Stuttering Prevalence Among Patients at Risk for Velopharyngeal Inadequacy: A Preliminary Investigation

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The purpose of the present investigation was to provide preliminary information concerning the prevalence of stuttering among patients manifesting structural abnormalities of the velopharyngeal complex. Certain theoretical considerations suggested, *a priori*, that the prevalence of stuttering might be higher among these patients, although unsubstantiated clinical impressions indicated that the opposite was true. A retrospective study of 534 patients uncovered one individual whose speech was characterized by stuttering. This prevalence rate of 1.87 per 1,000 did not differ significantly from the 7 per 1,000 prevalence rate that would be expected in the general population. However, the lack of statistical significance may have resulted from the small sample size. Additional information from other craniofacial centers is needed to address this issue adequately. The theoretical implications of these initial findings are explored.

It has been suggested by Randall et al (1983) that successful management of a patient with an orofacial cleft is not fully realized until that individual manifests "unusually good speech with the free, easy, relaxed conversational speech of a normal child" (p 626). While such a definition is of limited usefulness when attempting to quantify the results of therapeutic intervention, it does highlight an important concept. To be normal, speech must be not only acoustically correct but also devoid of disruptions in fluency that call attention to themselves and interfere with overall communicative effectiveness.

A great deal is known about the communication skills of individuals born with orofacial clefts. However, an exhaustive search of the literature uncovered only three references to a possible relationship between stuttering and patients at risk for velopharyngeal impairment. The first is a reference provided by Schwartz (1976). In a book written for lay readers, Schwartz tells of a 4-year-old child with cleft palate who stuttered when wearing a pharyngeal extension appliance. The fact that this child did not stutter when the appliance was removed led Schwartz to pursue the idea that stuttering is related to reflexive laryngeal reactions to air pressure changes that occur in the vocal tract during speech production.

The second reference to stuttering problems among patients at risk for velopharyngeal impairment was made by Bloodstein (1981). In discussing possible causative agents, he suggested that stuttering may result from virtually any situation that causes a child to perceive speech as a laborious task that requires effort and care. In that context, Bloodstein reported the following observation: "clinical experience suggests that stuttering may sometimes develop as a reaction to cleft palate speech..." (p 329). He went on to say that instances of stuttering as a reaction to cleft palate speech "are not common, of course" (p 329). However, no quantitative data were provided to substantiate this suggestion.

Finally, Blood and Seider (1981) reported information obtained by surveying 650 speech-language pathologists selected from the 1978 ASHA directory. As part of the data obtained from the 358 responses received, they reported that cleft palate patients constituted 1 percent of the total caseload and that of these 11 patients, six (55 %) were receiving therapy that included treatment for stuttering.

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A number of investigations have reported the prevalence of stuttering in the general population (see Young, 1975 and Andrews et al, 1983 for reviews). Estimates vary anywhere from 2.7 percent in a population of 1,196 first- through third-grade children (Mills and Streit, 1942) to 0.6 percent in a sample of 5,138 college students (Sheehan and Martin, 1970). When all available information is considered, it appears that an overall prevalence rate of 0.7 percent in the general population is a reasonable estimate (Young, 1975).

In their scholarly review of the literature, Andrews et al (1983) note that "epilepsy, cerebral palsy and other neurological...(conditions are) associated with higher-than-expected prevalence of stuttering" (p 228). Excluding such obvious conditions, they imply that there is still considerable evidence to suggest that dysfluent speech may be causally related to subtle central nervous system dysfunction. Indeed, this premise seems to be the driving force behind much of the recent research concerning stuttering (e.g., Curlee and Perkins, 1984; Stromsta, 1986).

Although less attention has been paid to the possibility of central nervous system (CNS) dysfunction in the cleft palate population, there is some evidence to support the view that the behaviors manifested by this group of patients may not be wholly explicable in terms of aberrant oral morphology. For example, at least some individuals with cleft palate seem to manifest disturbances that are reflected in visual-spatial disabilities (Smith and McWilliams, 1968), visual processing deficits (Brennan and Cullinan, 1974), brain stem auditory processing impairments (Gould, 1980), sensory integrative dysfunction (Chapparo et al, 1981) and "associative language deficiencies" (Richman, 1980; Richman and Eliason, 1984). While no causal relationship has been demonstrated to date, subtle CNS differences in patients with cleft might not be completely unexpected since disturbances to neural crest cell development and migration may play a pathogenic role in clefting (e.g., Johnston, 1975; Vanlimborgh et al, 1983).

It is interesting to consider that the characteristics differentiating stutterers from nonstutterers (Andrews et al, 1983) are also characteristics that can be found among patients with clefts of the lip and palate (see Kuehn and Dalston, 1986 for a review). Of course, superficial similarities do not necessarily indicate common causality. Nevertheless, the indications of possible central nervous system involvement in at least some children with cleft palate are provocative, considering the thrust of much of the stuttering research in vogue today.

The prevalence of stuttering among patients with cleft palate also should be of interest, since a number of surveys have suggested a high concordance of stuttering with other communicative disorders (Williams and Silverman, 1968; Blood and Seider, 1981). Since patients with velopharyngeal impairments frequently experience communicative disorders, it would seem reasonable to assume that these individuals might be at increased risk for manifesting dysfluent speech. Indeed, as noted above, the survey information obtained by Blood and Seider (1981) suggested that 55 percent of the children with cleft palate who were receiving speech therapy services were being treated for stuttering in addition to other speech impairments.

Finally, the cleft palate population seems tailor-made to test many of the hypotheses concerning the genesis of stuttering. Parental concerns about communication skills, impaired middle ear function that may adversely affect auditory feedback, compensatory adjustments that complicate laryngeal control, and atypical behaviors directed toward maintaining normal air pressures during speech all are possible sequelae of this birth defect. Similarly, each of these factors has been implicated in the development of stuttering.

Notwithstanding the multiplicity of variables that might impact negatively upon the communication skills of patients with velopharyngeal impairment, our unsubstantiated clinical impression has been that there are remarkably few patients with cleft palate who stutter. Informal questioning by the authors revealed a similar impression among several other clinicians working in centers serving these patients. For this reason, a retrospective study was undertaken to determine the prevalence of stuttering among patients seen for evaluation at the University of North Carolina Oral-Facial and Communicative Disorders Program.

METHOD

Subjects

The subjects for this investigation were selected from the 973 patients seen for evaluation at the Oral-Facial and Communicative Disorders Clinic between August, 1979 and March, 1986. A wide variety of information has been collected on these individuals and stored in a computer database. The nature of the data collected and the mechanism for data storage have been described elsewhere (Dalston, 1983).

Selection of subjects for the present study was based upon the following criteria: each subject

had to be at least 3 years of age and had to manifest a structural abnormality that might be expected to affect velopharyngeal closure. All but one of the subjects who met these criteria were included for study. The one exception was a patient in whom stuttering onset occurred at 19 years of age. This patient manifested a complete cleft of the secondary palate and moderate mental retardation. At the time of his latest team evaluation in April, 1986, his stuttering pattern was reported to be mild and characterized by clonic syllable repetitions. Stuttering had not been observed in the past and was thought to have been precipitated by traumatic family events over the past year that included the death of his mother and remarriage of a noncommunicative father to a strict disciplinarian. This man currently is enrolled in a classroom for trainably mentally handicapped individuals, where he reportedly began showing signs of nervousness and frustration several months ago.

The final sample of 534 patients consisted of 273 males and 261 females. At the time of their last evaluation, the patients ranged in age from 3 years, 0 months to 66 years, 10 months. Of this total, 74 (13.9 percent) were between 3 years, 0 months and 5 years, 11 months; 205 (38.4 %) were between 6 years, 0 months and 12 years, 11 months; 166 (31.1 %) were between 13 years, 0 months and 17 years, 11 months; and 89 (16.6 percent) were 18 years of age or older.

The diagnostic categories and number of patients in each are presented in Table 1. With one possible exception, all of these diagnostic categories include a structural abnormality involving the secondary palate. The category labeled "velopharyngeal inadequacy without evidence of overt, submucous or occult submucous cleft" may or may not contain within it some individuals who had structural abnormalities of the palate that simply went undetected. By design, this category did include those patients who were judged to have a deep nasopharynx ("megapharynx") with or without concomitant cervical vertebra abnormalities.

Structural abnormality does not necessarily indicate the presence of velopharyngeal impairment, although referral for evaluation suggests that impairment may have been present at least initially. As shown in Table 2, pressure-flow testing at the time of their most recent team evaluation revealed that 63.6 percent of the subjects in the current investigation manifested adequate velopharyngeal closure as defined by that technique (Warren, 1975). Borderline adequacy was observed in 18.2 percent of the subjects, and 18.2 percent presented with velopharyngeal inadequacy. These figures do not necessarily reflect the extent of successful management by the team, since some of the patients were being seen for the first time.

Procedures

Each patient seen by the Oral-Facial and Communicative Disorders Clinic team was evaluated by one of four speech-language pathologists on at least one occasion. Patients seen in this clinic typically return for reevaluation every 2 years, although patients undergoing surgical treatment are seen much more frequently. For this reason, the number of times subjects in the present investigation had undergone evaluation of the sort described here ranged from one to seven.

During the course of each hour-long evaluation, the patient was required to speak in a variety of situations, ranging from single-word productions to spontaneous discourse. At the end of the evaluation, an assessment was made concerning the perceived fluency of the total speech sample. The clinician was required to specify whether the patient's speech fluency was normal, normal for age, or characteristic of stuttering. When present, stuttering severity was rated using a six-point equal-appearing interval scale. However, for the purpose of this study, stuttering was only recorded as present or absent. In accordance with Wingate (1964), stuttering was considered present if there was a "disruption in

TABLE 1 Primary Diagnosis of the 534 Subjects Employed in the Investigation

Diagnosis	*Frequency (N)*	*Percent*
Right unilateral complete cleft of the primary and secondary palate	52	9.7
Left unilateral complete cleft of the primary and secondary palate	108	20.2
Bilateral complete cleft of the primary and secondary palate	91	17.0
Cleft of the soft palate only	46	8.6
Cleft of the secondary palate involving the hard palate as well as the velum	90	17.0
Submucous cleft	48	9.0
Occult submucous cleft	29	5.4
Velopharyngeal inadequacy without evidence of overt, submucous or occult submucous cleft	39	7.3
Incomplete cleft involving both the primary and secondary palate	13	2.4
Pierre Robin sequence	18	3.4

TABLE 2 Velopharyngeal Adequacy Rating of 534 Patients when Evaluated at Their Most Recent Team Evaluation.*

Velopharyngeal Function	*Patients (%)*
Adequate	63.6
Borderline	18.2
Inadequate	18.2

* Velopharyngeal adequacy was assessed using the pressure-flow technique.

the fluency of verbal expression...characterized by involuntary, audible or silent, repetitions or prolongations in the utterance of...sounds, syllables, and worlds of one syllable'' (p 488).

Information is available concerning the interjudge reliability for some of the judgments made by the clinicians whose records were included for analysis here (e.g., Dalston and Warren, 1986). However, no rigorous assessment of intrajudge or interjudge reliability for judgments of stuttering was possible for this retrospective study. There is some evidence in the literature suggesting a lack of agreement among listeners on what constitutes an instance of stuttering (Curlee, 1981), but this was not considered to have any necessary bearing upon the global judgment of speaker fluency made by the listeners in this investigation.

In addition to clinician assessment, patient information was available from mail-home questionnaires, follow-up parent and patient interviews, and questionnaires filled out on each patient by the classroom teacher and the school speech-language specialist. The questions asked were intended to uncover the nature of past and present speech characteristics that were of concern to the patients, their parents, and school personnel. No pointed references were made to speech dysfluencies per se, but informants were encouraged to describe any and all perceived speech problems in considerable detail. With the exception of the one patient discussed below, problems with speech fluency were never reported by any of the informants involved in the present investigation.

Results And Discussion

Only one stutterer was found among the group of 534 patients included in this investigation. Close scrutiny of the findings obtained at the time of each patient's last evaluation and all previous clinic visits failed to uncover any other reports of stuttering in this group.

The one subject judged to be a stutterer was a youngster originally diagnosed as manifesting mild hypernasality and mild nasal emission in the absence of discernible palatal or pharyngeal abnormality. At the time of her last team evaluation, this 7-year-old girl presented with moderate stuttering characterized by sound and syllable repetitions with obvious tension and concomitant eye blinking. No definitive statement concerning velopharyngeal adequacy was attempted that day, because she had considerable nasal congestion due to a cold. Pressure-flow testing was not undertaken for the same reason. She was scheduled to return for further evaluation in 3 months. She did not return and was lost to follow-up in 1980 at which time she reportedly was receiving school-based stuttering therapy. At no time during the 5 years that this youngster was followed at this clinic did either parent acknowledge the occurrence of, or concern about, dysfluencies in their daughter's speech.

Given a theoretical prevalence rate of 0.7 percent, or 7 per 1,000, the expected number of stutterers in a group of 534 individuals drawn from the population at large would be 3.74. A simple Chi Square analysis failed to reveal a significant difference between the expected and observed frequency in this sample ($\chi^2 = 2.02$; df = 1; $p > 0.05$). However, given the low expected prevalence rate, it is mathematically impossible to attain a statistically significant χ^2 value with the sample size currently available for study at this clinic. Therefore, an increased sample size is needed to shed additional light on the possible relationship between stuttering and structural abnormalities of the velopharyngeal mechanism.

The most that can be said at this point is that there may be a trend toward a reduced prevalence of stuttering in the population of patients manifesting such abnormalities. At least there does not appear to be an increased risk for stuttering in this population. This finding is interesting when one considers the profile of patients in this group. Moreover, the possibility that stutterers may be underrepresented in this population could have important implications for understanding the etiology and appropriate treatment of dysfluent speakers.

Stuttering as a Learned Behavior

A number of authors have suggested that stuttering behavior is related to negative reactions experienced by the developing child (e.g., Johnson, 1959; Sheehan, 1975; Shames and Sherrick, 1963). To the extent that this is true, one might reasonably expect that at least some children with impaired velopharyngeal function would be exposed to adults with heightened sensitivites to communicative problems. Indeed, one of the questions most frequently asked by parents of children with cleft palate during the neonatal period is, "Will my child be able to speak normally?"

Parental concern regarding communicative interaction appears to persist well beyond the neonatal period. Long and Dalston (1982a; 1982b; 1983) compared the preverbal communicative skills of 10 infants with cleft lip and palate to those of 10 infants without clefts, matched on the basis of age, sex, socioeconomic status and dialect. The most striking difference between these two groups of 12-month-old children was the increased frequency of intrusive behavior by the mothers of the cleft youngsters. These parents frequently intruded upon the play of their children or attempted to redirect their attention with a resultant exhibition of refusal behavior by the child. Similar noncompliant behaviors have been observed in older preschool children (Lynch and Fox, 1985).

It is not known to what extent intrusive parental behaviors and noncompliant responses typify early parent-child interactions when the child has an orofacial cleft. Similarly, the effect such interactions may have upon linguistic and social development has not been determined. However, such behaviors are interesting in light of evidence presented by Brantley and Clifford (1979), suggesting that older children with orofacial clefts perceive themselves as being controlled by forces beyond their control.

From the limited information currently available, it seems reasonable to suggest that children with orofacial clefts probably develop in home environments that place at least as much importance upon successful communication as do the home environments of children in the general population. Of course, it could be argued that parents of children with structural abnormalities of the velopharyngeal mechanism actually may be more accepting of communication differences. Such accepting behavior, if it exists, might be due to early intervention counseling by speech and language pathologists. Clearly, additional information is needed concerning the social milieu that exists in the homes of cleft palate youngsters.

Stuttering as a Physiologic Impairment

The literature on stuttering is replete with references to possible physiologic bases for this speech impairment. Recent research in this area has addressed the respiratory (Baken et al, 1983), laryngeal (Adams et al, 1984; Conture et al, 1985), aerodynamic (Samur et al, 1986), articulatory (Zimmermann, 1980; Janssen et al, 1983; Stromsta, 1986), auditory (Moore and Haynes, 1980; Hannley and Dorman, 1982) and neuropsychological (Rosenfield, 1980; Boberg et al, 1983; Kent, 1983) characteristics of persons who stutter. Information stemming from this research increasingly suggests that stutterers exhibit some degree of dysfunction in one or more of these systems.

In addition, several current approaches to the treatment of stuttering seem to result in modification of one or more physiologic parameters when fluent speech is effected. Two examples may serve to illustrate the potentially intriguing relationship between the speech of patients with cleft palate and that of stutterers.

Samar et al (1986) recently studied certain aerodynamic phenomena in the speech of 15 adult stutterers before and after a concentrated program of therapy. They monitored four aerodynamic variables during the intervocalic interval of VCV sequences where C was the voiceless stop /p/. Among the four variables, they found that an increase in duration of the intraoral volume velocity rise interval following release of the /p/ contact appeared to be the most sensitive indicator of change following successful stuttering therapy.

An increase in the time during which airflow out the mouth is rising after plosive release of the /p/ should be highly correlated with an increase in the time during which air pressure within the mouth is dropping after release of the /p/ contact. For this reason, it is interesting to note preliminary evidence indicating that the intraoral pressure drop following release of the /p/ contact is prolonged considerably in cleft palate speakers when compared to a matched group of normal subjects (Warren et al, 1985). Moreover, this relationship appears to exist regardless of the adequacy of velopharyngeal closure in these patients. Comparing these admittedly preliminary data to those reported by Samar et al (1986), it is tempting to consider that individuals with palatal clefts may make compensatory adjustments during speech that reduce the likelihood that stuttering will occur.

On a more simplistic level, and perhaps directly related to the phenomenon discussed above, overall speech duration appears to be prolonged

among individuals with cleft palate who manifest hypernasal speech (Forner, 1983). The consequent reduction in overall speaking rate might facilitate fluent speech in these individuals just as it does among persons who stutter (Curlee, personal communication).

A second example that may help illustrate the potentially intriguing relationship between the speech of cleft palate patients and that of stutterers is based upon a recent report by Borden and her colleagues (1985). These investigators employed the electroglottograph (EGG) to study the acoustic waveforms of the first few glottal pulses of voicing in stuttered and fluent utterances produced by eight adult stutterers. In keeping with several traditional forms of therapy, the authors found that easy onset of voicing, evidenced by a gradual growth of the EGG envelope, was a successful strategy for initiating voice after a stuttering block. This kind of gradual onset of vocal fold vibration is the antithesis of laryngeal behavior expected among cleft palate speakers who evidence glottal stop substitutions in their speech.

To the extent that easy onset of vocal fold vibrations is associated with fluent utterances, glottal stop productions might conceivably create a physiologic environment conducive to disruptions in the normal flow of speech. If so, then the prevalence of stuttering might be expected to be higher among patients manifesting such substitutions. Since the number of cleft palate patients with glottal stop substitutions in their speech appears to be diminishing (see Kuehn and Dalston, in press, for a review), this hypothesis will be difficult to assess unless a number of clinics share information about their patients.

It is quite clear that the actual relationship between dysfluent speech and structural impairments of the velopharyngeal mechanism, and all the attendant consequences of such a relationship, cannot be answered by this preliminary study. Indeed, it is not the intent of this study to address these questions directly. The eventual goal of research of the sort reported here is to determine whether the cleft palate population represents a valuable resource to those interested in seeking information concerning the etiologic basis of stuttering and its treatment. Before this can be determined, however, clinicians working in craniofacial centers will need to provide information concerning the prevalence of stuttering among their patients. If the prevalence rate of 1 per 534 or 1.87 per 1,000 (0.2 %) found in this study proves to be characteristic of the cleft palate population in general, then this information should be shared with those whose primary clinical interest is in the area of dysfluency.

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