Brain Models and the Clinical Management of Stuttering
Les modèles cérébraux et le traitement en clinique du bégaiement

by • par

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ABSTRACT

A theoretical model of the brain mechanisms associated with stuttering is described together with the research on which it is based. The two factors of the model, each of which appears to be a necessary but not sufficient condition for stuttering, are an inefficient supplementary motor area, which is unusually susceptible to interference from other ongoing neural activities, and a labile system of hemispheric activation, which results in periodic over-activation of the right hemisphere. It is suggested that paralleling these two hypothesized anomalies are the approaches that characterize most contemporary stuttering treatment programs: Modification of speech or stuttering behaviour, and modification of attitudes and responses related to apprehension and avoidance of social and speaking situations. These latter are an integral part of the stuttering experience and if not dealt with, interfere with using fluency skills in everyday speaking situations. It is suggested that the model provides a rationale and validation for contemporary approaches to the treatment of stuttering, and has heuristic value for clients attempting to understand their stuttering and acquiring and using fluency and cognitive/behavioural skills to manage their speech.

ABRÉGÉ

On décrit un modèle théorique des mécanismes cérébraux associés au bégaiement ainsi que la recherche sous-jacente. Les deux facteurs du modèle, chacun pouvant représenter une condition nécessaire mais non suffisante au bégaiement, sont une zone motrice supplémentaire inefficace, et exceptionnellement susceptible à l'interférence d'autres activités neurales continues, et un système labile d'activation hémisphérique, ce qui produit une suractivation périodique de l'hémisphère droit. Il est proposé que les méthodes caractérisant la plupart des programmes contemporains de traitement du bégaiement parallélisent ces deux anomalies hypothétiques : modification de la parole ou du bégaiement, et modification des attitudes et réactions associées à l'appréhension et à l'évitement de situations sociales et du parler en public. Ces dernières font intégralement partie de l'expérience du bégaiement et, si elles ne sont pas corrigées, nuisent à l'usage d'aptitudes de fluidité dans les situations locutrices communes. Il est suggéré que le modèle explique et valide les méthodes contemporaines de traitement du bégaiement, et qu'il présente une valeur heuristique pour les clients souhaitant, d'une part, comprendre leur bégaiement et, d'autre part, acquérir et utiliser la fluidité et des aptitudes cognitives ou comportementales leur permettant de contrôler la parole.

KEY WORDS: stuttering • speech motor control • brain models • clinical management of stuttering

The Canadian psychologist, Donald O. Hebb, one of the fathers of modern neuropsychology, consistently maintained that nothing is more practical than good theory. According to Hebb (1958), a critical understanding of theory can guide practice and provide a basis other than trial-and-error or intuition for modifying and enhancing it. Theory also allows the professional to understand and appreciate new methods and approaches and to develop strategies for dealing with novel or unusual situations or clients. On the other side of the coin, experience with practical or applied matters informs research hypothesis formulation and theory development, and enriches the potential linkages between theory and practice.

As evidenced through his publications, the late Professor Einer Boberg had an intense interest in matters practical, committed as he was to determining what works to improve fluency, communication, and lifestyle in people who stutter and to implementing the most effective fluency programs he and his colleagues could devise (Boberg & Kully, 1985). But Boberg's interest extended beyond simply what does or does not work (Boberg & Kully, 1988, 1989) to why different approaches or methods work or might work. This interest in

theory was reflected in Boberg's continuing concern (Boberg, 1981, 1985, 1993) for mechanisms and the underlying factors and causes (real, possible, and imaginable) of stuttering. In my interactions with Professor Boberg over a number of years, it was always clear that he appreciated that an understanding of stuttering and its management must be approached from an interdisciplinary perspective. Accordingly, his interest in theory related to stuttering was broad and included theory based in neurology, genetics, developmental biology, neuropsychology, and social and developmental psychology as well as that based in the traditional domain of speech-language pathology.

In light of Professor Boberg's interest in theory, it is appropriate for this contribution to the special *JSLPA* issue honouring him to focus on a discussion of how clinical practice related to the management of stuttering is informed by current working models or theories of the neuropsychological basis of stuttering that have emerged from recent research.

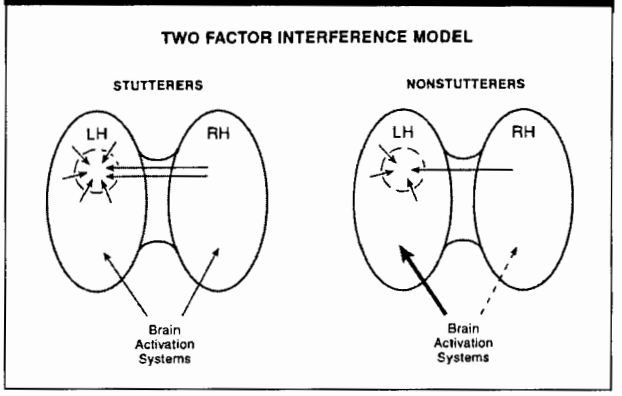
During the past twenty years, a growing body of literature in speech-language pathology and neuropsychology has pointed to a significant role for biological factors underlying stuttering. Noteworthy in this regard are studies of the inheritance of stuttering, particularly those that allow for inferences to be made about genetic factors (Howie, 1981; Kidd, 1984; Ambrose, Cox, & Yairi, 1997) and those that have focused on brain mechanisms. It is this latter body of literature that is the emphasis of this paper, but in trying to sort out whether particular brain characteristics of stutterers bear a cause or effect relationship to their speech, the role of genetics as a factor in both the onset of stuttering and its severity is important to bear in mind.

Current research suggests a working model of brain mechanisms associated with stuttering that has the three elements illustrated schematically in Figure 1: (a) normal left hemisphere lateralization or specialization for speech and language mechanisms in people who stutter; (b) a left hemisphere speech motor control area, possibly the supplementary motor area (SMA), that is unusually susceptible to interference from other on-going brain activity, particularly that in the right hemisphere; and, (c) a lack of the left hemisphere activation bias found in most right-handed, normally fluent speakers, resulting in greater than usual lability of hemispheric activation in stutterers and a consequent tendency for the right hemisphere to become overly activated.

Element 1: Unilateral Speech Motor Control

One of the more notable features of interhemispheric relations in normal human brain organization is hemispheric specialization. Although the two cerebral hemispheres are of more or less the same size and shape, certain cognitive and behavioural functions including speech and language are mediated

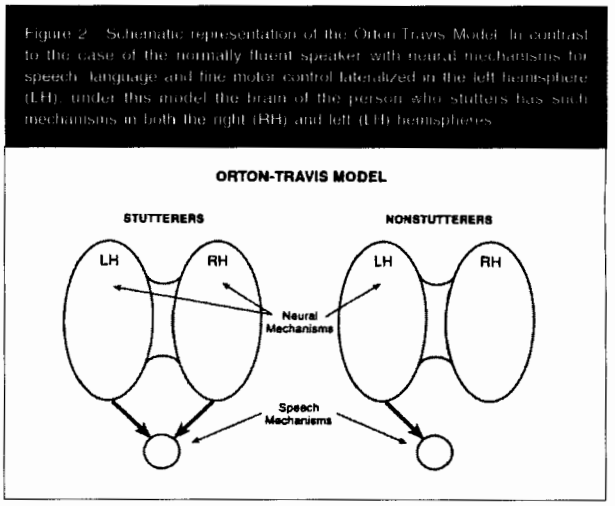
Figure 1. Schematic representation of the Two-Factor Interference Model of Stuttering. Under this model, compared to normally fluent (right-handed) speakers, people who stutter have left hemisphere (LH) speech control mechanisms that are more susceptible (represented by more "pores" around the speech motor control area) to inter- and intra-hemispheric interference, possibly have higher levels of interference (represented by larger numbers of arrows which represent sources of interference), combined with a more equal hemispheric distribution of brain activation and hence a propensity for right hemisphere (RH) over-activation).



primarily by one or the other hemisphere (Webster, 1997). One of the earliest models of neural mechanisms underlying stuttering (Orton, 1928; Travis, 1931) postulated that people who stutter have bilateral rather than left hemisphere speech control mechanisms (Figure 2). According to this idea, the bilateral neural control means that two sets of commands are sent to the speech musculature resulting in discoordination (and hence disfluency) when those commands are out of synchrony. This model has now been tested using a number of different approaches, and it is clear that with respect to hemispheric specialization (as opposed to hemispheric activation, an important distinction to be returned to later), people who stutter show the same organization as normal speakers.

Research in this laboratory to test the Orton/Travis model has made use of an indirect method whose rationale is based

Figure 2. Schematic representation of the Orton-Travis Model. In contrast to the case of the normally fluent speaker with neural mechanisms for speech, language and fine motor control lateralized in the left hemisphere (LH), under this model the brain of the person who stutters has such mechanisms in both the right (RH) and left (LH) hemispheres.



on the concept and evidence discussed elsewhere (Webster, 1990a, 1993) that the neural mechanisms associated with speech overlap those involved in the organization and control of other coordinated sequential motor movements. In an initial study, Webster (1985) found that when participants were required to repeatedly tap telegraph keys with their fingers in a particular sequence, people who stutter showed the same right hand advantage (in speed and accuracy) as normal fluent speakers. The data were interpreted as providing no evidence that the neural mechanisms for sequencing (and by implication, those of speech) are bilaterally represented as postulated in the Orton/Travis model, but instead constitute evidence of normal left hemisphere lateralization of such mechanisms in stutterers. This conclusion is similar to that reached from studies that more directly assessed cerebral dominance for speech and language in people who stutter through injections of sodium amytal into one carotid artery or the other (Wada technique). This procedure is used routinely in neurosurgical contexts and results in a short-term anesthetization of the ipsilateral forebrain which allows for the identification of which hemisphere is specialized for speech and language. As described by Andrews, Quinn, and Sorby (1972) and Luessenhop, Boggs, Lororwit, and Walle (1973), people who stutter respond to these right- and left-sided injections in the same way as do fluent speakers, suggesting again a normal pattern of hemispheric specialization for speech and motor sequencing mechanisms.

This is not to suggest that the right hemisphere is not involved in speech or other fine motor movements in either stutterers or nonstutterers; the evidence to the contrary is clear. A number of studies of the neural correlates of movement using methodologies which include regional cerebral blood flow (Roland, 1993), positron emission tomography (Herholz et al., 1994; Petersen, Fox, Posner, Mintun, & Raichle, 1988), and the analysis of DC cortical potentials recorded from the right and left hemispheres prior to and during movement (Wohlert, 1993), have all demonstrated that areas of primary motor cortex associated with movements of the face, tongue, and jaw are active in both the right and left hemisphere during speech. They have also demonstrated, however, that the same primary sensorimotor areas show increased activity during oral movements, suggesting that the bilateral activation reflects a motor movement effect rather than a speech effect per se.

A second type of involvement of the right hemisphere in speech may relate to prosody. Although open to various interpretations, findings ftom a number of studies of patients with unilateral anterior damage to the right hemisphere (e.g., Behrens, 1988; Ross, 1981; Shapiro & Danly, 1985) suggest that, although such damage may leave speech articulation more or less intact, it can affect the use of prosody and intonation in both speech production and comprehension.

The real concern in the context of stuttering, however, relates to the neural mechanisms associated with the organization and control of coordinated motor movements of a sequential nature, including speech. It is these that are lateralized in the left hemisphere in stutterers just as they are in normal fluent speakers.

Element 2: A Fragile System of Left Hemisphere Speech Motor Control Mechanisms

Despite the evidence for normal left hemispheric specialization for speech processes in people who stutter, a case can be made for these mechanisms not being as efficient as they are in fluent speakers.

This was apparent in the results of an experiment (Webster, 1986b) involving stutterers and nonstutterers performing a sequence reproduction, finger tapping task. The task required participants to tap a new sequence of key presses on each trial rather than tap the same sequence repeatedly, as in the sequential finger tapping task described earlier. Stutterers were found to be slower in initiating their responses and to make more errors in carrying out the first sequence, but once the sequence got started, their performance was just as fast and accurate as that of nonstutterers. One implication of these data is that people who stutter have difficulty, not only with the initiation of their speech utterances, but also with the initiation of new, nonspeech sequential movements. The difficulty appears to be related to response planning and organization. In a separate study, Webster (1989a) found that even when participants were not under time pressure, stutterers still made significantly more errors than did fluent controls in reproducing sequences. This finding, together with others to be described below, led to the hypothesis (Webster, 1988) that a key area of the brain implicated in stuttering is the supplementary motor area (SMA). A similar conclusion was reached by Caruso, Abbs, and Gracco (1988) based on their analysis of the sequential organization of lip and jaw movements in stutterers and nonstutterers.

The hypothesis of a simple inefficiency in the speech motor control system does not provide a satisfactory explanation for variations in stuttering severity across time and situations, a characteristic of the disorder that makes it so frustrating for those who stutter and at the same time makes it so interesting to study scientifically. Such variability implies that the underlying mechanisms involve dynamic processes rather than a static structural defect or anomaly. In other words, this variability makes it likely that the problem will not prove to be that people who stutter have a lesion in the brain but instead some peculiarity in how information is integrated and processed in different regions of the brain.

But what is the nature of these dynamic processes? A number of experiments have suggested that they may include

left hemisphere interference by ongoing neural activity in the right, a model referred to as the Interhemispheric Interference Model (Webster, 1986a, 1993). As illustrated in Figure 3, the model has three major elements: (a) Normally lateralized speech mechanisms; (b) a normally functioning right hemisphere; but, (c) a lack of normal gating of information flow between hemispheres. This third element would

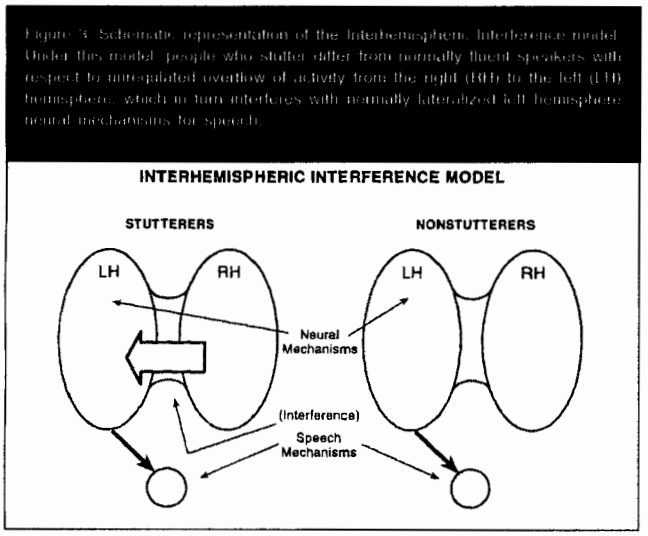


Figure 3. Schematic representation of the Interhemispheric Interference model. Under this model people who stutter differ from normally fluent speakers with respect to unregulated overflow of activity from the right (RH) to the left (LH) hemisphere, which in turn interferes with normally lateralized left hemisphere neural mechanisms for speech.

result in an overflow of activity from the right to the left hemisphere, producing interference in the left hemisphere (in an area I suspect is the SMA). Within this model, variation in stuttering severity would reflect variations in the amount of overflow from the right to the left hemisphere.

A number of experiments (Webster, 1986a, 1988, 1989b, 1990b) were designed to test this model using experimental analogues of the old parlor game of rubbing one's stomach while patting one's head at the same time. The hypothesis of ungated callosal function and consequent excess overflow from the right to the left hemisphere underlay the prediction that people who stutter should have more difficulty than fluent speakers in performing different activities with the two hands at the same time. This is precisely what was found. Stutterers performing the repetitive sequential finger tapping task (Webster, 1986a) or the sequence reproduction task (Webster, 1989b) using the right hand, while performing a paced, knob-turning task with the left hand, showed more interference between the tasks than did fluent controls. A similar differential interference effect was found in a bimanual handwriting task (Webster, 1988) that required participants on each trial to write simultaneously with both hands the first letter of four words that had just been read aloud. Stutterers not only took longer than controls to write the four letters, but they made more errors, particularly mirror reversal errors. A fourth experiment (Webster, 1990b) involved a 2:1 tapping task that required participants to tap one key twice for every single tap of a key by the other hand. As in the other experiments, stutterers showed more inter-hand interference than fluent controls but, as will be discussed at greater length below, they also showed a pattern of results suggesting that a process or brain anomaly other than interhemispheric gating is critical for understanding stuttering.

In summary, then, the evidence is clear that the neural mechanisms underlying speech motor control in stutterers are lateralized in the left hemisphere as they are in normally fluent speakers. However, the difficulties of stutterers in organizing new sequences of responses for both finger movements as well as speech and nonspeech oral movements and for carrying out two tasks with two hands simultaneously, suggest that these mechanisms are inefficient because of interference from other activity in the brain. Because of generalized interference effects we have found in our research (e.g., Webster, 1987; Webster & Forster, 1991), the focus of the current model is on susceptibility of the speech motor area to inference (schematically represented in Figure 1 by the greater number of "pores" in stutterers than nonstutterers). However, I do not entirely discount the possibility of there also being higher levels of interference (schematically represented by the larger number of arrows representing sources of inter- and intra-hemispheric interference) under certain conditions, particularly those of fear or anxiety, as will be discussed later. Nonetheless, brain imaging findings do not suggest any greater overall levels of brain activation in stutterers.

As was suggested above, the specific area of the left hemisphere implicated in the stutterers' fragile speech motor control is the supplementary motor area (SMA). Part of Brodmann area 6, and first identified by Penfield and Welch (1949) during their clinical research involving electrical stimulation of exposed cerebral cortex of human patients, the SMA is located on the medial banks of both hemispheres, just above the cingulate cortex and anterior to the hind limb region of the primary motor cortex (Tanji, 1994). Goldberg (1985), Tanji (1994), and Wise (1984) have reviewed the approaches and methodologies used in recent years to study SMA function in the normal organization of behavior, and a number of consistent sets of findings emerge from the research literature. Four seem particularly germane for present purposes.

First, as found in single unit studies of nonhuman primates (Mushiake, Inase, & Tanji, 1990; Tanji & Shima, 1994) as well as in brain imaging studies of human participants (Roland, 1993), activity in the SMA is associated with planning complex sequential movements of either the limbs or the speech

musculature (Ikeda et al., 1995). The critical words here are planning and complex or sequential. The area does not seem to be critically involved in the execution of well practiced or simple movements but shows evidence of increased activity both when a person is simply thinking about carrying out an action or when movement involves a number of components (Simonetta, Clanet, & Rascol, 1991; Wohlert, 1993). These findings are consistent with reports from human patients that electrical stimulation of the area results in a feeling of intention to move (Penfield & Welch, 1949) but does not result in movement as such. Electrical stimulation of the area also interferes with speech, resulting specifically in slowing, hesitation, and an inability to initiate speech sounds (Ojemann, 1994).

The second point is that there are rich inter- and intrahemispheric connections associated with the SMA. In fact, most interhemispheric connections between motor areas go through the SMA (Rouiller et al., 1994).

The third set of findings (and this should not be surprising in light of the rich interhemispheric connections) implicates the SMA as being crucial in bimanual coordination (Lang, Obrig, Lindinger, Cheyne, & Deecke, 1990). Damage to the SMA interferes with the ability to coordinate hand movements and to do two different things with the hands at the same time (Brinkman, 1984). The fact that in humans damage to the SMA (among other areas) has been reported to result in temporary mutism (Jonas, 1981) and in acquired stuttering (Van Borsel, Van Lierde, Van Cauwenberge, Guldemont & Van Orshoven, 1998) is consistent with a role in the bilateral coordination of the speech musculature.

And finally, there is evidence that the SMA is crucial for the planning of self-initiated and internally guided movements rather than ones that are externally signaled and externally guided (Halsband, Matsuzaka, & Tanji, 1994; Passingham, 1989).

The SMA is an attractive candidate for being a critical locus for stuttering for three principle reasons. First, the basic research findings on SMA summarized above links the area to speech and nonspeech motor functions, both of which are anomalous in people who stutter in ways that were described. Second, the sequence reproduction findings reported by Webster (1986b) suggest a particular problem in the planning and initiation of new sequences of movements, a function that seems to be well associated with the SMA, but not in the execution of practiced movements, a function more associated with other premotor areas. And finally, the anomalies in bimanual coordination in people who stutter (Webster, 1986a, 1988) discussed above also point to the SMA as a potential locus of difficulty. Recent studies using positron emission tomographic (PET) scans to study brain activation in people who stutter have reported activity anomalies that include the SMA and other motor areas (Braun et al., 1997; Fox et al., 1996; Kroll, De Nil, Kapur, & Houle, 1997). What remains to be explored and elaborated is the significance of other areas of anomalous brain activation in stutterers, including the anterior cingulate cortex and temporal cortex, as well as more general questions of how these anomalies and the hypothesized interference susceptibility of the SMA, become translated into the speech repetitions, hesitations, and blockages and the sense of impending loss of speech control (Perkins, 1985) that characterize stuttering. But these important questions are beyond the scope of this particular paper.

Element 3: An Anomaly of Hemispheric Activation

The results of two experiments reported by Webster (1990b) and Forster and Webster (1991) indicated that superimposed on a fragile left hemisphere speech motor system in stutterers is an anomaly in attentional mechanisms with respect, in particular, to hemispheric activation. These two factors together provide the basic framework for the Two-Factor Interference Model illustrated schematically in Figure 1.

A large literature in neuropsychology (Bradshaw & Nettleton, 1983; Bryden, 1982) leads to a distinction between hemispheric specialization and hemispheric activation. The concept of hemispheric specialization refers to the underlying structural specializations of the hemispheres discussed earlier. It is these underlying structural specializations that appear similar in people who stutter to those of fluent speakers. In contrast, the concept of hemispheric activation refers to hemispheric arousal or utilization, either as an enduring bias or one that is temporary and task-specific. Such differential arousal or activation is manifest in the distribution of attentional resources that favour one side of body space or the other (Bryden, 1982). For example, in an experimental dichotic listening context, the expectation of verbal processing (by the structurally specialized left hemisphere) could lead to left hemisphere activation and a preferential attention to the contralateral right ear. Although hemispheric specialization and activation normally go hand in hand (i.e., the hemisphere that is more activated is the one with the neural mechanisms required to perform efficiently the required or anticipated information processing), specialization and activation are, in principle, dissociable or independent depending on the attentional demands of the situation.

Related to the concept of hemispheric activation is the concept of an inherent bias towards left hemisphere activation. A number of lines of evidence, mainly from experimental neuropsychology (Annett, 1978; Bryden, 1978; Cohen, 1982) and in particular from the study of perceptual asymmetries in normal participants (Bryden & Mondor, 1991), suggest that right-handers have a predisposition for the left hemisphere to

maintain a state of greater readiness to process information and to respond. This situation is illustrated schematically in the right side of Figure 1. This same body of literature suggests that left-handers, or at least some left-handers, do not have this bias and appear to be able to direct their attention equally readily to both right and left perceptual space. One clear demonstration of this phenomenon was provided by Peters (1987) who developed a task that required participants to tap a key twice for every single tap of a key by the other hand. Peters reported that right-handers (nonstutterers) performed this task better when it was the right hand that tapped twice (R2/L1 condition) than when it was the left hand (L2/R1 condition). Among left-handers, however, performance was similar under the two lead hand tapping conditions. Drawing upon Annett's (1978) single gene model of handedness, which suggests that right-handers but not left-handers have an inherent directional bias, Peters has argued that the differences he observed between right- and left-handers reflect the role of hemispheric attention in the expression of handedness. More specifically, he argued that right-handers have an attentional bias to the right hand that facilitated performance in the R2/L1 condition, but that left-handers are more flexible in focusing lateralized attention and can attend with equal facility to the right or left hand when leading. Underlying the right side attentional bias in right-handers is thought to be a tonic or on-going left hemisphere activation not found in left-handers who show greater lability of hemispheric activation.

When Webster (1990b) repeated the Peters paradigm with stutterers as a task involving bimanual coordination, he found that both left- and right-handed stutterers performed like the left-handed nonstutterers, their performance being similar regardless of handedness under the two lead hand conditions. Following Peter's interpretation of performance asymmetry, these results suggested that stutterers lack a left hemisphere activation bias and have a greater lability or flexibility of hemispheric activation, or biasing, than do nonstutterers, the situation schematically illustrated in the left side of Figure 1.

A similar conclusion was reached following another experiment (Forster & Webster, 1991) which had a very different purpose and methodology. It was designed to determine whether the dual task interference results discussed above were specific to tasks that involved the opposite hemispheres, as implied by the Interhemispheric Interference Model (Figure 3), or whether the same results would be obtained when any two tasks are performed concurrently, including those mediated by a single hemisphere. The paradigm used was similar to Webster's (1986a) study involving one hand performing finger tapping while the other hand performed a concurrent stimulus-contingent knob turning task. In this case, however, participants performed a sequential finger tapping task with one hand while either the contralateral or ipsilateral foot pressed a pedal in response to a periodic onset of a tone . The fact that intrahemispheric conditions produced interference similar to interhemispheric conditions was contrary to predictions of the callosal gating hypothesis and indicated that the SMA may be susceptible to interference from any concurrent neural activity, not just that in the right hemisphere. More germane to the present point, however, was the fact that responding with the left foot interfered more with nonstutterers' finger tapping (with either hand) than did responding with the right foot. In light of the motoric simplicity of the interfering response, this finding was interpreted as implicating attentional factors whereby a response with the left foot required a rapid and transient switch in attention from the left hemisphere to the right. The fact that the interference on finger tapping was the same for responses with the right foot and the left foot in stutterers led again to the conclusion that stutterers lack a left hemisphere attentional bias and can switch hemispheric attention or engagement more readily than can nonstutterers.

The convergence of conclusions from the two studies with very different purposes and methodologies provides some basis for confidence that the hypothesized mechanisms reflect a principle of brain organization in stutterers, namely that "people who stutter (right- and left-handed) do not demonstrate a left hemisphere activation bias but are similar to fluent left-handers by showing a distribution of hemispheric activation that is more equal and more labile" (Webster, 1997, pp. 128-129).

This hypothesized lack of activation bias in stutterers is consistent with the results of many experiments of perceptual asymmetries in people who stutter. The performance of stutterers on dichotic listening tasks (e.g., Blood, 1985; Brady & Berson, 1975; Curry & Gregory, 1969; Rosenfield & Goodglass, 1980) and tachistoscopic half-field recognition tasks (e.g., Johannsen & Victor, 1986; Moore, 1976) indicate that ear or visual field asymmetries, respectively, are attenuated and more variable than those found in normally fluent speakers. The present discussion suggests further that the appropriate interpretation of these differences is not that they reflect differences in stutterers' and fluent speakers' hemispheric specialization but instead differences in their hemispheric activation biases.

The significance of this third element is that it provides a mechanism for understanding relative periodic over-activation of the right hemisphere in stutterers, an idea which has attracted considerable interest over the years (Boberg, Yeudall, Schlopflocher, & Bo-Lassen, 1983; Moore, 1993; Moore & Haynes, 1980; Rastatter & Dell, 1987) and which continues to do so in the context of recent PET scan studies (Braun et al., 1996; Fox et al., 1996; Kroll et al., 1997). What is perhaps

most original in this third element is the idea that over-activation contributes to stuttering by being a source of interference with left hemisphere processes. Although the basis for this interference may include anomalous processing of linguistic information by the right hemisphere (Moore, 1993; Rastatter & Dell, 1987) or compensatory processes with respect to stuttered speech (Braun et al., 1996), another possible basis, having substantial clinical significance, may be related to emotions and behavioural approach/withdrawal.

Patterns of electrical activity recorded from the frontal cortical areas of the left and right hemispheres (Ahern & Schwartz, 1985; Davidson, 1984; Davidson & Fox, 1982; Fox & Davidson, 1988) suggest there is an association between left hemisphere activation and the experience of positive emotions and behavioural approach, and between right hemisphere activation and the experience of negative emotions (like fear, anxiety, and apprehension) and behavioural withdrawal. This leads to the suggestion (Webster, 1993) that the apprehension and behavioural avoidance tendencies associated with stuttering affect the speech of people who stutter by evoking (or being associated with) right hemisphere activation, and this activation, in turn, interferes with the left hemisphere SMA. Such interference results in increased dysfluency which in turn confirms the fear and avoidance of the social and speaking situation. From this perspective, there is a positive feedback loop involving both neurology and psychology underlying stuttering, one implication of which is that successful rreatment or management of stuttering requires severing this feedback loop. Another implication, and one that is entirely speculative but which could have significant clinical implications, is that the hemispheric activation anomaly associated with stuttering may make the person who stutters (as well as the left-handed non-stutterer) more sensitive to negative reinforcement and more prone to withdrawal and avoidance responses than the right-handed fluent speaker who has a left hemisphere activation bias.

Developmental Issues

Forster (1996) recently examined the independence of the two major factors in the model, a fragile SMA and a lack of normal left hemisphere activation bias, and in so doing explored possible mechanisms for recovery from stuttering. Briefly, he compared the performance of (a) adults who stutter, (b) adults who reported having once stuttered but no longer do so ("ex-stutterers"), and, (c) adults who reported never having stuttered on two sets of behavioural tasks. One set comprised motor control tasks (sequence reproduction, bimanual coordination) intended to assess the integrity of the SMA; the other involved visual half-field tasks (lexical decision, dot enumeration) intended to detect hemispheric activation biases.

Following from the hypothesis that spontaneous recovery from stuttering is associated with maturation of the speech-motor control system, in particular the supplementary motor area, it was predicted that nonstutterers and ex-stutterers would perform similarly on the motor control tasks and do better than stutterers. On the visual half-field tasks, it was expected that the stutterers and ex-stutterers would show similarly reduced, or possibly even reversed, perceptual asymmetries compared to normally fluent speakers, a finding that would be consistent with the idea of an enduring anomaly in hemispheric activation. Both hypotheses were confirmed, indicating that both the SMA and hemispheric activation factors are necessary but neither is sufficient for stuttering to develop. The results further indicated that these two factors are independent: Compared to normally fluent speakers, stutterers showed impairments on both the motor tasks and on the hemispheric activation tasks, whereas ex-stutterers showed impairments only on the hemispheric activation tasks. (Presumably left-handed nonstutterers would show this same pattern of anomalies, but that remains to be tested). The data also bear, in an obvious manner, on the question of mechanisms underlying recovery from stuttering, a matter to be discussed at greater length by Forster and Webster (1998).

Implications for the Treatment and Management of Stuttering

A biological basis for stuttering and the perspective afforded by this model of neural mechanisms lead to a number of general points related to treatment issues. Only three will be touched on here, leaving a goodly number of others for later discussion: (a) The model supports contemporary approaches to the clinical management of stuttering; (b) whether treatment actually can or does actually alter brain function; and, (c) whether a biology of stuttering necessarily means an inevitability of stuttering.

Paralleling the two brain anomalies that are hypothesized to underlie stuttering, an inefficient and fragile supplementary motor area and a lack of hemispheric acrivation bias, are the approaches that characterize most contemporary treatment programs: (a) Modification of speech or stuttering behaviour, and (b) modification of attitudes and responses related to apprehension and avoidance of social and speaking situations that are an integral part of the stuttering experience and which, if not dealt with, interferes with using fluency skills in everyday speaking situations.

At the risk of oversimplification, approaches to the treatment of stuttering fall into two general categories (Guitar & Peters, 1980). The first approach, stuttering modification therapy (e.g., Sheehan, 1970; Van Riper, 1973) attempts to modify the form of stuttering behaviour by having the client

learn to use easy repetitions, accept easy stuttering, and control speaking fear and avoidance behaviour. Although dysfluencies may continue to occur in the speech, they come to interfere less with the flow of communication, and the person deliberately seeks out and enters new speech situations so as to minimize the impact of stuttering on his/her life. The second approach, fluency shaping, attempts to reconstruct a fluent manner of speech through acquisition and use of fluency skills related to breathing, voice initiation, articulation, and speaking rate (e.g., Webster, 1980). There is also an emphasis on the continued practice of those motor skills in various speaking contexts.

The two approaches are similar in that each includes the acquisition of speech motor skills of one form or another and the systematic practice of those skills outside the clinic. Each also includes, to some degree, the acquisition of cognitive/behavioural skills to help the client deal with negative attitudes and emotions related to speech difficulties. The two approaches differ, however, in their goals (easy stuttering vs. controlled fluent speech) and in the relative emphasis placed on the modification of attitudes and feelings. In fact fluency shaping often leaves their modification to occur adventitiously through speech success.

In describing how these approaches relate to the brain model illustrated in Figure 1, I am moving into the realm of speculation and conjecture. I like to imagine that the acquisition and practice of fluency skills counteract in some way the fragility of the left hemisphere speech motor control system. Specifically, I envisage the skills associated with controlled and deliberate speech motor movements having the effect of simplifying speech and bringing it within the capability of the inefficient speech motor control system. The deliberate use of these skills may also focus attention or neural activation on the fragile left hemisphere SMA and away from other potentially competing and interfering activities. This voluntary attentional process may also have a generalized effect on the left hemisphere that supports or facilitates the activation of that hemisphere's motivational system involved in approaching and entering social and speaking situations.

As I suggested earlier, the second component of most stuttering therapy involves dealing with the negative attitudes and emotions surrounding stuttering. Different programs and therapists approach this issue differently, but fundamentally their common objective is to help clients to modify their self-talk so that it supports and facilitates entering rather than avoiding speaking situations, using fluency skills effectively rather than panicking at the thought of speaking, and focusing on the successes rather than the failures of those attempts. Clients also learn to use progressive relaxation to reduce tension and deal with the ineffective self-talk that can arise from the experience of tension, and to attack avoidance behaviours systematically. Continuing to be speculative, we like to believe that the impact of changing emotional reactions and countering the tendency to avoid and withdraw from social situations is to reduce right hemisphere activation and hence reduce or control a source of interference with the speech motor control system. I would emphasize, however, that while clinical experience would suggest that the successful use of cognitive/behavioural skills certainly appears to facilitate fluency, whether it does so through altering brain function in the manner I have been suggesting remains to be seen.

The issue of the relationship of brain function to therapy components was of real interest to the late Professor Boberg who, together with his colleagues (Boberg et al., 1983), reported that an anomalous pattern of right hemisphere EEG activation observed in pretherapy stuttering clients was altered following therapy to a more normal pattern of left hemisphere activation. More recent and powerful brain imaging studies involving PET scans of stutterers have also reported changes in brain activation going hand in hand with improved fluency resulting from either the use of fluency inducing techniques like choral reading (Fox et al., 1993) or fluency shaping therapy (Kroll et al., 1997). Various methodological and conceptual issues cloud the appropriate interpretation of all brain imaging research findings (e.g., does a particular change in brain activation reflect a direct effect of treatment on the brain which in turn results in greater fluency, or does it reflect a consequence on the brain of improved fluency and reduced struggle and apprehension? In the particular experimental design, how appropriate is the linear additive model of information processing components that underlies the subtraction method whereby areas of altered brain activation are identified?) but there is certainly good reason to suppose that in principle brain activation can and will be affected by successful therapy.

Modern concepts of brain organization indicate clearly that not only is brain activity the origin of behaviour, thought, and feelings, but behaviour, thought, and feelings are themselves in part the origin of brain activity (Webster, 1997). When speech motor control processes are brought under voluntaty control through the deliberate and systematic use of stuttering modification or fluency shaping techniques, inevitably brain activation is being focused more in the left hemisphere motor systems, and probably the supplementary motor area in particular. And when cognitive/behavioural techniques are used to bring fears under control and to counter tendencies to avoid social and speaking situations, inevitably right hemisphere activation is being kept under control. As clients practice their skills and become more proficient in an ever broadening range of social and speaking situations, the skills become more automatic and require less concentration. Going hand in hand with

this, maintenance of the altered state of brain activation will also become more automatic. Although a neurological basis for stuttering implies a predisposition for stuttering and a likelihood that the core behaviours of stuttering will return if clients do not consistently practice their fluency and cognitive/behavioural skills in everyday situations, this interplay in principle between behavioural change and brain activation should provide clients with some confidence that stuttering is not as immutable as it might first seem.

Speaking anecdotally, I have found that the theoretical model of neural mechanisms outlined in Figure 1 seems also to have heuristic value at least among the better educated and reflective clients with whom I have worked. It helps them imagine what may be going on in their brains as they learn new speech and cognitive/behavioural skills in the clinic, as they encounter difficulties in using those skills in everyday life, as their fluency deteriorates and they start to panic, and as they use their skills deliberately to overcome difficulties. As well, it provides a rationale for learning in the first place, a reminder to use those skills in everyday situations, and a reasonable explanation they can provide when discussing with others their stuttering and therapy program. Presumably and hopefully there is some correspondence between the model and reality, at least on a conceptual level. In all of my experiences with the late Professor Boberg, this was the kind of issue that held a special fascination for him, and we dedicate its further exploration to his memory.

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