A Call for New Directions in Cleft Palate Speech Research

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Research and clinical evaluation of the speech disorder associated with cleft palate have certain distinct advantages and disadvantages when compared to research and clinical evaluation of disorders stemming from other etiologies. The major advantage is, of course, the obvious presence of the cleft—even though repaired—and the potential for inadequate velopharyngeal closure that results. The "fact" of the cleft is overwhelming and the seemingly straightforward relationship between velopharyngeal inadequacy and faulty consonant articulation is of paramount importance and cannot be disregarded. However, the degree to which this causes us to focus our research and clinical efforts on the velopharyngeal port, to the exclusion of other variables, represents a major disadvantage. This is a subtle disadvantage because it is attitudinal, yet it is significant because it manifests itself in our choice of research questions, our selection of research and clinical procedures, and our interpretation of research and clinical evaluation data.

The purpose of this paper is to suggest that variables other than the primary effects of velopharyngeal closure deserve a greater research emphasis. It is also a thesis of this paper that multivariate research designs and procedures that involve simultaneous measurement of multiple parameters are the preferred methods in studying speech associated with cleft palate.

A fair statement of what one reads most frequently in our literature about the causes of the speech problem associated with cleft palate would be something similar to the following. The disordered speech associated with cleft palate is primarily a function of velopharyngeal inadequacy with unspecified maturational factors involved to a lesser extent. Oral cavity deviations, hearing loss, psychosocial factors, etc., represent a small portion of the total variance and are only significant in individual cases.

It is not the validity of this statement that is questioned in this paper; we question the complacency that results from the inference that we have meaningfully described the speech disorder. Yes, velopharyngeal inadequacy is the primary influence on the speech problem, but how much of what we see in the speech behavior is directly related to velopharyngeal...

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patency? How much of the speech behavior is indirectly related to velopharyngeal inadequacy? How much is maturation a factor? What are the components of maturation and what are their individual contributions to the speech behavior? How large is the portion of the variance contributed by other factors, and more importantly, what is the nature of their contribution?

The existing literature provides us with a partial answer to the questions about the contribution of velopharyngeal inadequacy. The answer is only partial because the relationship between velopharyngeal closure and speech has been studied, for the most part, using a two-parameter model to describe a complex, interactive relationship. It appears that regardless of which parameters are used as coordinates—oral breath pressure ratio, area of the V-P port, millimeter gap between the velum and posterior pharyngeal wall, velopharyngeal resistance, etc., plotted against judged defectiveness of speech, nasality, number of articulation errors, intelligibility of consonants, etc. (select any pair)—the correlation ranges between .30 and .80, with an average somewhere around .50.

One way of interpreting these data is that on the average, velopharyngeal inadequacy accounts for about 25 percent of the total variance (the $r^2$). This leaves unaccounted-for approximately 75 percent of the variance. Refining the speech measure seems to increase the correlation, whereas refining the measure of velopharyngeal inadequacy does not. It is obvious that the influences of velopharyngeal inadequacy on speech are not described totally along a continuum of degree of oro-nasal coupling. Size of the opening probably relates directly to the primary effects on speech, but the primary effects explain only a portion of the total variance.

At this point, it might be helpful to distinguish between primary and secondary effects of velopharyngeal inadequacy. This distinction is important because it helps us explain the low correlation of .50 and points to an area about which we have very little information.

Primary effects of velopharyngeal inadequacy are the predictable aerodynamic and acoustic events directly related to increased oro-nasal coupling in the speech production system. Secondary effects would include all those events that are the system’s reaction to the primary effects and all other consequences of the velopharyngeal inadequacy observed in the speech product.

Simply listed, the primary effects would be: 1) the alterations in the acoustic spectral characteristics resulting from the additional cavity resonance ($5, 6$); 2) reduction in SPL of the speech wave, probably due to greater damping of acoustic energy in the nasal cavities ($1, 5, 10$); 3) nasal air flow ($\bar{V}_n$) which, when sufficiently turbulent, can serve as an audible noise source ($9, 11, 16, 17$); 4) reduced oropharyngeal air pressure for all non-nasal consonants, resulting in reduced potential for turbulent flow across articulatory constrictions ($9, 11, 16, 17$); 5) reduced transglottal pressure during voiceless stop production ($2$).
The net result on speech would be: 1) perceived nasal resonance on voiced phonemes; 2) greatly reduced acoustic energy of all oral consonants; and 3) audible nasal air flow on consonants sufficient to override the orally produced source.

Though some degree of each of these effects may be noted in the speaker with palatal insufficiency, the primary effects alone do not explain the variety of misarticulation patterns reported for this group. They are not sufficient to explain substitution errors, omission errors, glottal stops and pharyngeal fricatives, the high number of fricative and sibilant errors, duration differences in oral port constriction and altered prosodic features; indeed, the heterogeneous speech patterns of cleft palate children. These characteristics reflect in large part the reaction of the speaker and the speech system to the velopharyngeal patency: in other words, secondary effects of velopharyngeal inadequacy.

It should be obvious at this point that primary effects do not transfer directly into the speech product but are altered by the reaction (compensation) of the speaker and his system, so that what we observe is the interaction of primary and secondary effects. Because of their interactive nature and because of the confounding with maturation and other variables, one can not study secondary effects simply by subtracting the primary effects (we do not have a series of independent, additive terms). If we are to understand fully the contribution of velopharyngeal inadequacy to the cleft palate speech disorder, we must understand more of the secondary effects and the other contributors to the total speech variance. Much of our information about the primary effects of velopharyngeal coupling on speech is generalized from analog studies (10) or subjects in highly contrived experimental situations (1, 2, 12) in which substantial efforts were made to reduce or neutralize secondary influences. Since a portion of the secondary effects is directly observable in articulatory gestures, the several articulators must be allowed to vary naturally. This will require a much greater emphasis on multichannel recording than has been done in the past.

The advantages of multichannel recording in applied speech physiology are vast in comparison with the more usual single-parameter investigation, and are well within the present technology (7, 14). The greatest advantage is that the investigator can gather information simultaneously from different levels of the speech production system, as well as observe the interaction of several articulators. For example, an investigation of velopharyngeal influence on speech might include EMG recordings from levator, a strain gage apparatus to transduce velar movement (13), a magnetostor system to monitor tongue and/or mandibular movement (7), oropharyngeal pressure, nasal air flow, and the audio signal. With such a system, and through use of appropriately structured speech utterances, information would be available about the timing and magnitude of the neuromuscular signals to levator, the displacement and velocity of the
velum in relation to the tongue or jaw, and the influence of the structural movements on the aerodynamic pressures and flows. Rather than measure only EMG, or only pressure-flow, or structural movement, we should select among parameters that allow direct inference about speech production as it progresses from the neural signal, through the structural movement patterns and resultant pressure-flow changes, to the acoustic signal. These notions borrow heavily from the excellent clinical-research protocol developed by Dr. Ronald Netsell (14) for studying dysarthric subjects.

The physiological studies are easily accomplished in contrast to the equally important goal of determining the influences on speech of maturation, learning, and other factors such as hearing loss. It is important to remember that the cleft palate child must approximate normative speech during the acquisition stage with a faulty production system, and often with a fluctuating hearing level. Although most of us could generate a lengthy list of variables that might potentially affect speech and language development in the cleft palate child, we frankly have little information about anything other than velopharyngeal closure. We are just beginning to recognize, for example, that a minimal hearing loss can have a detrimental effect on language development (3, 8). Multivariate analysis procedures seem particularly well-suited to the task of defining relevant variables that contribute to the total speech product. Multiple correlation and factor analysis procedures (4) would be helpful in identifying which of the many possible maturational and psychosocial variables are significant for speech and language development.

Suppose, for example, that we wanted to test the hypothesis that some combination of experiences, abilities, physical traits, etc. characterizing the child prior to initial palatal repair determine the child's subsequent level of speech and language functioning. Multiple predictor variables and multiple criteria variables would be required to address these questions adequately. A canonical correlation procedure (4) is the appropriate statistic to study the interrelations between two sets of measurements.

Detailed analysis of the speech behavior for a group of subjects with cleft palate, perhaps along the lines of distinctive production features, in combination with significant maturational-psychosocial measures, may lead to meaningful subtyping within the cleft population. After all, we can cluster subjects as well as articulation scores. Subgroups that differ along behavioral dimensions such as an articulation profile may provide a more appropriate way to categorize individuals with cleft palate for research and clinical purposes than do categories such as cleft type, etc. Multiple-discriminant analysis and classification procedures (4) could and should be used to this end.

Finally, a word about the role of maturation in speech and language acquisition. With the increasing emphasis on early assessment and treatment of children with clefts, the ability to parcel out the developmental components from the structurally based components becomes crucial to
treatment planning. Currently, the only maturational criterion against which we evaluate speech is chronological age. Yet the age variability for normative speech sound development is very large (15) (and probably not an appropriate yardstick for children with structural anomalies, anyway). More precise maturational indices for speech must be developed. It seems reasonable that a prediction equation for speech development could be derived from the combination of properly weighted biological and behavioral measures; again, necessitating multivariate procedures.

Summary

This paper is basically a position paper on the importance of researching variables that influence speech development in children with cleft palates other than the primary effects of V-P inadequacy, and urges the use of multivariate research designs to accomplish that research. A premise is stated that primary (predictable aerodynamic consequences) effects of V-P inadequacy are greatly confounded with secondary effects (speaker's and his system's reactions to the V-P inadequacy), requiring multi-channel physiological recording and multivariate designs to determine the relative contributions of each to the speech product observed in the cleft child.

References