Iowa School and Neurologic Theories of Stuttering

Théorie de l'Iowa School et théorie neurologique du bégaiement

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Abstract

The popular Iowa School theory is contrasted to a neurologic explanation for stuttering onset and development. Ten Iowa School assumptions are identified, and the authors recommend alternative interpretations for each of these. The evidence supports a view that stuttering is due to neural incoordination and that direct clinical approaches are indicated as soon as the problem is identified.

Résumé

Le présent article compare la populaire théorie de l'Iowa School sur le bégaiement et son développement à l'explication neurologique. Dix hypothèses de la théorie de l'Iowa School ont été cernées et les auteurs recommandent une interprétation différente pour chacune d'elles. Les preuves existantes appuient le point de vue que le bégaiement résulte d'une incoordination nerveuse et que des approches cliniques directes sont indiquées dès l'identification du problème.

The Iowa School theory of stuttering contains a number of assumptions that have a direct impact on clinical management, especially parent counselling. We will specify those assumptions in a straightforward manner to be concise; that is, we understand that any given clinician may want to qualify these assumptions in various ways. Then, we will compare those assumptions to counterparts that derive from evidence that stuttering may be a simple neural incoordination for speech. This, then, is not a comprehensive review of the literature on these matters. Rather, our purpose is to cite a sample of current literature that seems to offer support for each perspective (Iowa School and Neurologic), and then to state simply the clinical ramifications of both.

Iowa School Theory

The Iowa School theory represents the most widely accepted explanation for stuttering onset and development (Hamre, 1992a,b; Duckworth, 1988). This view originated with Wendell Johnson in the 1930's at the University of Iowa, and agreement with its basic tenets can be found in the current literature (Bloodstein, 1993; Conture, 1990; Cooper & Cooper, 1991; Ham, 1990; Kelly & Conture, 1992; Perkins, Kent, & Curlee, 1991; and others). The crux of Iowa School theory is that all young children are disfluent, but only those who become anxious about their speech will develop stuttering. This anxiety is usually ministered by parental concern directed to these disfluencies – sometimes by a well-meaning clinician helping a child with an articulation problem (Bloodstein, 1993).

Neurologic Theory

A variety of findings have appeared suggesting that stuttering may be due to a basic incoordination for the rapid, complex adjustments required for fluent speech. Considerable evidence concerning regional cerebral blood flow abnormalities in stutterers has been reported by the Callier Center Team in Dallas. They recently suggest that "stuttering can be viewed as one symptom of a defective fluency-generating system. If this system is, indeed, widely represented in the CNS, then it is not surprising that we observe multiple sites of CNS abnormality in stutterers" (Watson, Pool, Devous, Freeman, & Finitzo, 1992).

A comprehensive review of neurolinguistic evidence on stuttering suggested to Wingate (1988) that "the converging lines of evidence indicate that the disorder reflects a special kind of neurologic dysfunction involving, at least as the principal focus, neuronal systems of the left prefrontal cortex and related subcortical structures."

McClean (1990) has provided an excellent review of findings on the more peripheral motor dynamics of stuttering, and his heuristic scheme for organizing the evidence is particularly helpful. First, McClean discussed *structural impairments* resulting in "acquired stuttering"; for example, cerebellar, basal ganglia, and cortical systems. Second, he presented *functional impairments* found in the

fluent speech of stutterers; for example, anomalies in the temporal pattern of stutterers' motor speech movements. Third, he discussed *physical events associated with stutters*; for example, tremors and co-contraction of antagonist laryngeal muscles. Among his cautious interpretations and research recommendations was a basic agreement with Ray Kent's observation that "the critical problem in stuttering relates to temporal coordination of multiple structures or muscle systems" (McClean, 1990).

In summary, whether the incoordination is central, peripheral, or both, several investigators are pursuing the thesis that the onset and development of stuttering may be guided by principles of neuropathology.

Stuttering Subgroups

We need to acknowledge that many investigators beyond those cited here interpret the physiologic evidence as indicating that there may be several sub-types of stuttering. For example, one might conjecture that some children are born with limited *capacities* (e.g., a fragile CNS), but actual stuttering will not appear unless untoward *demands* (e.g., parental pressure) tax those capacities. Borden (1990) suggested a different perspective on the current state of evidence and direction for heuristic research:

> Just as a headache can be a symptom of differing etiologies, it is tempting and facile to attribute stuttering to every theory that comes along and include them all in a multiple etiology explanation... In my view, the most productive use of research time would be to seek the core behavior of stuttering across all sub-types. Subgroupings of stutterers and of disfluency types tell us more about overlaid coping behaviors than they do about the disorder itself.

It is at least parsimonious to suggest that stuttering may be one of many childhood neurologic disorders in which symptoms persist for some but not others. In pediatric neurology it is recognized that epilepsy, Sydenham's chorea, and Tourette's syndrome follow a similar course (Daniel Hurst, M.D., personal communication). The latter is our preferred interpretation, and the following clinical implications are coherent with this perspective.

Clinical Implications

Table 1 contrasts Iowa School and Neurologic theories on ten issues of clinical relevance. An extensive review of the evidence pertinent to these issues can be found in Hamre (1992a,b) as they relate specifically to the "prevention" construct. Beyond that, our view is that right column assumptions are most compatible with the weight of scientific evidence.

Table 1. Assumptions derived from Iowa School and Neurologic theories of stuttering.

		Iowa School	Neurologic
1.	Identification of stuttering is	difficult	easy
2.	Defining stuttering is	difficult	easy
3.	Use of "disfluency" instead of "stuttering"	avoids negative label	confounds categories
4.	Stuttering	"grows out of" normal disfluency	& normal disfluency progress differently
5.	A "phase of stuttering" is seen in	all children	5 percent of children
6.	Simple (early) stuttering is likely to	become more severe	disappear
7.	Parental mismanagement	causes stuttering	is irrelevant to onset of stuttering
8.	Prevention of stuttering is	important	impossible
9.	Calling attention to stuttering is	harmful	helpful
10.	Clinicians should treat stuttering	cautiously	aggressively

1. Stuttering identification. Contrary to a key Iowa School assumption, misdiagnosis of stuttering occurs rarely, if at all (Hamre 1992a,b). Van Riper (1992) reports that this conforms to his clinical experience as well. It ought to be as easy for clinicians as it is for parents (Zebrowski & Conture, 1989) to recognize stuttering because "stuttering is one of the most conspicuous types of communication problems" (Weiss & Lillywhite, 1976). Onslow (1992) argued that "the problem of identification of early stuttering is one that, for the present, calls for a rational rather than an empirical solution." With an identification focus on salient features of speech, he pointed out that "clinicians may be confident that false positive identification will not cause significant harm to a child."

2. Stuttering definition. Considering the "ease of identification" evidence, any definitional confusion (Bloodstein, 1990) seems to reinforce Robert West's (1957) observation that "everyone but the expert knows what stuttering is." The fluency disruptions recognized as stuttering are Van Riper's "oscillations or fixations" of a posture, Wingate's elemental repetitions or prolongations, or the synonymous "clonic or tonic" stuttering. These are also the speech aberrations that "covert stutterers" seek to avoid.

3. Disfluency and stuttering. Johnson coined the word "disfluency" because he disliked the word "stuttering" (Van Riper, 1992). Many experimenters have asked listeners to judge whether certain events (e.g. phrase repetitions) are instances of "disfluency" or "stuttering". Finding that some subjects use both designators for a single event, it is often suggested that listeners are confused about the difference between "disfluency" and "stuttering".

However, there is a more parsimonious explanation, namely, that stuttering is a category but disfluency is not for these subjects. First, most subjects for these studies are not speech-language pathology students, and it is important to realize that the word "disfluency" is a neologism for them; it is not listed in any dictionary and, unlike "stuttering", it is not a category outside of this profession. Second, subjects likely believe that some things they hear will be "disfluencies" and, having no category for them, they try to cooperate by applying this designator to some events.

There is considerable evidence that stuttering is a well established category. See, for example, the findings of Zebrowski & Conture (1989) on the ease with which mothers identify stuttering. We recommend the use of the word "stuttering" instead of "disfluency" when counselling parents.

4. Stuttering "grows out of" normal disfluency. The literature on normal hesitation phenomena ("disfluency") reveals that human speech (young and old) is characterized by breaks in fluency. These are neither heard by listeners – nor are they a cause for alarm, even among "highly disfluent" children (Hamre, 1992a,b). Therefore, it is not clear how stuttering might "grow out of" normal disfluency, and Van Riper (1992a,b) said that he has found no evidence to support this idea. To the contrary, normal disfluency is an obligatory (often helpful) characteristic of human speech that continues throughout life. Stuttering progression is quite different. Most stuttering appears for a few months and then disappears, some remains unchanged, and stuttering becomes more severe for a minority of cases (Andrews, 1984; Yairi & Ambrose, 1992). The progress of stuttering corresponds to that of many childhood neurological disorders, and may represent maturational changes compatible with early neural plasticity.

5. A "phase of" stuttering. One of the most famous Iowa School notions about stuttering onset is that all children go through a phase of stuttering (or normal disfluency). It is clear from a large literature on hesitation phenomena (e.g. Goldman-Eisler, 1968) that disfluency is typical of normal speech. On the other hand, approximately 5% of children experience stuttering. The evidence seems to require that we abandon the "phase of" disfluency/stuttering idea.

6. Simple stuttering becomes more severe. In spite of considerable evidence to the contrary, it is still commonly said that stuttering tends to become more severe over time. Van Riper & Emerick (1984) said that this is "one of the essential evils of stuttering." After acknowledging some difficulty with this view, Ham (1990) said that "there is no question, or there should not be, that most stutterers develop in their stuttering." Actually, there has been evidence available for over a decade that this assumption ought to be discarded (Ingham, 1983; Wingate, 1976; Young, 1975). That is, most stutterers recover without treatment within a year, either "spontaneously" or via their own compensatory strategies.

7. Parental role in stuttering onset. Johnson's diagnosogenic theory launched a five decade search for parental variables that induce stuttering, and this search continues unrewarded. For example, Kelly & Conture's (1992) first sentence notes how common it is for the "...behaviors and attitudes of parents [to be considered] importantly related to the onset and development of stuttering," and many authors are cited. No matter how strong a belief this is, we need to examine it in the light of scientific evidence. To the authors' knowledge, there is no experimental support for this view (including the Kelly & Conture findings), but there is some evidence to the contrary. With respect to the "development" of stuttering, some 35 years ago Glasner & Rosenthal (1957) reported that *not one* of 153 stuttering children had become worse although most of their parents had actively intervened. The so-called "negative" suggestions (e.g., "slow down", "think before you speak") were evidently not harmful and may have been helpful — most of the children no longer stuttered.

The evidence that stuttering is due to neural incoordination carries the implication that parents can be absolved of guilt with respect to onset. Of course, after stuttering appears, it is likely that willing and able parents can assist in a treatment plan.

8. Stuttering prevention. While popular, claims about our ability to prevent stuttering need to be re-examined. In some of the literature, there is a recognition that we cannot prevent the appearance of the disorder ("primary" prevention), but that we ought to pursue "secondary" prevention — early identification and intervention to prevent a disorder from becoming chronic or more severe. Hamre (1992a,b) presented reasons why the secondary prevention construct is less applicable to *stuttering* than is the treatment construct. Moreover, some of the literature invokes the possibility of primary prevention based on the unfounded assumption (#4) that stuttering grows out of normal disfluency. The suggestion is that intervention with disfluent children can prevent stuttering; this logic is seriously flawed as per the previous discussion on assumptions 4, 5, and 6.

Another perspective is that, like other childhood neurological disorders, stuttering appears in 5% of children for reasons that are not well understood. Further, early treatment

seems wise because it is not currently possible to predict which children will overcome the disorder on their own.

9. Calling attention to stuttering. The Iowa School assumption is that calling attention to a child's disfluencies might make him anxious and induce struggle that becomes stuttering. Happily for clinicians and childhood stutterers, many modern treatment programs embrace the benefit of directly showing the client how to talk more easily (for example, Shine, 1984).

What about the question of parents' calling attention to stuttering? The authors recommend that clinicians discard the Iowa School advice that parents should avoid calling attention to stuttering. Evidently, much of the "calling attention to" is useful (cf. Glasner & Rosenthal), and so it might be wise to determine *how* this is done. After determining what is done "when Johnny stutters," it may be important to find out what the effect of this is on Johnny's speech. If the parents' "slow down" advice is usually followed by improved fluency, that advice might wisely be continued as one aspect of treatment.

10. Clinicians' fear of stuttering. Wingate (1971) discussed the fear that clinicians might experience if they believe Iowa School assumptions about the onset and development of stuttering (especially assumptions 4, 5, 6, 7, 9 above). The notion that clinicians might cause stuttering was most recently presented by Bloodstein (1993). He reiterated what he has said before — that a child with an articulation disorder might start stuttering *because* the clinician treats articulation.

Another perspective ought to be considered. Stuttering is often associated with other language system difficulties (Hamre & Harn, 1993), but there is no evidence that communicative pressure causes any of them. Clinicians can be as direct and aggressive in treating stuttering as they are when treating other speech and language problems.

Conclusions

A reasonable reaction to this paper is that it seems to treat stuttering as a neural incoordination without considering the whole person. Stuttering does involve considerable psychological hardship for many people, and children who are ridiculed can develop marked self-esteem damage. However, we would suggest that the last sentence applies broadly to all persons with communication disorders, and it may be proper to "normalize" stuttering in this sense. That is, stuttering is not remarkable as a disorder occurring in people who have unique circumstances, doubts, and needs. The panoply of voice and articulation problems, aphasias, dysarthrias....and stuttering occur in children and adults who require our clinical artistry and knowledge for managing individual needs.

Finally, the clinician is obligated to adopt a tentative position on stuttering etiology, if only because almost all parents ask for this information. The authors recommend a reply to the question framed as follows: "The weight of the most recent evidence suggests that Johnny stutters because of an incoordination for speech." One can elaborate for parents who reveal a sense of guilt about how they may have caused the stuttering. Of course, the question about etiology will be answered quite differently by clinicians who feel that "the weight of recent evidence" supports the Iowa School thesis.

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