach to studying speech movement timing, Smith and colleagues used a spatiotemporal index (STI) that reflects variability of LL movement across repeated productions of an utterance. Kleinow and Smith (2000) found that adults who stutter showed overall higher STI values (i.e., more variability) than did adults who do not stutter, but Smith and Kleinow (2000) found no statistically significant STI difference between adults who stutter and nonstuttering adults.

Yet another approach to examining speech timing in stuttering has focused on the coordination of oral and laryngeal movements. Based on physiological examination of a few individual moments of stuttering, Yoshioka and Löfqvist (1981) and Hutchinson and Watkin (1976) suggested that some stuttering moments may be characterized by laryngeal movements that are improperly timed relative to oral or respiratory movements. Borden and Armson (1987) and Caruso, Conture, and Colton (1988), on the other hand, concluded that oral–laryngeal coordination can be appropriate during moments of stuttering. Using physiological measures during perceptually fluent speech, Conture, Colton, and Gleason (1988) found no differences in stuttering versus nonstuttering children's coordination of articulation and phonation. Using acoustic measures during perceptually fluent speech, Boutsen (1995) and Zebrowski, Conture, and Cudahy (1985) reported that, across subjects, nonstuttering individuals showed a negative correlation

between mean SG duration and mean aspiration duration or mean VOT, whereas stuttering individuals showed no correlation or a much smaller negative correlation. In subsequent studies, however, both acoustic and physiological data failed to show such a between-group difference when correlations were computed on a within-subjects basis to reflect adjustments in oral–laryngeal relative timing across productions of the target consonants (Borden, Kim, & Spiegler, 1987; Max & Glass, 2001; Max & Gracco, 2003).

Additional studies of speech motor timing have made use of paradigms in which groups of stuttering and non-stuttering subjects performed rhythmic timing tasks. For self-paced responses, Brown and colleagues (Brown, Zimmermann, Linville, & Hegmann, 1990) found that stuttering individuals' speech movements were associated with decreased timing variability as compared with those of nonstuttering individuals. For responses synchronized with an external stimulus, one study suggested that PWS show increased variability when producing sequences of syllables with different stress patterns (Boutsen, Brutten, & Watts, 2000). However, for responses that were performed first during a synchronization phase and then maintained during a continuation phase, three different studies found stuttering and nonstuttering participants to show similar variability in vowel and syllable production during both synchronization and continuation (Hulstijn, Summers, van Lieshout, & Peters, 1992; Max & Yudman, 2003; Melvin et al., 1995).

A possible involvement of *sensory* systems in stuttering is supported primarily by studies focusing on the fluency-enhancing effects of altered auditory feedback (e.g., Stuart, Kalinowski, & Rastatter, 1997). Little attention has been paid to a possible role for proprioceptive inputs. Nevertheless, in a study by Caruso, Gracco, and Abbs (1987), 3 adults who stutter, as compared with 3 nonstuttering adults, showed longer compensation latencies and smaller articulatory displacements in response to unexpected perturbations applied to the jaw during bilabial closing gestures. Additionally, McClean (1996) found that only 4 of 14 adults who stutter showed the same attenuation of mechanically elicited lip reflexes that nonstuttering adults showed immediately preceding fluently produced monosyllabic words. These two studies suggest that stuttering may be associated with problems in the afferent systems, in the central processing of afferent information, in using such information for updating motor commands, or in the appropriate priming of afferent systems during planning of the motor commands.

Overall, the results summarized above suggest a general slowness, possibly sensory based, in the speech movements of individuals who stutter rather than a difference specifically in the timing of those movements. Although subjects in some of the reviewed studies had not received stuttering treatment for many years before the data collection (e.g., more than 8 years in Max, Caruso, et al., 2003), it cannot be ruled out—based on speech data alone—that the subjects who stutter use slower speech movements because they have been taught or advised to speak slower in order to increase fluency. Furthermore, most of the analyses were completed on perceptually fluent speech, and they do not provide information regarding the possibility that

individuals who stutter may experience specific timing difficulties that occur intermittently during actual moments of stuttering. Nevertheless, it appears that, during fluent speech, the movements of PWS are performed more slowly than those of individuals who do not stutter, but that they are appropriately timed relative to other movements within and across the articulatory, phonatory, and respiratory subsystems. This distinction between slower and mistimed movements may be critically important in light of the question of whether differences between stuttering and nonstuttering individuals during perceptually fluent speech reflect basic aspects of the mechanisms underlying the disorder or a preferred motor strategy that is used to avoid/minimize dysfluencies.

Nonspeech Movements

One possible way to reveal the implications of the documented slowness of stuttering individuals' speech movements may consist of studies analyzing identical or similar movement parameters in these subjects' nonspeech movements. Longer movement durations have been confirmed in both orofacial nonspeech movements and finger movements (Max, Caruso, et al., 2003; Webster, 1997). Additionally, studies of nonspeech movements in stuttering versus nonstuttering individuals have revealed that between-group differences also exist in finger movement accuracy and initiation time (Webster, 1997), manual reaction times (Bishop, Williams, & Cooper, 1991; Webster & Ryan, 1991), bimanual coordination (Forster & Webster, 2001; Zelaznik, Smith, Franz, & Ho, 1997), and possibly lip isometric force generation when visual feedback is present (Grosjean, van Galen, de Jong, van Lieshout, & Hulstijn, 1997). Isometric force generation tasks, however, have also resulted in negative results (Zelaznik, Smith, & Franz, 1994).

Similar to the studies of speech movements, some studies of nonspeech movements have focused specifically on timing variability within trains of isochronous responses. Although one study found increased timing variability in stuttering individuals' self-paced finger tapping (Cooper & Allen, 1977), another study found *decreased* variability in stuttering individuals' orofacial nonspeech movements and finger tapping (Brown et al., 1990). When subjects were required to synchronize their finger tapping with a metronome, stuttering and nonstuttering individuals showed similar levels of variability (Zelaznik et al., 1994). For finger movements performed first during a synchronization phase and then during a continuation phase, three studies found stuttering and nonstuttering adults to show no differences in the variability of either synchronization or continuation responses (Hulstijn et al., 1992; Max & Yudman, 2003; Melvin et al., 1995).

A second type of timing analysis that was initially used with speech movements but recently also applied to nonspeech movements is the analysis of peak velocity sequencing across the different effectors contributing to a single task. For both orofacial nonspeech movements and finger movements, results have shown highly similar sequencing patterns for stuttering and nonstuttering individuals (Max, Caruso, et al., 2003).

Also of interest is that, similar to the situation for speech movements, some evidence suggests that the nonspeech motor difficulties of PWS may have a basis in sensory deficiencies. De Nil and Abbs (1991) observed that, when instructed to make the smallest possible movements in the absence of visual feedback, adults who stutter made larger oral movements than did nonstuttering adults. When visual feedback was added, however, performance of the two groups became similar. Howell, Sackin, and Rustin (1995) obtained similar findings for the lip movements of stuttering versus nonstuttering children. Later, Loucks and De Nil (2001) also found that adults who stutter performed less accurately than nonstuttering adults when making jaw opening movements to visually presented spatial targets. Again, performance of the two groups became similar when visual feedback was added. Based on a bimanual coordination task, Forster and Webster (2001) reported additional data confirming this facilitating effect of visual feedback on the nonspeech motor performance of PWS.

It seems appropriate to conclude from these nonspeech data that differences between stuttering and nonstuttering individuals are not limited to speech movements. Rather, differences in certain movement parameters (in particular, movement initiation latency and movement duration) also exist for the orofacial system when used for nonspeech tasks and in unrelated effector systems such as the fingers and hands. Furthermore, limited evidence is available to suggest that these nonspeech differences may be based on stuttering individuals' difficulties with the processing of proprioceptive sensory information or with the integration of sensory processing and motor planning.

Brain Activation Patterns

Several brain imaging studies have revealed hemispheric lateralization differences between adults who stutter and adults who do not stutter. Adults who stutter typically showed decreased left hemisphere activation or increased right hemisphere activation (Braun et al., 1997; De Nil, Kroll, Kapur, & Houle, 2000; De Nil, Kroll, & Houle, 2001; Fox et al., 1996; Fox et al., 2000; Ingham, 2001; Wu et al., 1995). It is possible, however, that these findings may be of limited value for understanding the mechanisms underlying stuttering. This reasoning is based on observations indicating that it is possible for an increase in right-hemisphere activation, and thus a decrease or reversal of normal left-hemisphere lateralization, in a variety of tasks to occur as a result of the neural plasticity associated with long-term motor experiences (Mikheev, Mohr, Afanasiev, Landis, & Thut, 2002). Most important may be the more specific findings indicating that stuttering is associated with an atypical dopamine metabolism (Maguire, Riley, Franklin, & Gottschalk, 2000; Riley, Maguire, & Wu, 2001; Wu et al., 1995; Wu et al., 1997), hyperactivation of (right hemisphere) motor and premotor cerebral and (left) cerebellar areas (Braun et al., 1997; De Nil et al., 2000; De Nil et al., 2001; Fox et al., 1996; Fox et al., 2000), and absent or reduced activation (or sometimes even deactivation relative to rest conditions) of auditory and possibly other sensory cortical areas (Braun et al., 1997; Fox et al.,

1996; Fox et al., 2000). Findings from the same studies further suggest that during fluent speech induced by chorus reading, both the hyperactivity of the motor areas and the lack of activation or deactivation of auditory areas were reduced or eliminated. With regard to the level at which breakdowns in speech production occur in PWS (i.e., cognitive–linguistic vs. motor), it is worth mentioning that the imaging data of Braun et al. (1997) showed the differences between stuttering and nonstuttering individuals in the activation of motor and sensory cortical areas to be present even when subjects performed nonspeech oral movements. Thus, the latter brain imaging data are consistent with the observation of between-group differences in kinematic movement parameters even when the motor tasks are not of a linguistic nature or are performed with nonspeech motor systems.

Combining the brain imaging data with the above discussed acoustic and physiological speech and nonspeech sensorimotor data, the overall findings provide compelling evidence to suggest that stuttering may result from problems with movement preparation and sensory monitoring or sensorimotor integration. In this context, one implication of the dopamine-related findings may be related to the role of the basal ganglia dopaminergic system in both motor planning/programming (Amabile et al., 1986; Fattapposta et al., 2000; Fattapposta et al., 2002; Mattay et al., 2002; Suri, Bargas, & Arbib, 2001) and sensorimotor integration and learning (Fattapposta et al., 2000; Fattapposta et al., 2002, Huda, Salunga, & Matsunami, 2001; Suri et al., 2001). This perspective is also consistent with De Nil et al.'s (2001) interpretation of their cerebellar activation data as indicating an increased need for sensorimotor monitoring in PWS.

Given that the goal of this article is to develop in more detail specific hypotheses based on the perspective that stuttering is a sensorimotor disorder, we will discuss in the next section selected topics from the current literature on the neuroscience of motor control and summarize recent insights into the neural processes and substrates involved in movement planning and sensory monitoring. Topics have been selected based on their contributing directly to the two hypotheses to be formulated subsequently or their ability to explain how the hypotheses may account for some of the phenomena known to be associated with stuttering.

RECENT INSIGHTS INTO THE NEUROSCIENCE OF MOVEMENT CONTROL

A Global Model of Sensorimotor Control

Figure 1 shows a global model of motor control that is based on currently widely used notions in the literature on the neuroscience of motor control. The model has evolved from efforts to resolve the previously existing dichotomy between feedforward and feedback models of motor control. *Feedforward* models suggest that motor commands are prepared before the onset of movement and then issued to the musculature for execution without further alterations. In these models, feedback has been typically considered to be

Figure 1. Schematic representation of a global model of motor control. The model represents a hybrid control scheme consisting of a feedforward controller and a feedback controller that make use of inverse and forward internal models, respectively.
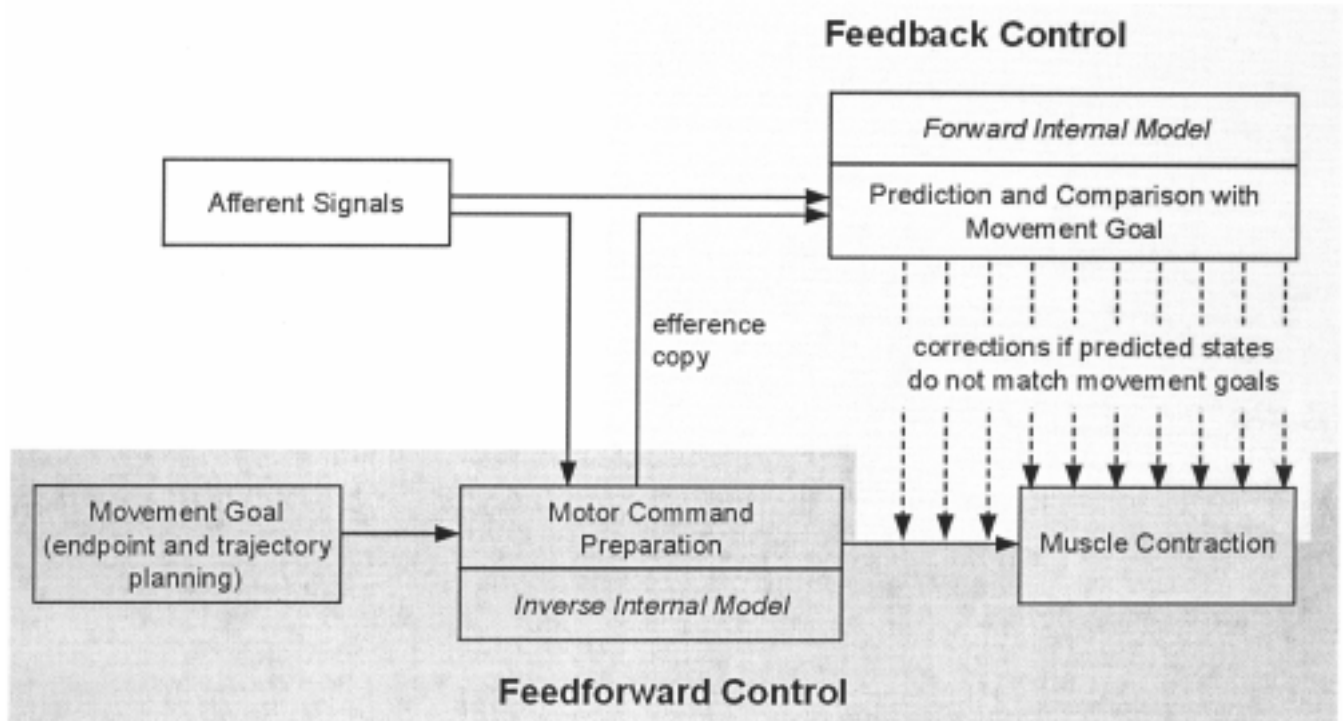
of minimal importance, except maybe for the final stages of the movement. *Feedback* models, on the other hand, argue against the preparation of such a motor plan before movement onset and suggest that motor commands are generated in parallel with the movement on the basis of an error signal resulting from a continuous comparison of sensory information regarding effector (e.g., hand) position and the target position.

Integrative models, like the one represented in Figure 1, suggest a control scheme that combines both a feedforward control system and a feedback control system. A basic motor plan is assembled before movement onset and then executed by a feedforward controller, but these commands can be adjusted in real time by a feedback controller. It is important to emphasize that the feedback controller integrates both afferent (sensory) and efferent signals (Desmurget & Grafton, 2000; Wolpert, Ghahramani, & Jordan, 1995; Wolpert, Miall, & Kawato, 1998). This aspect of the model is crucial because it has often been argued that the contributions of purely afferent feedback are limited due to the involved time lags relative to the events that generated the feedback. In the integrative model, however, this problem is minimized or avoided by using the combination of a copy of the prepared motor commands (efference copy or corollary discharge; Sperry, 1950; von Holst & Mittelstaedt, 1950/1973) and the afferent information available at that time to *predict* the sensory consequences of the planned movement (see below). Thus, the system does not need to wait for afferent inputs signalling

movement errors and required compensations, but it can update or correct motor commands in an anticipatory manner by predicting the movement outcome and then comparing the predicted and desired outcomes. In current formulations of this general model, both controllers depend on the availability of internal inverse and forward representations or models of the relationship between centrally generated motor commands and the sensory consequences of the resulting movements.

The feedforward control system depends on continually updated and accurate inverse internal models of the system characteristics to compute the necessary motor commands that would achieve a planned movement goal given the system's current state (Desmurget & Grafton, 2000; Shadmehr & Holcomb, 1997; Shadmehr & Mussa-Ivaldi, 1994; Wolpert, Ghahramani, & Flanagan, 2001). The inverse transformation from desired movements to the required motor commands is complex due to the time-varying influence of various neural and muscular physiological factors, the current state of the system, and biomechanics. More specifically, two different types of inverse transformations can be distinguished (Atkeson, 1989). An inverse kinematic transformation refers to a conversion from end-effector position (e.g., hand position in space) to the necessary body-based coordinates for all involved segments (e.g., elbow and shoulder joint angles). Inverse dynamic transformations are seen in the conversion from desired body-based coordinates to the necessary muscular forces (the consequences of muscular forces are

influenced by, for example, simultaneously co-contracting muscles, inertia, friction, and gravity) and in the conversion from muscular forces to the necessary motor commands (the consequences of motor commands are influenced by, for example, muscle mechanical properties and the length-force and velocity-force relationships of the muscular system). Both these types of inverse transformations are assumed to be represented in inverse internal models that are used by the feedforward controller. Thus, an inverse internal model can be considered a neural map that inverts the complex input–output relationships such that it allows the controller to compute from the desired sensory consequences the central commands necessary to achieve those consequences. After establishing the desired movement outcome, the feedforward control system accesses the information represented in the inverse internal models to accomplish inverse computations and prepare the motor commands that will be executed to the effector musculature.

The feedback control system, on the other hand, depends on accurate forward internal models. As mentioned, this controller is believed to monitor and correct, if necessary, ongoing movements based on combined afferent and efferent inputs. The efferent component consists of a copy (efference copy) of the prepared motor commands that is used by the feedback system to predict the sensory consequences of the movements resulting from those commands. In order to be able to make such predictions, the controller has access to a forward internal model that can be used to evaluate the efference copy (Bhushan & Shadmehr, 1999; Blakemore et al., 2001; Desmurget & Grafton, 2000; Flanagan & Wing, 1997; Mehta & Schaal, 2002; Wolpert & Miall, 1996; Wolpert et al., 2001). A forward model is a neural map of the input–output relationships, in this case, one that allows the controller to compute in a predictive manner the sensory consequences of the prepared motor commands. It contains detailed information about the system properties that will determine the system's response to a given input of motor commands. The availability of a forward model offers important advantages to the feedback controller. With purely afferent feedback control, the sensory consequences for a system state at time point t_x would only become available at a later time t_{x+DA} (with DA being the delay associated with afferent input). Forward modeling, on the other hand, allows a prediction of the sensory consequences to be made at an earlier time point $t_{x-DF+DE}$ (with DF being the delay associated with the feedforward signal, or in other words, the delay between motor command preparation and muscle contraction such that t_{x-DF} is the time when the commands resulting in the system state at t_x are prepared; and with DE being a delay associated with forward model-based evaluating of an efference copy of the commands). If the predicted consequences differ from the desired and planned movement goal, corrections to the efferent signals can be made early in the movement or possibly (depending on the extent of delay DE) even during command preparation/execution before movement initiation.

This motor control scheme with both a feedforward and a feedback controller and associated inverse and forward

internal models is supported by several lines of research. Some of the older work supporting this model includes data indicating that motor commands are not generated online during the movement (i.e., arguing against pure feedback control), that feedforward control is needed because feedback control by itself would be inefficient due to the delays involved in afferent feedback pathways (i.e., also arguing against pure feedback control), and that feedback-based online corrections do occur even during fast movements (i.e., arguing against pure feedforward control but also against feedback control that is strictly afferent) (see Desmurget & Grafton, 2000, for a succinct overview of this literature). Evidence for the involvement of inverse and forward internal models in such a control scheme with both feedforward and feedback controllers has come primarily from two recent research paradigms.

The first paradigm has been used to investigate how and to what extent movement planning is adjusted in the presence of sensory perturbations that alter the system's input–output relationships. If movements and/or their sensory consequences are experimentally manipulated, the central commands normally generated for the task will result in sensory feedback that differs from the predicted and desired feedback, and different commands will need to be generated to achieve the desired outcome on future trials. Studies have shown that such adjustments do indeed occur, and that, after an initial learning period, subjects are able to plan and perform correct movements even in the presence of experimentally manipulated sensory consequences or movement paths—a phenomenon known as sensorimotor adaptation. In fact, when the feedback perturbation is suddenly removed after the subject has started to adapt, movement errors occur as a result of continued, but now unnecessary, compensation. These errors—known as after effects—confirm that, after the initial learning, the movements had been planned based on knowledge of the altered input–output relationships. In other words, observations of sensorimotor adaptation and after effects suggest that the CNS updates and accesses internal representations of the inverse input–output relationships (i.e., inverse internal models). Evidence of sensorimotor adaptation and after effects is available for various motor systems and tasks. Examples include arm movements to visual targets in the presence of visuomotor rotations (Flament, Ellermann, Kim, Ugurbil, & Ebner, 1996; Klapp, Nordell, Hoekenga, & Patton, 1974; Lackner & Lobovits, 1977), arm movements in force fields (Bhushan & Shadmehr, 1999; Shadmehr & Mussa-Ivaldi, 1994; Thoroughman & Shadmehr, 1999), and articulatory speech movements performed under conditions of formant-shifted auditory feedback (Houde & Jordan, 1998; Max, Wallace, & Vincent, 2003).

The second paradigm has been used to investigate how much and which information is taken into account by the CNS when planning movements and predicting the consequences of those movements. Studies have shown, for example, that subjects appropriately adjust the grip force used to hold an object, or the downward force to slide an object, in parallel with (rather than in reaction to) the self-generated forces that are used to move the object

(Blakemore, Goodbody, & Wolpert, 1998; Flanagan & Lolley, 2001; Flanagan & Wing, 1993, 1997), and that they show appropriate anticipatory shoulder or elbow muscle activity to compensate for the interaction torques resulting from movement only at the other joint (Gribble & Ostry, 1999). These findings demonstrate that the CNS correctly predicts the consequences of its own movements and generates appropriate compensatory responses. Such detailed predictions and high-precision anticipatory or parallel responses have been interpreted as evidence for the availability of internal representations of the forward input–output relationships (i.e., forward internal models).

Also of potential importance when trying to interpret the stuttering literature in the context of this general model of motor control are the underlying neural processes. Recent theoretical and experimental work has suggested primarily the cerebellum (Blakemore et al., 2001; Imamizu et al., 2000; Miall, Weir, Wopert, & Stein, 1993; Wolpert, Miall, & Kawato, 1998) and posterior parietal cortex (Desmurget & Grafton, 2000; Wolpert et al., 1998) as possible sites for the formation of internal models. It also appears that changes occur in the neural activation pattern during the stage of motor memory consolidation, even when motor performance remains unchanged. In particular, there is strong evidence suggesting that cerebellar blood flow initially increases during the early learning stages but then decreases as learning proceeds (Flament et al., 1996; Friston, Frith, Passingham, Liddle, & Frackowiak, 1992; Grafton, Woods, & Tyszka, 1994; Seitz et al., 1994). Other work has suggested that global activation changes in the cerebellum may reflect learning processes, whereas local changes in an area near the posterior superior fissure may reflect the actual formation of an internal model (Imamizu et al., 2000). Thus, it is important to distinguish between movement acquisition and motor memory consolidation or internal model formation. For example, one study investigating sensorimotor adaptation in a monkey showed that even when movements were practiced until no movement errors were present, the effects were only transient, and that additional practice with no errors was necessary to achieve stable consolidation (Yin & Kitazawa, 2001). Such findings may be of relevance when interpreting the literature on reductions in stuttering after short-term practice (e.g., the adaptation effect) or long-term practice (e.g., treatment).

Cortical Activation Patterns During Speech and Nonspeech Tasks

Some of the most essential aspects of the above-described global model of motor control are the existence and use of efference copies of planned motor commands and the integration of these efference signals with afferent signals in order to allow the feedback controller to implement online (non-delayed) motor command updates or corrections. Some illustrations of evidence supporting the notion that the CNS monitors efference copies of its own motor commands are the suppression of self-generated versus external somatosensory stimuli (Blakemore, Wolpert, &

Frith, 1998, 2000), the modulation of external somatosensory inputs before and during self-generated movement (Cohen & Starr, 1987; Nelson, 1996), the attenuation of activity in cortical areas involved in the visual perception of motion when the observed movement is self-generated (Leube et al., 2003), and the activation of somatosensory cortical areas before movement onset (Lin, Murray, & Sessle, 1994; Nelson, 1987).

In our earlier review of the stuttering literature, we pointed out that a number of studies have shown reduced or absent auditory cortex activation during speech production in stuttering versus nonstuttering individuals. This reduced or absent activation could possibly reflect, or even cause, an insufficient evaluation of efference copies with the forward internal models (at least in terms of their *auditory* consequences), if the available data on efference copies in general motor control can be generalized to the motor task of speech production. We therefore briefly review here some of the currently available evidence suggesting that speech production is indeed associated with motor-to-sensory priming such as one would expect if an efference copy is sent for evaluation by the auditory and/or somatosensory cortex. Best documented for speech production is the priming of auditory cortical areas.

Magnetoencephalography (MEG) studies have revealed that the auditory cortex M100 response (a response to auditory stimulation with a latency of approximately 100 ms) is reduced in amplitude during self-production of vowels as opposed to hearing a tape recording of one's own vowel productions (Houde, Nagarajan, Sekihara, & Merzenich, 2002). Furthermore, the M100 response to short tones is reduced in amplitude during reading aloud as compared with reading silently (Numminen, Salmelin, & Hari, 1999) or as compared with hearing a recording of one's own speech (Curio, Neuloh, Numminen, Jousmäki, & Hari, 2000; Houde et al., 2002). In the study by Curio et al., the auditory cortex also did not react with an additional response to a vowel that was infrequently produced during a self-uttered sequence of vowels although an additional response was observed for an infrequent vowel in a sequence played back from a recording. In addition, studies using positron emission tomography (PET; which has a lower temporal resolution, and, thus, reveals activation on a slower time scale) have shown overall activation of auditory cortical areas during speech production (Braun et al., 1997; Fox et al., 1996, 2000). In fact, functional magnetic resonance imaging (fMRI) has shown that even while subjects subvocally named visually presented objects, the dorsal portion of the left posterior superior temporal gyrus was activated (Hickok et al., 2000). When speech was whispered, but auditory feedback was masked, activation of secondary auditory cortex was still observed (Paus, Marrett, Worsley, & Evans, 1996; Paus, Perry, Zatorre, Worsley, & Evans, 1996). Combined, these MEG, PET, and fMRI data demonstrate that producing speech modulates auditory cortex in ways that differ from the activation of the same cortical areas by external auditory stimuli and that at least some of the data are consistent with motor-to-sensory priming during movement planning.

HYPOTHESIZED SOURCES OF STUTTERING

Hypothesis 1: Unstable or Insufficiently Activated Internal Models

As one of two possibilities presented here, we hypothesize that PWS may have, or may have had during childhood, problems with the acquisition and updating, or alternatively with the activation and use, of the inverse and/or forward internal models that are part of an integrative control scheme with both feedforward and feedback controllers. In essence, this hypothesis suggests that an important aspect of the disorder may lie in some children's inability to acquire stable (e.g., not inappropriately updated in response to short-term time-varying aspects of afferent signals) and correct mappings (inverse, forward, or both) between motor commands and sensory consequences, to appropriately update these mappings during speech development, or to sufficiently activate and successfully use these mappings for efficient sensorimotor control of the speech mechanism. Use of such internal models may be particularly critical for speech production given that this task involves not only the kinematic and dynamic transformations involved in limb movements, but also additional transformations from vocal tract configurations to acoustic output. Thus, the CNS would need additional or more elaborate forward and inverse representations of the input–output relationships, in particular with regard to the acoustic output. Moreover, the rapid neural and craniofacial developmental changes during childhood require that the internal representations of each of the transformations be updated in parallel. It has been well documented that dramatic anatomical changes take place in the vocal tract during development (e.g., Kent & Vorperian, 1995). As a result, children's motor systems face the challenging task of acquiring and updating multiple internal models for a continually changing neuromotor system. If, for some currently unknown but possibly neuroanatomical or neurochemical reason, the CNS would fail to accurately update or sufficiently activate the internal models, it would become impossible for the feedforward controller to correctly derive the necessary commands for a desired sensory outcome or for the feedback controller to predict with great precision the sensory consequences of planned motor commands.

Based on the integrative model shown in Figure 1, problems with the inverse models would result in inaccurate computations of the feedforward commands. If incorrectly prepared motor commands are executed, their sensory consequences do not match the desired consequences. This could result in an increased need for feedback-based corrections, including interruptions or resets of the feedforward commands that give rise to sound/syllable repetitions and sound prolongations. In addition to this possibility, however, we speculate that it may be more likely that the types of speech dysfluencies that are characteristic of stuttering result from problems with forward internal models. Based on the integrative model, this would result in problematic feedback control. If the consequences of prepared motor commands cannot be

accurately predicted based on an efference copy and concurrent afferent inflow, a mismatch may arise between predicted and actual consequences of the executed movements, regardless of whether or not the generated commands were accurate with respect to the desired movement outcome. As a result of such a mismatch, the CNS may respond by re-attempting the movement and reissuing the central commands until the sensory consequences are interpreted as matching the desired consequences, sustaining the already ongoing commands until the conflict is resolved or avoided by relying on moment-to-moment afferent feedback, or generating a different set of commands. These types of attempted repairs could result in prolonged or repeated muscle contractions, and, thus, give rise to the sound/syllable repetitions and sound prolongations that are characteristic of stuttering.

In order to minimize the frequency of occurrence of the described maladaptive responses to mismatches between predicted and actual consequences—and thus to increase the likelihood that speech is produced fluently—the CNS of PWS may prefer a motor control strategy that involves longer movement durations because longer durations allow more time for the processing and integration of afferent inputs. This feedback could be used for subtle adjustments during completion of the movement, and, therefore, to compensate for the reduced efficiency of the typical feedback controller that relies heavily on efferent inputs and internal models. Hence, the slower movements observed in stuttering speakers may represent a preferred motor strategy rather than a physiological limitation in movement speed. This suggestion has also been made by others, and is consistent with, among other things, empirical data showing that speaking at a slower rate is a fluency-enhancing condition for most PWS (Adams, Lewis, & Besozzi, 1973) and our own preliminary finding that the largest difference in movement duration between stuttering and nonstuttering individuals occurs for those movements that are normally performed with the shortest durations and highest velocities (Max, Caruso, et al., 2003). Additionally, the perspective is also consistent with De Nil et al.'s (2001) cerebellar imaging data suggesting that stuttering individuals' movements may be less automatic and more dependent on sensory or motor monitoring than those of nonstuttering individuals. Importantly, it is also highly compatible with the work by Wu and colleagues (Riley et al., 2001; Wu et al., 1995, 1997) implicating basal ganglia involvement in stuttering: as reviewed above, recent data have suggested a major role of dopaminergic systems in sensorimotor integration and learning.

For completeness, it should be noted that this proposal shows some overlap with earlier work by Neilson and Neilson (1987). However, the current hypothesis differs fundamentally from that previous work in several aspects: (a) it integrates concepts from the most recent movement neuroscience literature, (b) it offers a different explanation for the basic nature of the speech dysfluencies, (c) it takes a different perspective in terms of the neural processes leading to those dysfluencies, (d) it provides a different explanation for the common observation of prolonged

speech and nonspeech movement durations in individuals who stutter, (e) it attributes a crucial role to anatomical structural and neural maturation, (f) it provides a different explanation for the fluency-enhancing effect of various conditions with altered auditory feedback, and (g) it proposes hypotheses for explanations of various phenomena associated with the disorder (e.g., age of onset, fluency-enhancing conditions, treatment outcomes—see below).

Hypothesis 2: Weak Feedforward Control and Overreliance on Afferent Feedback

A slightly different perspective on the possible sensorimotor sources of stuttering, but also framed within the global model shown in Figure 1 is formulated in our second hypothesis. One important difference with the hypothesis discussed above is that this second hypothesis does not assume any problems with stuttering speakers' internal models or their use. Another difference is that the second hypothesis proposes that an overreliance on strictly afferent feedback is not a strategy selected to avoid stuttering but rather a strategy that actually results in stuttering due to instabilities inherent in this type of control. Indeed, there is always a time lag between a motor command and its auditory and somatosensory consequences. When movements are primarily under afferent feedback control (i.e., weighted more toward afferent feedback control than, as is common for well-practiced tasks, toward feedforward control), the delay in arrival of the sensory signals may render the system unstable. Such instabilities, expected particularly for fast movements, could lead to effector oscillations and system resets. Similar to the proposal in our alternative hypothesis discussed above, resets of the sensorimotor system would result in the observable speech dysfluencies that are characteristic of stuttering.

If a control strategy that is biased toward afferent feedback control results in system instabilities and stuttering moments, the question can be raised why PWS would continue to use such a strategy that nonstuttering individuals replace with a feedforward strategy after initial practicing and learning of the motor task early in development. Our tentative suggestion is that PWS may have weakened feedforward control projections, and this, in turn, may lead to the need or preference for a speech motor strategy that depends primarily on afferent input. For example, using diffusion tensor imaging to investigate brain structure, Sommer, Koch, Paulus, Weiller, and Büchel (2002) found that stuttering adults show abnormalities in the white matter pathways underlying the orofacial area of the left-hemisphere primary sensorimotor cortex. Damage to these pathways may compromise the feedforward command from premotor to primary motor areas. In the context of this hypothesis, stuttering individuals' preference for longer movement durations during speech and nonspeech movements as well as improvements in speech fluency under conditions of decreased speech rate are attributed to the fact that slower movements, as compared with faster movements, are less affected by the delays associated with afferent information.

COMPUTER SIMULATIONS WITH THE DIVA MODEL

Since the mid 1990s, Guenther's research group has been developing, updating, and expanding a neural network model of the central control (and its acquisition) of speech movements (e.g., Guenther, 1994; Guenther & Ghosh, 2003). This model, known as the DIVA model, combines mathematical descriptions of underlying commands, cerebral and cerebellar neural substrates corresponding to the model's components, and computer simulations controlling an articulatory synthesizer.

During an initial babbling phase, the model (schematically represented in Figure 2) learns to control movements of the vocal tract by using the auditory feedback from self-generated speech sounds to learn the mappings between central commands and acoustic consequences. Once these neural mappings have been tuned, production of a particular sequence of speech sounds starts with the activation of speech sound map cells in premotor cortex (in the computer simulations, each speech sound map cell corresponds to one phoneme or syllable). Activation of a speech sound map cell results in the readout of a feedforward command from premotor cortex to primary motor cortex as well as a feedback command passing through the auditory and somatosensory areas before reaching motor cortex.

Early in development, the feedforward command is inaccurate, and the model depends on feedback control. The projections in the feedback control subsystem constitute *forward models* encoding the expected sensory consequences of the sounds to be produced. The feedback system compares these expectations to the system's current state as signaled by incoming *afferent* information. If the current auditory and somatosensory states are outside the target regions for the produced sound, error signals are generated in higher order sensory areas. The error signals are then transformed into corrective motor commands by *inverse model* projections from the sensory areas to the primary motor cortex. Over time, however, the feedforward command becomes well tuned through monitoring of the movements controlled by the feedback subsystem. Once the feedforward subsystem is accurately tuned, the system can rely almost entirely on feedforward commands because no sensory errors are generated unless external perturbations are applied to the system.

In the DIVA model, cells in the motor cortex generate the overall motor command M(t), which is a combination of feedforward and feedback commands:

$$M(t) = M(0) + \alpha_{ff} \int_0^t \dot{M}_{feedforward}(t)g(t)dt + \alpha_{fb} \int_0^t \dot{M}_{feedback}(t)g(t)dt$$

with α_{ff} and α_{fb} representing the amount of weighting toward feedforward and feedback control, respectively, and *g(t)* representing a speech rate signal that is 0 when not speaking and 1 when speaking at the maximum rate.

In computer simulations to date, our hypothesis that stuttering may result from weak feedforward control and overreliance on afferent feedback (i.e., the second of our two hypotheses described above) has been implemented in the DIVA model by using an inappropriately low value of

Figure 2. Left panel: Schematic representation of the directions into velocities of articulators (DIVA) model, a mathematical neural network model of speech movements (Guenther, 1994; Guenther & Ghosh, 2003). Right panel: Vocal tract model controlled by the DIVA model for speech synthesis.
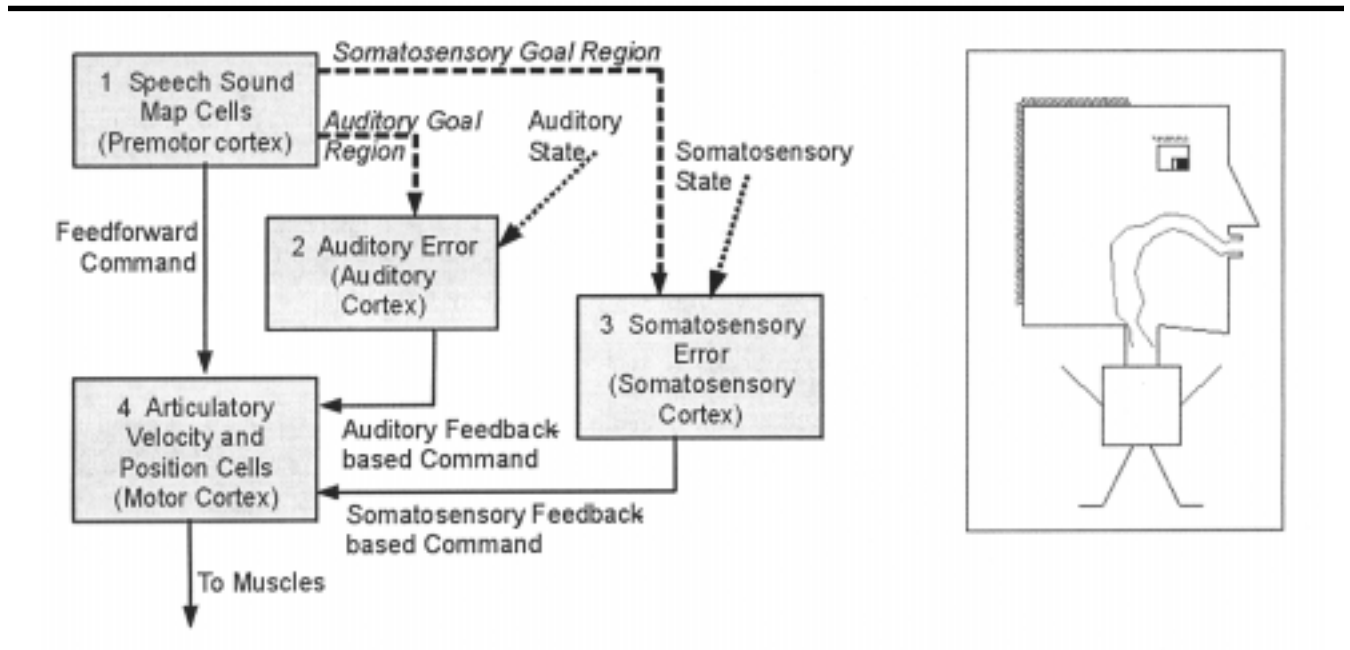
α_{ff} and/or an inappropriately high value of α_{fb} coupled with a reset signal triggered by the resulting large sensory errors. Introducing such a bias toward feedback control (which is unstable in this model due to the time lags associated with the afferent information) and the reset signal leads to stuttering behavior (in particular sound repetitions) in the vocal tract model. We hypothesize that early in development, children have a high threshold for sensory error-based motor resets, thus preventing them from constantly resetting (stuttering) while learning new sounds, and that this threshold decreases as a function of age (solid line in Figure 3). For normally developing children, the size of error signals during speech decreases due to improved use of feedforward commands (dotted line), but in other children, the weak feedforward system does not sufficiently decrease the magnitude of sensory error signals (dashed line), leading to the onset of stuttering when the threshold for motor reset dips below the error signal magnitude in these individuals (vertical line).

Work is currently underway to determine whether or not our hypothesis that stuttering may result from unstable or incorrect internal models (i.e., the first hypothesis described above) is also feasible within the DIVA model given its specific mathematical representations of internal models in the feedforward and feedback control subsystems.

CAN THE THEORETICAL PERSPECTIVE ACCOUNT FOR THE PHENOMENA ASSOCIATED WITH STUTTERING?

Above, we have described numerous findings from the stuttering literature and the general motor control literature

that led to, and thus are directly compatible with, the proposed hypotheses. However, for these hypotheses to have the potential to be developed into a comprehensive theoretical perspective, they need to be able to account not only for the primary characteristics or core behaviors (proposed sources for part-word repetitions and audible and inaudible sound prolongations were discussed above), but also for phenomena well known to be associated with the disorder. We therefore briefly suggest here for some of these phenomena (typical age of onset, spontaneous recovery, influence of genetics, fluency enhancing effect of altered auditory feedback, treatment efficacy with young children) the bases on which interpretations within the context of the proposed hypotheses can be developed.

Our overall perspective is highly consistent with the fact that the onset of stuttering typically occurs during early childhood, with the peak years of onset occurring between 2 and 5 years of age (Andrews et al., 1983). With regard to our first hypothesis, the suggestion is that stuttering may develop because of an incorrect learning/updating or insufficient activation of the various inverse and/or forward internal models used for sensorimotor control of speech movements. Such difficulties would be most likely to develop during an early childhood period in which rapid neural and musculoskeletal changes as a result of maturation require continuous updating and refining of the internal models. Our second hypothesis suggests that, as part of the crucial motor learning that takes place during speech development, it is during these early childhood years that nonstuttering individuals gradually replace a motor control strategy that is biased more toward afferent feedback control with one that is biased more toward feedforward control. When neuromotor limitations (possibly

Figure 3. Hypothesized threshold for motor reset (solid line) as a function of time during the first months of speech, along with a typical sensory error time course for normally developing individuals (dotted line) and individuals who develop stuttering (dashed line; vertical line denotes the onset of stuttering).
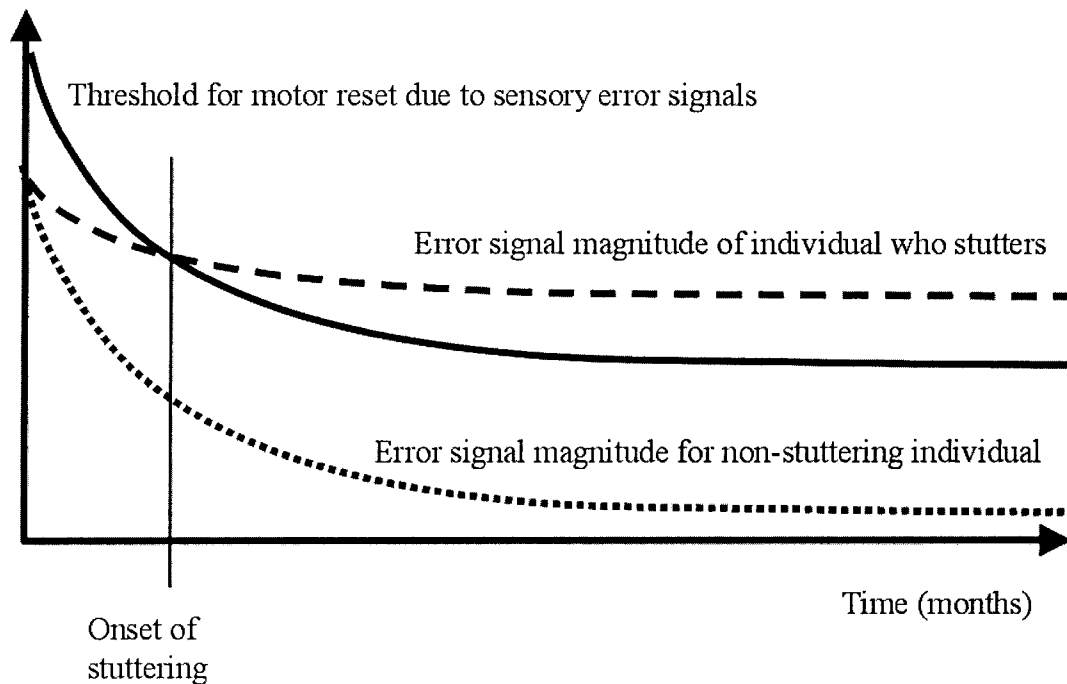
anatomically based) cause a failure to make this transition at the time when articulatory complexity and rate start to increase, the overreliance on feedback control may lead to the described instabilities. Along the same lines, the process of spontaneous recovery from stuttering during childhood may represent either a successful acquisition and updating of the required internal models or a successful transition toward more feedforward-based control as a result of sensorimotor learning in parallel with neuroanatomical/neurochemical maturation. Moreover, the role of genetic factors in stuttering (Yairi, Ambrose, & Cox, 1996) may be related to a genetic predisposition in the form of a neuroanatomical/neurochemical sensorimotor limitation that may or may not lead to persistent stuttering depending on the influence of other variables.

It is well documented that a variety of conditions of altered auditory feedback have a powerful fluency-enhancing effect (i.e., delayed auditory feedback or DAF, frequency altered auditory feedback or FAF, masking, unison or chorus reading). The interpretation of such improvements due to alterations in auditory feedback is again different for each of our two hypotheses. In the context of our first hypothesis, we suggest that this effect could be a result not of those modifications *per se*, but of their common role in providing an external auditory stimulus that facilitates activation of the auditory cortex. Given that the effect is fluency enhancing even for masking (and, in fact, even when the masking is present in the *silent* intervals during conversational or read speech [Sutton & Chase, 1961]), this external auditory stimulation may have its influence primarily through an overall activation of auditory cortex

that increases activation of the internal models used to monitor efference copies of the motor commands rather than through altered monitoring of actual auditory afferent signals (Paus, Marrett, et al., 1996; Paus, Perry, et al., 1996). In other words, the activation of the auditory cortex by external auditory stimuli may improve the efficiency of feedback monitoring by improving the feedback controller's predictions of the auditory consequences of planned movements. It is interesting in this regard that studies have also shown activation in the auditory cortex and neighboring areas such as middle and superior temporal gyrus and superior temporal sulcus in response to *visual* perception of a speaker's silent speech movements or even nonspeech movements that can be interpreted as speech movements (Calvert et al., 1997; Campbell et al., 2001). This may account for the finding that watching another individual perform silent speech movements (which clearly does not alter auditory afferent input) also has a fluency-enhancing effect (Kalinowski, Stuart, Rastatter, Snyder, & Dayalu, 2000). In the context of our second hypothesis, the suggestion would be that altered feedback effectively shuts down the feedback circuit because of the consistent perception of feedback that is inconsistent with the speaker's own actions. Because of a normalization process implemented in the model, driving down the output gain of the feedback controller drives up the output gain of the feedforward controller. This increased output gain corrects the problem with the previously weak feedforward signals.

We believe that the hypotheses presented here can also explain the well-established stuttering adaptation effect (i.e., averaged across subjects, stuttering frequency decreases by

approximately 50% during five repeated readings of the same text). We have previously shown that this decrease in stuttering frequency results from the repeated reading rather than repeated stuttering, and that some changes in the speech acoustics are consistent with those occurring during nonspeech motor practice (Max & Caruso, 1998; Max, Caruso, & Vandevenne, 1997). Consequently, we suggested in those previous publications that the improvements in speech fluency during an adaptation paradigm may represent a form of motor learning, albeit with rather short-term benefits due to the very limited amount of practice. To further examine this possibility, we recently completed a case study of an individual with acquired neurogenic stuttering. If individuals with stuttering due to a known brain lesion show no capability to reduce their stuttering with repeated readings, then the fact that individuals with developmental stuttering do benefit from such motor practice may indeed provide an important piece of information regarding the mechanisms underlying developmental stuttering. Interestingly, our study of a 57-year-old male who started stuttering after lesions to the right frontal lobe and the pons did not show any improvement in fluency during an adaptation paradigm (Balasubramanian, Max, Van Borsel, Rayca, & Richardson, 2003). Hence, the possibility for practice-induced fluency improvements in individuals with developmental stuttering but not in individuals with neurogenic stuttering suggests that mechanisms involved in the developmental form of the disorder can be influenced by practice and motor learning. Our hypotheses are in keeping with this conclusion: Repeated performance of sequences of speech movements could lead to increased precision in the preparation of motor commands by the feedforward controller, more accurate predictions of the sensory consequences by the feedback controller, or an increase of the output gain of the feedforward controller. We believe that this reasoning is also supported by findings that a motorically simplified (i.e., all voiced) speech task does not lead to an immediate decrease in stuttering frequency but to a more rapid decrease during repeated practice (Adams & Reis, 1974).

Last, we propose that the hypotheses are also consistent with the currently available data on treatment efficacy in children who stutter. More treatment outcome data are available for the Lidcombe Program developed by Onslow and colleagues (e.g., Lincoln, Onslow, & Reed, 1997; Lincoln & Onslow, 1997; Onslow, Andrews, & Lincoln, 1994; Onslow, Packman, Stocker, van Doorn, & Siegel, 1997) than for any other treatment program. Published evidence suggests that the Lidcombe Program is highly effective in improving speech fluency in children. The reasons for this success have remained unclear, and research to date has not shown treatment-related changes in temporal measures of the children's speech (Onslow, Stocker, Packman, & McLeod, 2002). During the Lidcombe Program, parents are carefully taught to "correct" some of the child's stuttering moments by describing in age-appropriate language that stuttering occurred and then asking the child to repeat that same word fluently. Some fluent productions are then repeated more than once, with each production followed by the parent's verbal reinforcement. We suggest that these repeated productions of fluent words may contribute greatly to the therapeutic effect. Evidence in support of this position can be found in the aforementioned data on behavioral improvements in nonhuman primate arm movements that were performed while the animal was wearing prism glasses: The most critical factor for motor memory consolidation was continued practice with minimal or no error (Yin & Kitazawa, 2001). In essence, our interpretation is that the Lidcombe Program's technique of repeatedly eliciting and reinforcing fluent productions facilitates motor learning, similar to that described above for stuttering adaptation paradigms. Improvements in speech fluency would result from the already described beneficial effects on the feedforward and feedback controllers or on the relative output gain of these controllers.

CONCLUSION

Based on recent insights into the neural control of movement and modeling of the underlying mechanisms, we have described here two specific hypotheses about the possible sensorimotor sources of stuttering. Both hypotheses propose specific components or processes within a widely used sensorimotor control scheme as possible sources for the speech dysfluencies in stuttering. Specifically, the hypotheses suggest that stuttering may result from (a) unstable or insufficiently activated internal models in the feedforward and feedback control subsystems for speech movements or (b) an overreliance on afferent feedback that, due to the time lags inherent in afferent signals, leads to system instabilities. We also presented hypothetical explanations for how the overall perspective may account not only for the primary characteristics of stuttering, but also for a variety of phenomena associated with the disorder and its development. Hence, these hypotheses are potential explanations of the so-called "proximal" sources of stuttering. That is, they propose explanations for what causes a single moment of stuttering when an individual who stutters is speaking. In their current stage of development, the hypotheses do not fully address the "distal" sources of stuttering (i.e., Why does a certain individual have the disorder?), although some preliminary speculations in this regard are included (e.g., anatomical and functional differences in neural pathways, basal ganglia dopaminergic imbalance), and understanding this aspect of the disorder is an integral part of the long-term goals of our collaborative work.

Of course, much of what we have presented here has been derived indirectly from the stuttering literature and remains speculative at this time. Direct empirical tests of these hypotheses need to be developed (work currently underway in our laboratories) and conducted with both children and adults who stutter. However, theory building is important in a scientific area that has recently shown a remarkable lack of new theoretical models (Adams, 1999). In the last two decades, there have been very few attempts at formulating a comprehensive framework that would account not only for the primary characteristics (i.e., sound and syllable repetitions, audible and inaudible sound

prolongations) of stuttering, but also for the various phenomena known to be associated with stuttering (e.g., typical range for the age of onset, empirical results regarding sensory and motor performance in individuals who stutter, fluency improvements during adaptation paradigms and fluency-enhancing conditions, positive results of specific treatment programs). Therefore, we would like to conclude by fully supporting Conture's (2001, p. 27) description of what contributes to scientific progress:

> At this point, we should be, in my opinion, willing to entertain likely rather certain explanations for why people stutter. Furthermore, the more sources of theoretical input we receive, from as many different perspectives as possible, the greater the possibility that no relevant issue will be overlooked.... it is only by offering different theories and therapies in the marketplace of ideas that the truth will emerge. Eventually. Disagreements, not agreements, typically foster and encourage new insights into old problems and are part of the stuff from which progress is made.

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REFERENCES

Adams, M. R. (1999). A perspective on stuttering. *Contemporary Issues in Communication Science and Disorders, 26,* 5–13.

Adams, M. R., Lewis, J. I., & Besozzi, T. E. (1973). The effect of reduced reading rate on stuttering frequency. *Journal of Speech and Hearing Research, 16,* 671–675.

Adams, M. R., & Reis, R. (1974). Influence of the onset of phonation on the frequency of stuttering: A replication and reevaluation. *Journal of Speech and Hearing Research, 17,* 752–754.

Alfonso, P. J. (1991). Implications of the concepts underlying task-dynamic modeling on kinematic studies of stuttering. In H. F. M. Peters, W. Hulstijn, & C. W. Starkweather (Eds.), *Speech motor control and stuttering* (pp. 79–100). Amsterdam: Elsevier.

Amabile, G., Fattapposta, F., Pozzessere, G., Albani, G., Sanarelli, L., Rizzo, P. A., et al. (1986). Parkinson disease: Electrophysiological (CNV) analysis related to pharmacological treatment. *Electroencephalography and Clinical Neurophysiology, 64,* 521–524.

Andrews, G., Craig, A., Feyer, A.-M., Hoddinott, S., Howie, P., & Neilson, M. (1983). Stuttering: A review of research findings and theories circa 1982. *Journal of Speech and Hearing Disorders, 48,* 226–246.

Atkeson, C. G. (1989). Learning arm kinematics and dynamics. *Annual Review of Neuroscience, 12,* 157–183.

Balasubramanian, V., Max, L., Van Borsel, J., Rayca, K. O., & Richardson, D. (2003). Acquired stuttering following right frontal and bilateral pontine lesion: A case study. *Brain and Cognition, 53,* 185–189.

Bhushan, N., & Shadmehr, R. (1999). Computational nature of human adaptive control during learning of reaching movements in force fields. *Biological Cybernetics, 81,* 39–60.

Bishop, J. H., Williams, H. G., & Cooper, W. A. (1991). Age and task complexity variables in motor performance of stuttering and nonstuttering children. *Journal of Fluency Disorders, 16,* 207–217.

Blakemore, S. J., Frith, C. D., & Wolpert, D. M. (2001). The cerebellum is involved in predicting the sensory consequences of action. *Neuroreport, 12,* 1879–1884.

Blakemore, S. J., Goodbody, S. J., & Wolpert, D. M. (1998). Predicting the consequences of our own actions: The role of sensorimotor context estimation. *Journal of Neuroscience, 18,* 7511–7518.

Blakemore, S. J., Wolpert, D., & Frith, C. (1998). Why can't you tickle yourself? *NeuroReport, 11,* 1211–1216.

Blakemore, S. J., Wolpert, D., & Frith, C. (2000). Central cancellation of self-produced tickle sensation. *Nature Neuroscience, 1,* 635–640.

Bloodstein, O. (1993). *Stuttering: The search for a cause and cure.* Boston: Allyn & Bacon.

Bloodstein, O. (1995). *A handbook on stuttering* (5th ed.). San Diego, CA: Singular.

Boberg, E., & Kully, D. (1989). A retrospective look at stuttering therapy. *Journal of Speech-Language Pathology and Audiology, 13,* 5–13.

Borden, G. J., & Armson, J. (1987). Coordination of laryngeal and supralaryngeal behavior in stutterers. In H. F. M. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 209–214). Wien, Austria: Springer-Verlag.

Borden, G. J., Kim, D. H., & Spiegler, K. (1987). Acoustics of stop consonant-vowel relationships during fluent and stuttered utterances. *Journal of Fluency Disorders, 12,* 175–184.

Boutsen, F. (1995). A comparative study of stress timing of stutterers and nonstutterers. *Journal of Fluency Disorders, 20,* 145–156.

Boutsen, F. R., Brutten, G. J., & Watts, C. R. (2000). Timing and intensity variability in the metronomic speech of stuttering and nonstuttering speakers. *Journal of Speech, Language, and Hearing Research, 43,* 513–520.

Braun, A. R., Varga, M., Stager, S., Schulz, G., Selbie, S., Maisog, J. M., et al. (1997). Altered patterns of cerebral activity during speech and language production in developmental stuttering: An $H_{2(15)}O$ positron emission tomography study. *Brain, 120,* 761–784.

Brown, C. J., Zimmermann, G. N., Linville, R. N., & Hegmann, J. P. (1990). Variations in self-paced behaviors in stutterers and nonstutterers. *Journal of Speech and Hearing Research, 33,* 317–323.

Brutten, E. J., & Shoemaker, D. J. (1967). *The modification of stuttering.* Englewood Cliffs, NJ: Prentice-Hall.

Calvert, G. A., Bullmore, E. T., Brammer, M. J., Campbell, R., Williams, S. C., McGuire P. K., et al. (1997). Activation of auditory cortex during silent lipreading. *Science, 276,* 593–596.

Campbell, R., MacSweeney M., Surguladze, S., Calvert, G., McGuire, P., Suckling, J., et al. (2001). Cortical substrates for the perception of face actions: An fMRI study of the specificity of activation for seen speech and for meaningless lower-face acts (gurning). *Cognitive Brain Research, 12,* 233–234.

Caruso, A. J., Abbs, J. H., & Gracco, V. L. (1988). Kinematic analysis of multiple movement coordination during speech in stutterers. *Brain, 111,* 439–456.

Caruso, A. J., Conture, E. G., & Colton, R. H. (1988). Selected temporal parameters of coordination associated with stuttering in

children. *Journal of Fluency Disorders, 13*, 57–82.

Caruso, A. J., Gracco, V. L., & Abbs, J. H. (1987). A speech motor control perspective on stuttering: Preliminary observations. In H. F. M. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 245–258). Wien, Austria: Springer-Verlag.

Cohen, L. G., & Starr, A. (1987). Localization, timing and specificity of gating of somatosensory evoked potentials during active movement in man. *Brain, 110*, 451–467.

Conture, E. G. (2001). *Stuttering: Its nature, diagnosis, and treatment*. Needham Heights, MA: Allyn & Bacon.

Conture, E. G., Colton, R. H., & Gleason, J. R. (1988). Selected temporal aspects of coordination during fluent speech of young stutterers. *Journal of Speech and Hearing Research, 31,* 640–653.

Cooper, M. H., & Allen, G. D. (1977). Timing control accuracy in normal speakers and stutterers. *Journal of Speech and Hearing Research, 1,* 55–71.

Curio, G., Neuloh, G., Numminen, J., Jousmäki, V., & Hari, R. (2000). Speaking modifies voice-evoked activity in the human auditory cortex. *Human Brain Mapping, 9,* 183–191.

De Nil, L. F. (1995). The influence of phonetic context on temporal sequencing of upper lip, lower lip, and jaw peak velocity and movement onset during bilabial consonants in stuttering and nonstuttering adults. *Journal of Fluency Disorders, 20,* 127–144.

De Nil, L. F., & Abbs, J. H. (1991). Kinaesthetic acuity of stutterers and non-stutterers for oral and non-oral movements. *Brain, 114,* 2145–2158.

De Nil, L. F., Kroll, R. M., Kapur, S., & Houle, S. (2000). A positron emission tomography study of silent and oral single word reading in stuttering and nonstuttering adults. *Journal of Speech, Language, and Hearing Research, 43,* 1038–1053.

De Nil, L. F., Kroll, R. M., & Houle, S. (2001). Functional neuroimaging of cerebellar activation during single word reading and verb generation in stuttering and nonstuttering adults. *Neuroscience Letters, 302,* 77–80.

Desmurget, M., & Grafton, S. (2000). Forward modeling allows feedback control for fast reaching movements. *Trends in Cognitive Sciences, 4,* 423–431.

Fattapposta, F., Pierelli, F., My, F., Mostarda, M., Del Monte, S., Parisi, L., et al. (2002). L-dopa effects on preprogramming and control activity in a skilled motor act in Parkinson's disease. *Clinical Neurophysiology, 113*, 243–253.

Fattapposta, F., Pierelli, F., Traversa, G., My, F., Mostarda, M., D'Alessio, C., et al. (2000). Preprogramming and control activity of bimanual self-paced motor task in Parkinson's disease. *Clinical Neurophysiology, 111*, 873–883.

Flament, D., Ellermann, J. M., Kim, S. G., Ugurbil, K., & Ebner, T. J. (1996). Functional magnetic resonance imaging of cerebellar activation during the learning of a visuomotor dissociation task. *Human Brain Mapping, 4,* 210–226.

Flanagan, B., Goldiamond, I., & Azrin, N. H. (1959). Instatement of stuttering in normally fluent individuals through operant procedures. *Science, 130,* 979–981.

Flanagan, J. R., & Lolley, S. (2001). The inertial anisotropy of the arm is accurately predicted during movement planning. *Journal of Neuroscience, 21,* 1361–1369.

Flanagan, J. R., & Wing, A. M. (1993). Modulation of grip force with load force during point-to-point movements. *Experimental Brain Research, 95,* 131–143.

Flanagan, J. R., & Wing, A. M. (1997). The role of internal models in motion planning and control: Evidence from grip force adjustments during movements of hand-held loads. *Journal of Neuroscience, 17,* 1519–1528.

Forster, D. C., & Webster, W. G. (2001). Speech-motor control and interhemispheric relations in recovered and persistent stuttering. *Developmental Neuropsychology, 19,* 125–145.

Fox, P. T., Ingham, R. J., Ingham, J. C., Hirsch, T. B., Downs, J. H., Martin, C., et al. (1996). A PET study of the neural systems of stuttering. *Nature, 382,* 158–161.

Fox, P. T., Ingham, R. J., Ingham, J. C., Zamarripa, F., Xiong, J.-H., & Lancaster, J. L. (2000). Brain correlates of stuttering and syllable production: A PET performance-correlation analysis. *Brain, 123,* 1985–2004.

Friston, K. J., Frith, C. D., Passingham, R. E., Liddle, P. F., & Frackowiak, R. S. (1992). Motor practice and neurophysiological adaptation in the cerebellum: A positron tomography study. *Proceedings of the Royal Society of London, B: Biological Sciences, 248,* 223–228.

Gracco, V. L. (1994). Some organizational characteristics of speech movement control. *Journal of Speech and Hearing Research, 37,* 4–27.

Grafton, S. T., Woods, R. P., & Tyszka, M. (1994). Functional imaging of procedural motor learning: Relating cerebral blood flow with individual subject performance. *Human Brain Mapping, 1,* 221–234.

Gribble, P. L., & Ostry, D. J. (1999). Compensation for interaction torques during single- and multijoint limb movements. *Journal of Neurophysiology, 82,* 2310–2326.

Grosjean, M., van Galen, G. P., de Jong, P., van Lieshout, P. H. H. M., & Hulstijn, W. (1997). Is stuttering caused by failing neuromuscular force control? In W. Hulstijn, H. F. M. Peters, & P. H. H. M. van Lieshout (Eds.), *Speech production: Motor control, brain research and fluency disorders* (pp. 197–204). New York: Elsevier.

Guenther, F. H. (1994). A neural network model of speech acquisition and motor equivalent speech production. *Biological Cybernetics, 72,* 43–53.

Guenther, F. H., & Ghosh, S. S. (2003). A model of cortical and cerebellar function in speech. In M. J. Solé, D. Recasens, & J. Romero (Eds.), *Proceedings of the 15th International Congress of Phonetic Sciences* (pp. 1053–1056). Barcelona, Spain.

Guitar, B. (1998). *Stuttering: An integrated approach to its nature and treatment* (2nd ed.). Baltimore: Williams & Wilkins.

Hickok, G., Erhard, P., Kassubek, J., Helms-Tillery, A. K., Naeve-Velguth, S., Strupp, J. P., et al. (2000). A functional magnetic resonance imaging study of the role of left posterior superior temporal gyrus in speech production: Implications for the explanation of conduction aphasia. *Neuroscience Letters, 287,* 156–160.

Houde, J. F., & Jordan, M. I. (1998). Sensorimotor adaptation in speech production. *Science*, *279*, 1213–1216.

Houde, J. F., Nagarajan, S. S., Sekihara, K., & Merzenich, M. M. (2002). Modulation of the auditory cortex during speech: An MEG study. *Journal of Cognitive Neuroscience, 14,* 1125–1138.

Howell, P., Sackin, S., & Rustin, L. (1995). Comparison of speech motor development in stutterers and fluent speakers between 7 and 12 years old. *Journal of Fluency Disorders, 20,* 243–256.

Huda, K., Salunga, T. L., & Matsunami, K. (2001). Dopaminergic inhibition of excitatory inputs onto pyramidal tract neurons

in cat motor cortex. *Neuroscience Letters, 307,* 175–178.

Hulstijn, W., Summers, J. J., van Lieshout, P. H. H. M., & Peters, H. F. M. (1992). Timing in finger tapping and speech: A comparison between stutterers and fluent speakers. *Human Movement Science, 11,* 113–124.

Hutchinson, J. M., & Watkin, K. L. (1976). Jaw mechanics during release of the stuttering moment: Some initial observations and interpretations. *Journal of Communication Disorders, 9,* 269–279.

Imamizu, H., Miyauchi, S., Tamada, T., Sasakim Y., Takino, R., Pütz, B., et al. (2000). Human cerebellar activity reflecting an acquired internal model of a new tool. *Nature, 403,* 192–195.

Ingham, R. J. (1984). *Stuttering and behaviour therapy: Current status and experimental foundations.* San Diego, CA: College-Hill Press.

Ingham, R. J. (2001). Brain imaging studies of developmental stuttering. *Journal of Communication Disorders, 34,* 493–516.

Jäncke, L., Kaiser, P., Bauer, A., & Kalveram, T. (1995). Upper lip, lower lip, and jaw peak velocity sequence during bilabial closures: No differences between stutterers and nonstutterers. *Journal of the Acoustical Society of America, 97,* 3900–3903.

Johnson, W. (1942). A study of the onset and development of stuttering. *Journal of Speech Disorders, 7,* 251–257.

Jonas, G. (1976). *Stuttering: The disorder of many theories.* New York: Farrar, Strauss and Giraux.

Kalinowski, J., Stuart, A., Rastatter, M. P., Snyder, G., & Dayalu, V. (2000). Inducement of fluent speech in persons who stutter visual choral speech. *Neuroscience Letters, 281,* 198–200.

Kent, R. D., & Vorperian, H. K. (1995). Development of the craniofacial-oral-laryngeal anatomy: A review. *Journal of Medical Speech-Language Pathology, 3,* 145–190.

Klapp, S. T., Nordell, S. A., Hoekenga, K. C., & Patton, C. B. (1974). Long-lasting aftereffect of brief prism exposure. *Perception & Psychophysics, 15,* 399–400.

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.

Lackner, J. R., & Lobovits, D. (1977). Adaptation to displaced vision: Evidence for prolonged aftereffects. *Quarterly Journal of Experimental Psychology, 29,* 65–69.

Leube, D. T., Knoblich, G., Erb, M., Grodd, W., Bartels, M., & Kircher, T. T. J. (2003). The neural correlates of perceiving one's own movements. *NeuroImage, 20,* 2084–2090.

Lin, L. D., Murray, G. M., & Sessle, B. J. (1994). Functional properties of single neurons in the primate face primary somatosensory cortex. II. Relations with different directions of trained tongue protrusion. *Journal of Neurophysiology, 71,* 2391–2400.

Lincoln, M., & Onslow, M. (1997). Long-term outcome of early intervention for stuttering. *American Journal of Speech-Language Pathology, 6*(1), 51–58.

Lincoln, M., Onslow, M., & Reed, V. (1997). Social validity of the treatment outcomes of an early intervention program for stuttering. *American Journal of Speech-Language Pathology, 6*(2), 77–84.

Loucks, T. M. J., & De Nil, L. F. (2001). Oral kinesthetic deficit in stuttering evaluated by movement accuracy and tendon vibration. In B. Maassen, W. Hulstijn, R. Kent, H. F. M. Peters, & P. H. H. M. van Lieshout (Eds.), *Speech motor control in*

normal and disordered speech (pp. 307–310). Nijmegen, The Netherlands: Vantilt.

Maguire, G. A., Riley, G. D., Franklin, D. L., & Gottschalk, L. A. (2000). Risperidone for the treatment of stuttering. *Journal of Clinical Psychopharmacology, 20,* 479–482.

Mattay, V. S., Tessitore, A., Callicott, J. H., Bertolino, A., Goldberg, T. E., Chase, T. N., et al. (2002). Dopaminergic modulation of cortical function in patients with Parkinson's disease. *Annals of Neurology, 51,* 156–164.

Max, L. (2004). Stuttering and internal models for sensorimotor control: A theoretical perspective to generate testable hypotheses. In B. Maassen, R. Kent, H. F. M. Peters, P. van Lieshout, & W. Hulstijn (Eds.), *Speech motor control in normal and disordered speech* (pp. 357–387). Oxford, UK: Oxford University Press.

Max, L., & Caruso, A. J. (1998). Adaptation of stuttering frequency during repeated readings: Associated changes in acoustic parameters of perceptually fluent speech. *Journal of Speech, Language, and Hearing Research, 41,* 1265–1281.

Max, L., Caruso, A. J., & Gracco, V. L. (2003). Kinematic analyses of speech, orofacial nonspeech, and finger movements in stuttering and nonstuttering individuals. *Journal of Speech, Language, and Hearing Research, 46,* 215–232.

Max, L., Caruso, A. J., & Vandevenne, A. (1997). Decreased stuttering frequency during repeated readings: A motor learning perspective. *Journal of Fluency Disorders, 22,* 17–34.

Max, L., & Glass, N. P. (2001). Relative timing of oral and laryngeal speech movements in individuals who stutter. In H.-G. Bosshardt, J. S. Yaruss, & H. F. M. Peters (Eds.), *Fluency disorders: Theory, research, treatment and self-help. Proceedings of the Third World Congress on Fluency Disorders* (pp. 78–82). Nijmegen, The Netherlands: University of Nijmegen Press.

Max, L., & Gracco, V. L. (2003). *Coordination of oral and laryngeal movements in the perceptually fluent speech of adults who stutter.* Manuscript submitted for publication.

Max, L., Gracco, V. L., & Caruso, A. J. (in press). Kinematic event sequencing in stuttering and nonstuttering adults. In A. Packman, A. Meltzer, & H. F. M. Peters (Eds.), *Proceedings of the 4th World Congress on Fluency Disorders.* Nijmegen, The Netherlands: University of Nijmegen Press.

Max, L., Gracco, V. L., Guenther, F. H., Ghosh, S. S., & Wallace, M. E. (in press). A sensorimotor perspective on stuttering: Insights from the neuroscience of motor control. In A. Packman, A. Meltzer, & H. F. M. Peters (Eds.), *Proceedings of the 4th World Congress on Fluency Disorders.* Nijmegen, The Netherlands: University of Nijmegen Press.

Max, L., Wallace, M. E., & Vincent, I. (2003). Sensorimotor adaptation to auditory perturbations during speech: Acoustic and kinematic experiments. In M. J. Solé, D. Recasens, & J. Romero (Eds.), *Proceedings of the 15th International Congress of Phonetic Sciences* (pp. 1053–1056). Barcelona, Spain.

Max, L., & Yudman, E. (2003). Accuracy and variability of isochronous rhythmic timing across motor systems in stuttering versus nonstuttering individuals. *Journal of Speech, Language, and Hearing Research, 46,* 146–163.

McClean, M. D. (1996). Lip-muscle reflexes during speech movement preparation in stutterers. *Journal of Fluency Disorders, 21,* 49–60.

McClean, M. D., Kroll, R. M., & Loftus, N. S. (1990). Kinematic analysis of lip closure in stutterers' fluent speech. *Journal of Speech and Hearing Research, 33,* 755–760.

Mehta, B., & Schaal, S. (2002). Forward models in visuomotor control. *Journal of Neurophysiology, 88,* 942–953.

Melvin, C., Williams, H., Bishop, J., McClenaghan, B., Cooper, W., & McDade, H. (1995). Vocal and manual timing control of adult stutterers and nonstutterers. In C. W. Starkweather & H. F. M. Peters (Eds.), *Stuttering: Proceedings of the First World Congress on Fluency Disorders* (pp. 35–38). Nijmegen, The Netherlands: University of Nijmegen.

Miall, R. C., Weir, D. J., Wopert, D. M., & Stein, J. F. (1993). Is the cerebellum a Smith predictor? *Journal of Motor Behavior, 25*, 203–216.

Mikheev, M., Mohr, C., Afanasiev, S., Landis, T., & Thut, G. (2002). Motor control and cerebral hemispheric specialization in highly qualified judo wrestlers. *Neuropsychologia, 40*, 1209–1219.

Neilson, M. D., & Neilson, P. D. (1987). Speech motor control and stuttering: A computational model of adaptive sensory-motor processing. *Speech Communication, 6*, 325–333.

Nelson, R. J. (1987). Activity of monkey primary somatosensory cortical neurons changes prior to active movement. *Brain Research, 406*, 402–407.

Nelson, R. J. (1996). Interactions between motor commands and somatic perception in sensorimotor cortex. *Current Opinion in Neurobiology, 6*, 801–810.

Numminen, J., Salmelin, R., & Hari, R. (1999). Subject's own speech reduces reactivity of the human auditory cortex. *Neuroscience Letters, 265*, 119–122.

Onslow, M., Andrews, C., & Lincoln, M. (1994). A control/experimental trial of an operant treatment for early stuttering. *Journal of Speech and Hearing Research, 37*, 1244–1259.

Onslow, M., O'Brian, S., & Harrison, E. (1997). The Lidcombe Programme of Early Stuttering Intervention: Methods and issues. *European Journal of Disorders of Communication, 32*, 231–250.

Onslow, M., Packman, A., Stocker, S., van Doorn, J., & Siegel, G. M. (1997). Control of children's stuttering with response-contingent time-out: Behavioral, perceptual, and acoustic data. *Journal of Speech, Language, and Hearing Research, 40*, 121–133.

Onslow, M., Stocker, S., Packman, A., & McLeod, S. (2002). Speech timing in children after the Lidcombe Program of Early Stuttering Intervention. *Clinical Linguistics and Phonetics, 16*, 21–33.

Orton, S. T. (1927). Studies in stuttering: Introduction. *Archives of Neurology and Psychiatry, 18*, 671–672.

Packman, A., Onslow, M., & Attanasio, J. (1997). Multifactorial does not necessarily mean multicausal [Abstract]. *Journal of Fluency Disorders, 22*, 92.

Paus, T., Marrett, S., Worsley, K., & Evans, A. (1996). Imaging motor-to-sensory discharges in the human brain: An experimental tool for the assessment of functional connectivity. *NeuroImage, 4*, 78–86.

Paus, T., Perry, D. W., Zatorre, R. J., Worsley, K. J., & Evans, A. C. (1996). Modulation of cerebral blood flow in the human auditory cortex during speech: Role of motor-to-sensory discharges. *European Journal of Neuroscience, 8*, 2236–2246.

Rieber, R. W., & Wollock, J. (1977). The historical roots of the theory and therapy of stuttering. *Journal of Communications Disorders, 10*, 3–24.

Riley, G., Maguire, G., & Wu, J. C. (2001). Brain imaging to examine a dopamine hypothesis in stuttering. In B. Maassen, W. Hulstijn, R. D. Kent, H. F. M. Peters, & P. H. H. M. van Lieshout (Eds.), *Speech motor control in normal and disordered speech* (156–158). Nijmegen, The Netherlands: Vantilt.

Seitz, R. J., Canavan, A. G., Yaguez, L., Herzog, H., Tellmann, L., Knorr, U., et al. (1994). Successive roles of the cerebellum and premotor cortices in trajectorial learning. *NeuroReport, 5*, 2541–2544.

Shadmehr, R., & Holcomb, H. H. (1997). Neural correlates of motor memory consolidation. *Science, 277*, 821–825.

Shadmehr, R., & Mussa-Ivaldi, F. A. (1994). Adaptive representation of dynamics during learning of a motor task. *Journal of Neuroscience, 14*, 3208–3224.

Sheehan, J. (1958). Conflict theory of stuttering. In J. Eisenson (Ed.), *Stuttering: A symposium* (pp. 121–166). New York: Harper & Row.

Silverman, F. H. (1996). *Stuttering and other fluency disorders* (2nd ed.). Boston: Allyn & Bacon.

Sommer, M., Koch, M. A., Paulus, W., Weiller, C., & Büchel, C. (2002). Disconnection of speech-relevant brain areas in persistent developmental stuttering. *Lancet, 360*, 380–383.

Smith, A., & Kleinow, J. (2000). Kinematic correlates of speaking rate changes in stuttering and normally fluent adults. *Journal of Speech, Language, and Hearing Research, 43*, 521–536.

Sperry, R. W. (1950). Neural basis of the spontaneous optokinetic response produced by visual inversion. *Journal of Comparative and Physiological Psychology, 43*, 482–489.

Stromsta, C. (1986). *Elements of stuttering*. Oshtemo, MI: Atsmorts.

Stuart, A., Kalinowski, J., & Rastatter, M. P. (1997). Effect of monaural and binaural altered auditory feedback on stuttering frequency. *Journal of the Acoustical Society of America, 101*, 3806–3809.

Suri, R. E., Bargas, J., & Arbib, M. A. (2001). Modeling functions of striatal dopamine modulation in learning and planning. *Neuroscience, 103*, 65–85.

Sutton, S., & Chase, R. A. (1961). White noise and stuttering. *Journal of Speech and Hearing Research, 4*, 72.

Thoroughman, K. A., & Shadmehr, R. (1999). Electromyographic correlates of learning an internal model of reaching movements. *Journal of Neuroscience, 19*, 8573–8588.

Travis, L. E. (1931). *Speech pathology*. Englewood Cliffs, NJ: Prentice-Hall.

Van Riper, C. (1970). Historical approaches. In J. G. Sheehan (Ed.), *Stuttering: Research and therapy* (pp. 36–57). New York: Harper & Row.

Van Riper, C. (1973). *The treatment of stuttering*. Englewood Cliffs, NJ: Prentice-Hall.

Van Riper, C. (1982). *The nature of stuttering* (2nd ed.). Englewood Cliffs, NJ: Prentice-Hall.

von Holst, E., & Mittelstaedt, H. (1973). The reafference principle. In R. Martin (Trans.), *The behavioral physiology of animals and man: The collected papers of Erich von Holst* (pp. 139–173). Coral Gables, FL: University of Miami Press. (Original work published in 1950).

Webster, W. G. (1997). Principles of human brain organization related to lateralization of language and speech motor functions in normal speakers and stutterers. In W. Hulstijn, H. F. M. Peters, & P. H. H. M. van Lieshout (Eds.), *Speech production: Motor control, brain research and fluency disorders* (pp. 119–139). Amsterdam: Elsevier.

Webster, W. G., & Ryan, C. R. L. (1991). Task complexity and manual reaction times in people who stutter. *Journal of Speech and Hearing Research, 34*, 708–714.

Wischner, G. J. (1950). Stuttering behavior and learning: A preliminary theoretical formulation. *Journal of Speech and Hearing Disorders, 15*, 324–335.

Wolpert, D. M., Ghahramani, Z., & Flanagan, J. R. (2001). Perspectives and problems in motor learning. *Trends in Cognitive Sciences, 5*, 487–494.

Wolpert, D. M., Ghahramani, Z., & Jordan M. I. (1995). An internal model for sensorimotor integration. *Science, 269*, 1880–1882.

Wolpert, D. M., & Miall, R. C. (1996). Forward models for physiological motor control. *Neural Networks, 9*, 1265–1279.

Wolpert, D. M., Miall, R. C., & Kawato, M. (1998). Internal models in the cerebellum. *Trends in Cognitive Science, 2,* 338–347.

Wu, J. C., Maguire, G., Riley, G., Fallon, J., LaCasse, L., Chin, S., et al. (1995). A positron emission tomography [18F] deoxyglucose study of developmental stuttering. *NeuroReport, 6,* 501–505.

Wu, J. C., Maguire, G., Riley, G., Lee, A., Keator, D., Tang, C., et al. (1997). Increased dopamine activity associated with stuttering. *NeuroReport, 8,* 767–770.

Yairi, E., Ambrose, N., & Cox, N. (1996). Genetics of stuttering: A critical review. *Journal of Speech and Hearing Research, 39,* 771–784.

Yin, P. B., & Kitazawa, S. (2001). Long-lasting aftereffects of prism adaptation in the monkey. *Experimental Brain Research, 141*, 250–253.

Yoshioka, H., & Löfqvist, A. (1981). Laryngeal involvement in stuttering: A glottographic observation using a reaction time paradigm. *Folia Phoniatrica, 33*, 348–357.

Zebrowski, P. M., Conture, E. G., & Cudahy, E. A. (1985). Acoustic analysis of young stutterers' fluency: Preliminary observations. *Journal of Fluency Disorders, 10*, 173–192.

Zelaznik, H. N., Smith, A., & Franz, E. A. (1994). Motor performance of stutterers and nonstutterers on timing and force control tasks. *Journal of Motor Behavior, 26,* 340–347.

Zelaznik, H. N., Smith, A., Franz, E., & Ho, M. (1997). Differences in bimanual coordination associated with stuttering. *Acta Psychologica, 96,* 229–243.

Zimmermann, G. (1980). Stuttering: A disorder of movement. *Journal of Speech and Hearing Research, 23*, 122–136.

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