Commentary on *Iowa School and Neurologic Theories of Stuttering*

Commentaire sur Théorie de l'Iowa School et la théorie neurologique du bégaiement

Luc F. De Nil, Ph.D.
Graduate Department of Speech Pathology
University of Toronto, Toronto

After reading Hamre & Harn's paper, I found myself basically in agreement with their premise that stuttering is best understood as a neuromotor deficiency affecting speech fluency. I also fully agree that it is important for clinicians to be aware of their own, often implicitly held, theoretical position on the nature of stuttering and how this position influences their clinical decision-making. Why then, despite all these basic agreements, was I left with a nagging uneasy feeling about the content of the article? The answer came to me one morning as I was struggling through a description of the λ-theory of human movement control (Latash, 1993) and realized that my uneasiness was triggered by Hamre and Harn's statement in the opening paragraph that "... stuttering may be a *simple* neural incoordination for speech." (p.149, italics added). I felt uneasy because, as anyone who has tried to follow and understand the literature on motor control will agree, "simple" is a word the brain does not seem to know, no matter whether it is coordinating "simple" single-joint limb movement or the multiple structures involved in speech production. Indeed, most, if not all, target-oriented motor behaviours are influenced by many different factors, including neural, cognitive, emotional, and environmental variables. Stuttering is not different. It is this interactionistic perspective, which currently seems to be widely accepted by researchers and clinicians in the area of stuttering, that I felt is missing in Hamre & Harn's paper.

In their efforts to present two contrasting approaches to the nature of stuttering in a "straightforward manner to be concise" (p.149), both viewpoints are presented in a greatly simplified black-and-white manner. Many of the so-called Iowa School researchers Hamre and Harn refer to in their article, recognize the possibility that neuro-biological variables play an important role in the onset and development of stuttering. For instance, Bloodstein (1993) writes: "...the best guess I can make about the cause of stuttering is that many, though perhaps not all, stutterers have a *hereditary something that has predisposed them* in some way to stutter and that the disorder is often, *though perhaps not always*, precipitated by circumstances that imbue the child with an expectation of failure at language, articulation or some other aspect of speech" (p. 177, italics added). Similarly, Kelly and Conture (1992) conclude from their study of speech interactions between young stutterers and their mothers, that stuttering can best be understood within a "...demands-capacity model in which variables *(i.e., child capacities and environmental demands)* interact in a bidirectional rather than unidirectional manner..." (p. 1265, comments in italics added). Similar interactionist viewpoints can be identified for most proponents of the so-called Neurologic Theory. For instance, McClean (1990) remarks in his review of the research on neuromotor involvement in stuttering that "...this is not to say that the etiology of stuttering ultimately will be understood *solely* in terms of the motor systems of the brain" (p. 64, italics added). Smith (1990) also concludes that stuttering needs to be viewed as a heterogeneous disorder in which "multiple risk factors" (p. 45) contribute to its etiology. While I do not argue with Hamre and Harn that some researchers or clinicians may differ in the relative importance given to the various variables thought to influence the onset and development of stuttering, there clearly does not seem to be a simple and straightforward way to divide them up into two opposite camps.

The same uneasy feeling that things are not always as clear-cut as one would wish for troubled me while reading Hamre and Harn's discussion of some of the clinical implications of the two theoretical positions. First of all, Hamre and Harn seem to imply in their article that, as long as one takes the position that stuttering is a neurologic disorder, defining and identifying stuttering really is an easy task. They go on to say that stuttering can best be defined as elemental repetitions or prolongations (or "oscillations or fixations"). It is unclear to me how this general and rather vague definition would help anyone to identify beyond doubt whether or not a young child displays stuttering. A review of the literature quickly shows that there is not one single disfluency type that categorically distinguishes young normal speaking from stuttering children (Colburn & Mysak, 1982; Johnson, et al., 1959; Yairi & Lewis, 1984). Whatever differences are found seem to be of a quantitative rather than a qualitative nature. That is, stuttering children typically experience *more* part-word repetitions and prolongations compared to normal speaking children. It is often left up to

the clinician to decide how much is too much. As long as we are unable to identify categorically distinct behaviours or conditions that differentiate between young normally disfluent and stuttering children, stuttering identification and differential assessment will remain difficult at times, especially in very young early stutterers.

Identification of stuttering is a lot easier once the child has developed clear signs of struggle and tension. But even then, it appears easier to answer the question "Is this child stuttering?" than it is to determine whether a given disfluency was a stuttering or a normal disfluency (Ingham, 1984; Kully & Boberg, 1988). It is precisely this inability to come up with categorically distinct behaviours that unequivocally identify stuttering that has led Onslow (1992) to propose a rational rather than an empirical solution. According to Onslow, the definition of stuttering that one wishes to use may vary depending on the context in which it is used, such as evaluation of in-clinic fluency levels versus maintenance of fluency, or professional communications versus public information. Moreover, is not the very fact that Onslow discusses the impact of false positive or negative identifications of stuttering, in itself an indication that early identification is not always an easy undertaking?

As long as our definition of stuttering is focused on the behavioral (i.e., types of disfluencies) characteristics of speakers, overlap between stutterers and nonstutterers will be present, and the question of whether stuttering grows out of normal disfluencies or is categorically different will remain largely unanswered. If we agree that part-word repetitions and prolongations are core characteristics of stuttering, what needs to be investigated is how these disfluencies in stuttering children differ qualitatively from similar ones observed in the speech of nonstuttering children. No such differences have yet been discovered. In my opinion, our definition of stuttering ultimately will need to go beyond the overt behavioral characteristics, and will need to point to underlying neuromotor variables affecting speech movement coordination and how they interact with other child-specific and environmental variables. Given our poor understanding of the neural processes involved in speech production (or, for that matter, even in "simple" single-joint arm movements), such a definition clearly will have to wait for now!

If one recognizes (as most researchers and clinicians seem to do) that stuttering onset and development are influenced by the interaction between a number of variables, both internal and external to the child's organism, a different perspective on the parents' role in stuttering onset may emerge. It appears from Hamre and Harn's discussion that they believe that under no circumstances do parents play a role in stuttering onset. However, even if stuttering is basically the result of a neural incoordination, as Hamre and Harn seem to believe, parents, or maybe more correctly, the child's environment may inadvertently help to create an environment in which this basic neural deficiency may or may not result in stuttering. Who, for instance, would argue against the view that parents can create an environment that is more or less optimal for the child's development of his or her innate cognitive skills, language abilities, or fine motor skills? Why then, can parents (or the environment) not have the same impact on a child's ability to develop the motor skills needed for fluent speech? A similar perspective has been described recently by Starkweather, Gottwald and Halfond (1990) within the framework of the capacity-demands hypothesis. This position is very different from "blaming" the parents for stuttering onset. As Starkweather, et al. (1990) have pointed out, even normal environmental demands may negatively affect a child's speech fluency development if they exceed the child's capacity for fluency. In contrast, parents may create an optimal environment in which a child who has an innate neural incoordination is able somehow to overcome or outgrow this deficiency and spontaneously continue to develop as a fluent speaker. It is not inconceivable that many children who spontaneously recover would fall under this category. It is precisely this interactive environment in which stuttering has its onset that allows us as clinicians to intervene in a child's stuttering development. As all clinicians who work with beginning stutterers know, early intervention that includes counselling parents on how to modify their child's speech environment can be a very effective strategy in helping the child to develop as a normally fluent speaker.

In conclusion, I think that the weight of the experimental and clinical evidence on stuttering fails to support a unidimensional view of stuttering as "one of the many neurologic disorders" (p.150), but rather leads one to assume an interactionistic perspective on the onset and development of stuttering in which many variables can contribute to the development or the recovery from this very complex speech disorder.

Address all correspondence to: Luc F. De Nil, Ph.D., Graduate Department of Speech Pathology, Tanz Neuroscience Building, University of Toronto, 6 Queen's Park Crescent West, Toronto, Ontario M5S 1A8.
Email: DENIL@MEDAC.MED.UTORONTO.CA

References

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

Colburn, N. & Mysak, E.D. (1982). Developmental disfluency and emerging grammar. I. Disfluency characteristics in early syntactic utterances. *Journal of Speech and Hearing Research, 25,* 414-420.

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

Johnson W. & Associates (1959). *The onset of stuttering. Research findings and implications.* Minneapolis: University of Minnesota Press.

Kelly, E. & Conture, E. (1992). Speaking rates, response time latencies, and interrupting behaviors of young stutterers, non-stutterers, and their mothers. *Journal of Speech and Hearing Research, 35,* 1256-1267.

Kully, D. & Boberg, E. (1988). An investigation of interclinic agreement in the identification of fluent and stuttered syllables. *Journal of Fluency Disorders, 13,* 309-318.

Latash, M.L. (1993). *Control of human movement.* Champaign, IL: Human Kinetics Publishers.

McClean, M. (1990). Neuromotor aspects of stuttering: Levels of impairment and disability. In *Research needs in stuttering: Roadblocks and future directions* (pp. 64-71). Rockville, MD: ASHA Reports 18.

Onslow, M. (1992). Identification of early stuttering: Issues and suggested strategies. *American Journal of Speech-Language Pathology, 1,* 21-27.

Smith, A. (1990). Factors in the etiology of stuttering. In *Research needs in stuttering: Roadblocks and future directions* (pp. 39-49). Rockville. MD: ASHA Reports 18.

Starkweather, C.W., Gottwald, S.R., & Halfond, M.M. (1990). *Stuttering prevention. A clinical method.* Englewood Cliffs, NJ: Prentice Hall.

Yairi, E. & Lewis, B. (1984). Disfluencies at the onset of stuttering. *Journal of Speech and Hearing Research, 27,* 154-159.

Reply to Commentary
Réponse au commentaire

Curt Hamre and William Harn

In his commentary, De Nil raises several concerns and some questions over the views expressed in "Iowa School and Neurologic Theories of Stuttering". His comments have identified for us some points in the original paper that are in need of elaboration or clarification. We are pleased to have the opportunity to respond.

First, De Nil objects to characterization of stuttering as a *simple* neural incoordination for speech. We recognize that stuttering occurs in contexts in which a multitude of factors impinge upon the speaker. Indeed, we have actively explored cognitive and linguistic factors in stuttering (Hamre, 1984; Hamre & Harn, 1993). However, we argue that not all variables that may potentially influence speech performance are equally viable explanations of stuttering. Furthermore, the search for etiology need not culminate in an amalgam of competing accounts (e.g., constitutional predisposition for expecting failure in talking). In a recent text on childhood language disorders, Nelson (1993) "encouraged the viewpoint that not all factors that influence the course of development in children with language disorders need necessarily to be viewed as causes of the disorder" (p. 150). We encourage a similar view with regard to stuttering. A useful analogy to stuttering may be found in epilepsy. While epileptic seizures may be triggered by exogenous factors (e.g., sounds possessing certain acoustic features; certain visual arrays), no one seriously doubts the essential neuropathologic nature of the disorder.

The search for a cause of specific language impairment in children has generated at least as much interest and debate in recent years as has occurred in stuttering. Laurence Leonard (1991) recently argued that "Many of these children may *simply* be limited in language ability in much the same way that others may be poor in musical, spatial, or bodily kinesthetic abilities" (p. 66, italics added). We do not interpret Leonard's comment as implying that language processes are simple or well understood. Our characterization of stuttering as neural incoordination for speech is not intended to trivialize the complexities of speech motor control. Our aim is to underscore the primacy of a neural basis for the disorder.

De Nil argues that researchers cannot be neatly divided into two opposite camps. We acknowledge the distinctions that exist across viewpoints of investigators and that many theories and hypotheses exist in strong and weak forms. Our aim is to illustrate the pervasive influence of Iowa School tenets in contemporary accounts of stuttering. It seems to us rather readily identifiable whether or not an author espouses any of the assumptions described in our article (e.g., implicates parental behavior as a causal or risk factor for stuttering, or finds hesitation phenomena and stuttering indistinguishable). A recent text by Peters & Guitar (1991) provides a case in point. In an early chapter, the authors seemingly reject Johnson's diagnosogenic theory, stating their support for the view that "...when their parents first become concerned, most children who stutter are disfluent in a way that is different from normal" (p.79). However, later in the text they offer clinical advice consistent with the Iowa School approach: "If the child is normally disfluent, but the parents are overly concerned, counselling may be directed at relieving this concern, so that the child does not become excessively concerned and thereby develop stuttering" (p. 163).

Next, De Nil points out that we seem to imply that as long as one takes the position that stuttering is a neurologic disorder, defining and identifying stuttering really is an easy task. In actuality, our view is that stuttering is readily identifiable irrespective of one's stance on etiology. In Onslow's

(1992) paper, cited in our original article and in De Nil's response, the author reaches the conclusion that "In all, there are grounds for a confident conclusion that early stuttered speech is readily perceptible to observers" (p. 23). Perhaps the most trenchant comment on the definition and identification of stuttering may be found in Wingate (1988):

> Stuttering has been identified and discussed at length for centuries, throughout which time, there is ample reason to believe, everybody has known what everyone else has been talking about. The efforts made to deny the distinctiveness of stuttering, a relatively recent preoccupation, have been a wasteful and futile pursuit. (pp. 8-9)

The mere existence of hundreds of published studies that include groups of "stutterers" and "non-stutterers" is not the least evidence for the identifiability of stuttering.

An apparently anomalous situation exists wherein the question "Is this child stuttering?" can be readily answered, but yet it is problematic to determine if a particular speech disruption constitutes stuttering. The search for a definition of stuttering has been conducted under a "criterial attribute" model of categorization in which investigators have attempted to delineate attributes of speech disruptions that are necessary and sufficient to assign exclusive membership in the categories of stuttering or normal disfluency (more accurately, hesitation phenomena). The failure to find criterial attributes unique to stuttering is frequently cited in support of various Iowa School assumptions. However, the criterial attribute model has been largely discredited (Lakoff, 1987) as a model of how individuals make categorical judgments. Most real-world phenomena, including stuttering, cannot be individually demarcated from similar phenomena by one or more 'necessary and sufficient' features. More plausible models have existed for a long time (e.g., prototype theory, Rosch, 1978; exemplar theory, Smith & Medin, 1981) which better accommodate the nature of the phenomena being categorized and the cognitive processes of the individual doing the categorizing.

Finally, De Nil observes that it appears from our discussion that we believe that under no circumstances do parents play a role in stuttering onset. He has it precisely right. The present authors' combined clinical experience totals over 42 years during which time neither author has ever found cause to suggest to a parent that the parent's behavior, inadvertent or not, may have contributed to a child's stuttering.

A half-century after its introduction, various forms of the diagnosogenic theory persist. The history of the search for the etiology of autism forms an interesting parallel to stuttering. Beginning in the 1940s, many theories emerged that inculpated parents as the principal causal agents in autism. This trend reached its apogee with the publication of Bettelheim's (1967) *The Empty Fortress*. However, the parent-as-cause hypothesis in autism was unable to survive the complete dearth of supporting evidence. A current summary concludes that "...as much evidence indicates that the behavior of children with autism causes their parents to act the way they do as vice versa. ...Most theorists now agree that, as opposed to causing their children's difficulties, the parents of children with autism often play significant positive roles in intervention programs" (Nelson, 1993). The final point is particularly relevant to the present discussion on stuttering: parents can play positive roles in intervention without having been contributing agents to the emergence of the disorder.

References

Bettelheim, B. (1967). *The empty fortress.* New York: Free Press.

Hamre, C. (1984). Stuttering as a cognitive-linguistic disorder. In Curlee, R. & Perkins, W. (Eds). *Nature and treatment of stuttering: New directions.* (pp. 237-259). San Diego: College-Hill Press.

Hamre, C. & Harn, W. (1993). Linguistic phenomenology in stuttering. In Dittmann, J. (Ed.). *Linguistic disorders and pathologies: An international handbook.* (pp. 870-876). Berlin: Walter de Gruyter.

Lakoff, G. (1987). *Women, fire, and dangerous things: What categories reveal about the mind.* Chicago: University of Chicago Press.

Leonard, L.B. (1991). Specific language impairment as a clinical category. *Language, Speech, and Hearing Services in Schools, 22,* 66-68.

Nelson, N.W. (1993). *Childhood language disorders in context: Infancy through adolescence.* New York: Merrill.

Onslow, M. (1992). Identification of early stuttering: Issues and suggested strategies. *American Journal of Speech-Language Pathology, 1,* 21-27.

Peters, T.J. & Guitar, B. (1991). *Stuttering: An integrated approach to its nature and treatment.* Baltimore: Williams & Wilkins.

Rosch, E. (1978). Principles of categorization. In Rosch, E. & Lloyd, B.B. (Eds.), *Cognition and categorization.* (pp. 27-47). Hillsdale, NJ: Erlbaum.

Smith, E.E. & Medin, D.L. (1981). *Categories and concepts.* Cambridge, MA: Harvard University Press.

Wingate, M.E. (1988). *The structure of stuttering: A psycholinguistic analysis.* Berlin: Spring-Verlag.