The Impact of Family History on Second Formant Transition in Early Childhood Stuttering.

Azza A. Aziz and Rasha F. Safwat

Phoniatric unit, Faculty of Medicine, Cairo University.

ABSTRACT

Background and purpose of study: *Most previous studies have compared children with persistent stuttering to normally fluent children, and those who recovered early were not considered differentially. The need to develop a scientifically-based objective means for discriminating sub-groups of young children who stutter - soon after stuttering onset - presents a challenging task.* ***Subjects and methods:*** *In this study, comparisons were made among 8 stuttering children with a positive family history of stuttering (2 girls and 6 boys, mean age = 44 months), 8 stuttering children with a negative family history of stuttering (3 girls and 5 boys, mean age = 46 months) – both groups were within the first 12 months after stuttering onset, and 8 age and gender-matched normally fluent control children. All the children repeated target words within a carrier phrase after the examiner to elicit fluent utterances. The second formant (F2) transitions of the initial consonant-vowel segment of the target words were acoustically analyzed to determine durational and frequency changes among the three comparison groups.* ***Results:*** *Findings revealed insignificant differences in the duration of F2 transition among the three comparison groups, whereas stuttering children with a positive family history showed significantly more restricted frequency changes of F2 transition than the other two groups.* ***Recommendations:*** *Further longitudinal studies of temporal and spatial changes of F2 transitions during fluent and disfluent speech of young stutterers may provide more objective criteria to structure the basis for a classification system that may be useful for early prediction of children who will continue to stutter and those who will exhibit natural recovery.* **(Int. J. Ch. Neuropsychiatry, 2005, 2(1): ??-??)**

INTRODUCTION

Both clinical and theoretical interest of stuttering as a disorder of speech motor control has led to numerous investigations in people who stutter, and has evaluated differences from non stuttering controls. The majority of these studies however, have been conducted with adult and school-age groups. Research with early childhood stuttering closely near to its onset has seen growing interest in internal subgroup comparisons. An important reason for this trend has been the phenomenon of natural or spontaneous recovery. Recent longitudinal studies supported high estimates of recovery that appeared to be at the level of 75-80%, much of which takes place during the first three years after the disorder begins. [1,2]

The phenomenon of spontaneous recovery gives rise to important questions about the nature of differences between children with persistent and recovered stuttering, whether they exhibit different speech and/or non speech characteristics even before developmental processes separate them? Developing data-based grounds for early prediction of the eventual course of the disorder will allow clinicians to make informed decisions about selective treatment strategies; to closely monitor for a period of time, or to start immediate clinical intervention. To develop these data, we have to discriminate the subgroups of stuttering early after stuttering onset, then to follow up patients in further longitudinal studies.

A surge of interest in early prediction of stuttering pathways has been seen during the past few years in a series of reports based on longitudinal investigations. The studies explored the possibilities of many risk factors as potentially predictive of persistence and recovery in stuttering; disfluency profiles,[1,3] language skills,[3,4] phonological skills,[5] and articulatory speech rate,[6,7] yet, familial inheritance of stuttering was considered to be the most consistent risk factor for occurrence and persistence of developmental stuttering.[8, 9]

The fact that stuttering runs in families has been documented over a long period and has led to speculations and researches about the role of a genetic component to this disorder.[3,9,10] New preliminary data also appeared to provide evidence that spontaneous recovery and chronicity were influenced by genetic factors, and that approximately half of all cases of persistent stuttering were accompanied with a positive family history.[8,11,12] Furthermore, results reported that recovery among females was more frequent than among males leading to the change in sex ratio from approximately 2:1 males to females close to onset of the disorder, to 4 or 5:1 in adulthood,[1] however, the results of Drayna et al.[11] revealed that persistent stuttering of non-genetic origin was largely a male disorder and may be related to a greater ability of females to overcome childhood stuttering, yet genetic stuttering, in contrast, affected males and females more equally. Generally, however, the review of incidence and twin studies, as well as of evidence for the various inheritance models, confirms previous conclusions about the interaction between genetic and environmental factors in stuttering.[9,10]

Amongst previous studies in stuttering, acoustic parameters were used as specific objective parameters by which stuttering children can be appraised.[13-15] One of increasingly importance acoustic parameters is formant transition. The significance of second formant transition (F2) received further support in recent years from the work of other investigators who used them to study coarticulation, and developing locus equations that predict the articulatory movement towards the stop sound.[16,17] Several reports demonstrated that adults exhibiting chronic stuttering tended to reveal appreciable variations in this acoustic parameter. Howell & Vause[18] found irregular or abnormal second formant transition (F2) variations in very high percentages of consonant-to-vowel junctions in either fluent or disfluent speech of adults who stutter. Klich & May[19] found limited F2 movement in fluency enhanced speech, and Robb & Blomgren[20] reported larger, faster transitions in fluent speech of stuttering than in non stuttering adults.

In children, Stomasta[21] defined the abnormal formant transitions, operationally, as a lack or aberrant second formant frequency changes as if following sounds were not anticipated and provided this as a basis for differentiating normal childhood disfluency form persistent stuttering. More recently, Yaruss & Conture,[22] Kowalcyzk & Yairi,[23] Chang el al.,[24] and Subramanian et al.,[25] studied several aspects of F2 transition in fluent and disfluent speech of young stutterers. They evidenced the presence of abnormal F2 transitions values between young children who stutter and normally fluent controls, and also between children with persistent and recovered stuttering.

This study aimed to investigate the potential use of second formant data in differentiating subgroups of young children- just beginning to stutter - according to the presence or absence of positive family history, and to compare those groups with the control non-stuttering children. Three questions were the concern of this study: 1- Are F2 transitions different for children who stutter and those who do not? 2- Are F2 transitions different among sub-groups of stuttering, based on genetic predisposition? 3- Does the extent of frequency change or duration contribute to differences in F2 transition?

SUBJECTS AND METHODS

24 children participated in this study categorized into three groups; 8 stuttering children with a positive family history of stuttering in 1st, 2nd, or 3rd degree relationship (2 girls and 6 boys, mean age = 44 months), 8 stuttering children with a negative family history of stuttering (3 girls and 5 boys, mean age = 46 months), and 8 age and gender-matched normally fluent control children.

The participated stuttering children were selected according to the following criteria: (a) under 5 years old at the time of the first visit, (b) first evaluation occurred no longer than 12 months after the onset of stuttering as reported by both parents. (c) judged by two staff members as exhibiting a stuttered speech that was rated as 2 or higher on a five-point stuttering severity scale (with 0 being "normal speech," 2 "mild" stuttering and 5 "very severe" stuttering), (d) no history of neurological disorders, and (e) non of the stuttering children received treatment prior to the recording of speech samples.

The present study focuses on F2 in perceptually fluent speech, a procedure that has been assumed to investigate the broader abnormality in the speech motor system of people who stutter. Each subject repeated three target Arabic words (*/bet/*, */tag/*, and */koral/*) within a carrier phrase following a model by an investigator, till the words sounded normal and natural. The initial consonant-vowel (CV) syllables of these words comprising either bilabial /b/, alveolar /t/, or velar /k/ consonants and number of vowels */e/*, */a/,* and */o/,* were used for acoustic analysis. Acoustic measurements were made from a wide-band (400 Hz) spectrogram display using the Computerized Speech Lab (CSL) Key model 4300. This display was selected because it enabled a visual representation of the formants and their transitions without the influence of the harmonics. First, the full word was displayed on the screen and then the target syllable was "zoomed in". The segment was visually and auditory verified to ensure that both the beginning and the ending of the transitions were included. Cursors were placed at the initiation and termination of the formant transition segment. The initiation of the transition (point a in Figure 1) was defined as the first glottal pulse following the release of the stop, Klich & May. [19] It was required that at this point both the first and the second formants be identifiable on the spectrographic display and that this point also correspond with the first peak of periodical spectral energy within the vowel on the time-intensity waveform as demonstrated by LPC splices at the points where the energy was strongest.

The end of the transition was defined as the first glottal pulse in which a steady state was identified in the following vowel (point b in Figure 1). When a steady state was not readily identifiable in the running speech, the first pulse in the region of unchanging frequency extended over three pulses was chosen. The onset and the offset frequencies of transition were measured using both the LPC spectrum and the spectrograph at the initiating and terminating points of F2 transitions, (points a, and b as specified above).

Because formant transitions depend on both the duration of the transition and the frequency change, two measures were made directly from the spectrogram: (1) - the extent of change in Hertz along the frequency domain between point a and point b, and (2) - the duration in millisecond along the time domain between the two points, as seen in (Figure 1).

To minimize individual bias in reading spectrograms, all measurements were performed jointly by two investigators to agree on the accuracy of both the initiating and terminating points of F2 transitions.

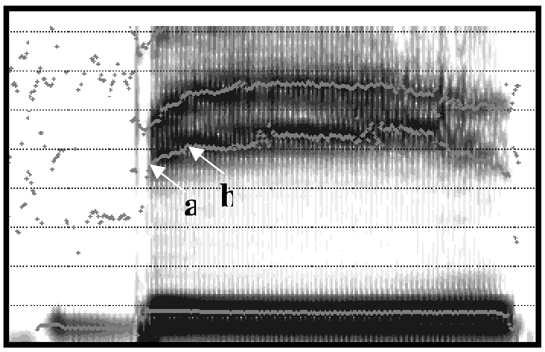

Figure (1): Spectrographic and LPC analysis of the Arabic word */bet/,* demonstrating the onset (a), and the offset (b) points of the second formant transition.

Statistical analysis:

The analyzed individual data were collected together in each of the three examined groups of children to yield mean values for frequency changes and duration of second formant transition averaged across the three places of articulation. Significance test for inter-group comparison was performed using One-way Analysis of Variance (ANOVA) test. The significance was set at *P* value < 0.05.

RESULTS

The statistical results of ANOVA test revealed insignificant changes in the duration of F2 transition between the group of children with positive family history of stuttering and those with the negative family history and the normally fluent control groups (*P* value > 0.05), also, there was insignificant changes between the group of children with negative family history and the normally control group, indicating that the three groups were nearly similar in the duration of their F2 transitions.

On the other hand, results of frequency changes between onset and offset frequencies of F2 transitions averaged across the three places of articulations revealed that stuttering children with positive family history produced significantly more restricted frequency changes than the other two groups, (*P* value < 0.05), while stuttering children with negative family history produced insignificant difference of frequency change relative to the control group (*P* value > 0.05), as seen in (Table 1).

Thus, the findings indicated that the changes in the frequency (spatial domain) of second formant transition were more relevant than the changes of duration (temporal domain); in differentiating between children with positive family history of stuttering and those with the negative family history and the normally control group.

Table (1): Means, (standard deviations), and significance of the three studied groups for frequency change and duration, (group no. = 8).

Measure	Stuttering children with positive family history.	Stuttering children with negative family history.	Normally fluent control children.
Duration (msec.)	52.50 (21.66)	54.87 (15.76)	**50.75 (16.09)**
Frequency change (Hz)	**400.25 (77.60)***	**473.37 (71.23)**	**499.62 (61.63)**

* Significant *P* value < 0.05 than the other two groups.

DISCUSSION

The need to develop a scientifically-based objective means for discriminating sub-groups of young children who stutter, soon after stuttering onset, presents a challenging task, important for structuring future prognostic instrument of stuttering. Past attempts at predicting persistent and recovered stuttering appeared to be greatly influenced by observation of older children that are more prone to become persistent, [26,27] whereas the most critical application for a prognostic instrument is at the early stage of stuttering.

It is important to study children with positive family history of stuttering and differentiate them from other children with early childhood stuttering. This study tested the hypothesis that those children who reported positive family history of stuttering- that may suggest a genetic background- present formant abnormalities from early on. This is particularly intriguing in light of reported genetic influence in the etiology of developmental stuttering, [3,9,10] and even in the persistence of stuttering among children, [8,11,12] also, in light of reported relevance of information regarding formant transition in people who stutter. [21-25]

Since stuttering is viewed as a disorder that involves difficulties with temporal programming and with executing complex articulatory movements or maintaining spatial organizations of the articulators, [15,28,29] therefore, this study examined variations in formant transitions among stutterers along both the temporal and frequency domains, which reflect articulatory dynamics and assess anticipatory coarticulation of speech as suggested by Lofqvist, [17] and Chang et al.. [24]

Three places of articulation (bilabial, alveolar, and velar) were chosen while examining the second formant transition of the target CV segments, and the mean values for frequency change and duration were averaged across them for all the studied groups. This was performed to overcome the expected variation in frequency change values across the various places of articulation as noticed in previous works of locus equation, [16,25] which indicated that frequency change was the greatest for bilabial sounds, smallest for the velar sound, and intermediate for the alveolar sounds.

The results of measurement of the duration of F2 transition revealed insignificant changes among the three comparison groups, in contrary to the previous findings of Robb & Blomgren, [20] who found larger, faster transitions in fluent speech of stuttering than non-stuttering adults. This was possibly due to the fact that the normal speech of young children is more variable than that of adults, with incomplete phonological mastery adding to the problem, resulting in large standard deviation values within each group. But still, the current finding regarding the duration of

F2 transition, agreed with the findings of Yaruss & Conture, [22] who found significant positive correlations between stuttered and fluent F2 transitions for all acoustic measures except for transition durations, which were also, insignificantly correlated for either high-risk or low-risk group for persistency of stuttering in children.

On the other hand, data of average frequency changes between F2 onset and offset frequencies revealed significant smaller frequency change in the stuttering group with positive family history than those with negative family history and the normal control group. Since F2 transitions represent the continued movement of the articulators either to realize the full vowel or to realize anticipatory coarticulation of the next sound(s), [16,17,21] this smaller frequency change may be interpreted to reflect restricted movement of the articulators on a spatial plane as suggested by Subramanian et al. [25] In other words, children with positive family history, that may suggest a genetic predisposition of stuttering, may have difficulty with transitions and/or blending across sounds resulting in undershooting their articulatory targets. This would appear to support previous speculations, [28,30] that the stutterer's difficulty is not with sounds but with transitions between them.

One point to reconsider is whether this restriction in the spatial plane of frequency changes represents a compensatory behavior to achieve fluency, a central programming characteristic, or a peripheral execution adjustment of stuttering children with genetic background?

First, because this study was conducted close to the onset of stuttering, the differences observed suggest the possibility that genetically-predisposed problems in speech programming or motor control are present at the formative stage of the disorder in children with positive history, and not as a compensatory behavior to induce fluency.

Second, these data being obtained from analysis of fluent speech appear to support the view that stuttering reflects a disorder that continually affects speech programming and execution and is not limited to the observed disfluent segments.

Thus in general, the direction of the present findings indicates that the frequency dimension of the formant transition, rather than the time dimension, is more significant contributor to the difference between children with positive family history of stuttering and those with the negative family history and the normally control group. These findings appear to complement the previous assertions of Stromasta, [21] and Van Riper, [28] and the recent results of Robb & Blomgren [20] on adults, and Chang et al. [24] on children, which indicated that a transition deficit is significant, perhaps central to stuttering; with drawing more attention to the importance of changes in the frequency domain of formant transitions as a parameter discriminating the possible subtypes of early childhood stuttering.

RECOMMENDATIONS

With establishment of more objective and more precise diagnostic criteria of formant transitions in both fluent and disfluent speech of young stutterers, it will be possible to structure the basis for a classification system of early childhood stuttering, possible to be used in future longitudinal studies for early prediction of children who will continue to stutter and those who will exhibit natural recovery..

REFERENCES

1. Yairi E & Ambrose N: Early childhood stuttering: Persistency and recovery rates. J Speech Hear Res. 1999; 42: 1097-1112.

2. Mansson H: Childhood stuttering: Incidence and development. J Fluency Disord. 2000; 25: 47-57.

3. Yairi E, Ambrose N, & Cox N: Genetics of stuttering: a critical review. J Speech Hear Res. 1996; 39: 771-84.

4. Watkins R, Yairi E, & Ambrose N: Early childhood stuttering III: Initial status of expressive language abilities. J Speech Hear Res. 1999; 42: 1125-35.

5. Paden E, Amborse N, & Yairi E: Phonological progress during the first 2 years of stuttering. J Speech Hear Res. 2002; 45:256-67.

6. Hall K, Amir O, & Yairi E: A longitudinal investigation of speaking rate in preschool children. J Speech Lang Hear Res. 1999; 42: 1367-77.

7. Kloth S, Kraaimaat F, Janssen P, & Brutten G: Persistence and remission of incipient stuttering among high-risk children. J Fluency Disord. 1999; 24: 253-65.

8. Ambrose N, Cox N, & Yairi E: The genetic basis of persistence and recovery in stuttering. J Speech Lang Hear Res. 1997; 40:567-80.

9. Felsenfeld S, Kirk K, Zhu G, Statham D, Neale M, Martin N: A study of the genetic and environmental etiology of stuttering in a selected twin sample. Behav Genet. 2000; 30: 359-66.

10. Buck S, Lees R, & Cook F: The influence of family history of stuttering on the onset of stuttering in young children. Folia Phoniatr Logop. 2002; 54: 117-24.

11. Drayna D, Kilshaw J, & Kelly J: The sex ratio in familial persistent stuttering. Am J Hum Genet. 1999; 65: 1473-5.

12. Viswanath N, Lee H, & Chakraborty R: Evidence for a major gene influence on persistent developmental stuttering. Hum Biol. 2004; 76: 401-12.

13. Howell P & Williams M: Acoustic analysis and perception of vowels in children's and teenagers' stuttered speech. J Acoust Soc Am. 1992; 91: 1697-706.

14. Hall K & Yairi E: Fundamental frequency, jitter, and shimmer in preschoolers who stutter. J Speech Hear. Res. 1993; 35: 1002-8.

15. Throneburg R & Yairi E: Temporal dynamics of repetitions during the early stage of childhood stuttering: an acoustic study. J Speech Hear Res. 1994; 37: 1067-75.

16. Sussman H, Bessell N, Dalston E, & Majors T: An investigation of stop place of articulation as a function of a syllable function; a locus equation perspective. J Acoust Soc Am.1997; 101: 2826-38.

17. Lofqvist A: Iterarticulator phasing locus equations and degree of coarticulation. J Acoust Soc Am. 1999; 106: 2022-30.

18. Howell P & Vause L: Acoustic analysis and perception of vowels in stuttered speech. J Acoust Soc Am. 1986; 79: 1571-9.

19. Klich R & May G: Spectrographic study of vowels in stutterers' fluent speech. J Speech Hear Res. 1982; 25: 364-70

20. Robb M & Blomgren M: Analysis of f2 transition of the speech of stutterers and non stutterers. J Fluency Disord. 1997; 22, 1-16.

21. Stromasta C: Elements of stuttering. 1986, Atsmorts Publishing. Oshtemo, Michigan.

22. Yaruss J & Conture E: F2 transitions during sound/syllable repetitions of children who stutter and predictions of stuttering chronicity. J Speech Hear Res. 1993; 36: 883-96.

23. Kowalcyzk P & Yairi E: Features of F2 transitions in fluent speech of children who stutter. A paper presented at the national convention of the American Speech-Language-Hearing association. 1995, Abstract published in Asha, 37, 79

24. Chang S, Ohde R, & Conture E: Coarticulation and formant transition rate in young children who stutter. J Speech Lang Hear Res. 2002; 45: 676-88.

25. Subramanian A, Yairi E, & Amir O: Second formant transitions in fluent speech of persistent and recovered preschool children who stutter. J Commun Disord. 2003; 36(1):59-75.

26. Riley G: Stuttering prediction instrument for young children, 1981. (Rev. ed.) Austin TX: Pro-Ed.

27. Cooper E, & Cooper C: Cooper personalized fluency control therapy handbook (Rev.) 1985. Allen, TX: DLM Teaching Resources.

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

29. Anderson J, & Conture E: Sentence-structure priming in young children who do and do not stutter. J Speech Lang Hear Res. 2004; 47: 552-71.

30. Zimmermann G: Articulatory dynamics of fluent utterances of stutterers and nonstutterers. J Speech Hear Res. 1980; 23: 95-107.