Unresolved Issues in Velopharyngeal Valving

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Although technology for the study and assessment of velopharyngeal function has advanced, we continue to classify that function in simplistic categories: closure, borderline, and no closure. Many questions remain unanswered. Information is needed about the dimensions of closure including depth and orifice configuration as they relate to speech. Under what circumstances does hypernasality involve sound transmission through hypoplastic tissue? Is the configuration of the nasopharynx important to hypernasality? Tension in the speech mechanism and also developmental factors deserve study. Nonsurgical variables that might enhance velopharyngeal function are poorly understood. These and other issues and the means of their investigation are worthy of discussion in our professional forums.

The development of instrumentation for assessing velopharyngeal (VP) valving has provided methodologies for studying the nature of the valve as well as for assessing its efficiency for clinical purposes. Technological advances in radiology have moved from cephalometrics and tomography to cine- (Moll, 1960) and videofluoroscopy (McWilliams and Girdany, 1964), from lateral, extended, and frontal views of the mechanism to the base view added by Skolnick (1970). Endoscopy has evolved from the panendoscope (Taub, 1966) to sophisticated endoscopes (Pigott, 1980), often coupled to videotape equipment.

Various aeromechanical devices have also been developed to measure nasal and oral pressures, to estimate the size of velopharyngeal openings, and to measure nasal airway resistance (Hixon et al, 1967; Warren and DuBois, 1964). Acoustic measures that correlate with perceived hypernasality have also been developed, tested, and used clinically (Fletcher, 1970; Stevens et al, 1975).

Electromyography has provided basic information about muscle activity during speech. The behavior of individual muscles and their dynamic interactions have helped to explain the movements observed by videofluoroscopy and endoscopy and to illuminate the sequence of events within the motor patterns (Lubker, 1968; Fritzell, 1969; Harris, 1970).

All of these techniques have helped us to understand how the VP valve functions for speech and to recognize that valving integrity is essential for normal speech. That knowledge alone has reduced the extent to which unwise and fruitless speech therapy is recommended to correct problems associated with VP incompetence and has led to our recognition of the validity of speech itself as an indicator of valvular integrity.

In spite of these advances in knowledge about VP valving, relatively simplistic views of the mechanism are still commonly held. We often think of closure as being either achieved or not achieved. When it is not achieved, consideration is given to borderline deficits and the possible benefits of
speech therapy. Recognition of more marked defects usually leads to surgical or prosthetic recommendations.

It is clear that there is not a one-to-one relationship between speech and VP deficiencies, especially among patients who have borderline mechanisms with deficits ranging in size up to 10 or 15 mm² (McWilliams et al, 1981). While such patients usually have mildly disordered speech, there are notable exceptions. A few patients appear, by all testing techniques, to have borderline mechanisms; but their speech symptoms, including degree of hypernasality, suggest greater deficiencies. On the other hand, there are occasional patients who demonstrate gross incompetence but whose speech is significantly better than would be predicted. It is obvious in both of these extreme cases that Curtis (1968) was accurate when he stated that the primary cause of excessive nasalization is inadequate VP function and that this fundamental truth is not likely to change by new discoveries concerning speech generation. However, it is also evident that speech is influenced by other factors as well. For lack of more attractive explanations, we have often attributed such variations to differences in learning. While learning undoubtedly plays a role that is as yet unspecified, other attributes of the vocal tract are also implicated. To date, we have devoted too little effort to unlocking these subtle intricacies.

Even when closure occurs on speech tasks, a fundamental question arises as to how speech is influenced by the manner in which VP closure is achieved. When closure is not achieved, the nature of the orifice and its differential effects on speech are of major concern.

More specifically, clinical experience suggests, and limited research evidence (McWilliams and Bradley, 1964, 1965) supports the contention, that we must be concerned with issues that have thus far received only scant attention. Among these are the vertical aspects of closure and how the tubal characteristics of VP valving influence speech.

Depth of closure is one feature. Speech appears to be less adequate as the vertical valving dimension shortens (McWilliams and Bradley, 1964). The vertical configuration, variation from one level to another, and the level at which major constriction occurs are all features which are of potential concern.

Another issue which must be considered is orifice configuration. We are not even in agreement about the size of orifice that is compatible with normal speech (McWilliams et al, 1981). Almost nothing is known about the importance of the shape of the orifice, although we have long suspected that it plays a vital role. Skolnick (1975) described a variety of closure patterns in both normal and cleft speakers but did not relate them to speech. Osberg and Witzel (1981) reported that subjects free of hypernasality achieved closure primarily by action of the velum in concert with the posterior pharyngeal wall with little lateral wall motion. Patients who were hypernasal, on the other hand, approximated closure primarily by action of the lateral pharyngeal walls. The authors hypothesized that the narrow, slit-like aperture which is seen when the velum is active is conducive to more acceptable speech than is a circular orifice of similar dimensions. Minifie (1973) discussed this issue, and his work suggested that the cleft-palate literature does not reflect all that is known about orifice shape as it relates to air and sound transmission. This question should be explored in relationship to a variety of orifice configurations and sizes. Until that is done, it is our impression that closure obtained primarily by the lateral pharyngeal walls is often not effective for adequate speech production.

Palatal bulk and the characteristics of palatopharyngeal tissues are other factors that sometimes appear to be implicated. Some patients close the VP valve during speech but have thin palates, even though there is no observable evidence of submucous clefts. These patients often have mild hypernasality and highlight the probability that the nature of valvular tissue and bulk plays a significant role in the transmission of sound. A thin velum provides
less impedance to sound than does a thick velum. Sound energy passes through tissue and is undoubtedly modified by its character. Learning more about these aspects of VP closure could add to our diagnostic armamentarium.

The architecture of surrounding structures may also be important in understanding the effects of varying degrees of VP incompetence. For example, we recently examined a boy with a very small midsaggital orifice during speech, but the speech was more hypernasal than would have been expected. Associated with this orifice of less than 10 mm² in size was a high, straight, superiorly vaulted nasopharynx. Would speech have been different had the nasopharynx been less capacious and the vault lower? These are issues that we have not been pursued that can be studied relatively easily and that may provide data useful in planning treatment and in defining the limitations of various forms of intervention in the presence of known valvular apertures.

We are aware that nasal resistance influences the quality of speech (Warren and Ryon, 1967; Warren et al, 1969) and that it can significantly change speech characteristics in the presence of VP valving deficits. We are less certain of the relationship between orifice size and air flow through the orifice on the one hand and the precise nature of nasal resistance on the other. How speech is affected by nasal resistance is undoubtedly influenced by the location of the resistance in the nasal pharynx and nasal passages. Resistance that keeps air flow and sound energy out of the nasal passages should be effective in reducing or eliminating hypernasality and nasal emission, whereas resistance in the anterior part of the airway seems likely to result in cul-de-sac resonance, which may or may not be accompanied by nasal emission. A similar degree of resistance in another part of the airway may affect speech differently, even though it may not alter measurable air flow. In some cases, both hyper- and hyponasality will be heard in the same speaker. Thus, the size, location, and configuration of the nasal resistance in association with varying VP apertures appear to have differential effects on speech. These attributes of nasal resistance are only infrequently taken into consideration when planning treatment. The nasal airway and the nasopharynx are important frontiers for future research.

Motor attributes of VP valving present additional questions. The strength of the VP seal varies from speaker to speaker, and evidence of this supports the contention that closure occurs on a continuum. Velar stretch, a phenomenon which is both age- and context-related and correlates with velar height (Mourino and Weinberg, 1975; Simpson and Austin, 1972; Simpson and Chin, 1981) presumably, in turn, affects the strength of the seal.

We know little about the developmental aspects of either velar stretch or lateral and posterior pharyngeal wall movement which includes Passavant's ridge. The limited data that are available suggest that the palate and the pharyngeal walls function in harmony but that there are vast differences in the relative contributions which these structures make to closure, even in normals. These variations account for the differences in configuration mentioned earlier. It would be useful to understand more about how these behaviors develop from infancy through adulthood. Such information might also shed new light on the troublesome timing problems that plague us all.

Tension in the vocal tract reflecting hyperfunctional behaviors also remains to be studied. These behaviors include changes in respiratory effort for speech production, the laryngeal compensations that often lead to vocal-fold nodules in subjects with borderline valving capacities, and the lingual alterations with which we are all familiar. The question is to what extent such maneuvers may be successful in masking hypernasality. Two teenage male patients are cases in point. Both demonstrated hyperfunctional voices and tension throughout the respiratory system including the larynx, pharynx, and soft palate. Both used clavicular breathing patterns. Neither was hypernasal until relaxation was achieved.
Then both demonstrated speech evi-
dences of VP incompetence. One had gross
incompetency. When he was asked to speak
in his habitual manner, the tension in the
system was immediately apparent, and the
soft palate looked like a drum head. These
cases are extreme, but they demand that
we explore vocal-tract tension as it relates
to varying degrees of VP incompetence and
to a variety of speech symptoms, including
hypernasality.

All of these questions pose complicated
problems of data collection, but they are
simple when compared to the intricacies
of investigating the interrelationships
among the structural and functional vari-
ations found in the system and how they,
in turn, interact with varying degrees of
VP competence and incompetence to in-
fluence the speech we are called upon to
assess and alter by surgery, prosthetics, or
speech therapy. While we know more to-
day than we did 10 years ago, we are not
making the progress that ought to be pos-
sible given today's technical advances.

There is still rampant disagreement as
to when speech therapy designed to elim-
nate mild degrees of hypernasality or to
improve VP valving is a viable option. It
is probable that therapy is not useful as
often as we might hope, but we lack the
information required to make sophisti-
cated predictions. In order to become more
effective in treating VP incompetence, we
must focus our efforts on learning more
about the VP valve as part of an inte-
grated vocal system, and we must deter-
mine the extent to which structural and
functional attributes of the whole system
work in concert with VP valving to create
speech variations. If we can do that, we will
be able to specify the limits beyond which
speech therapy cannot be effective and of-
er more accurate prognoses when other
forms of intervention are required.

I would be interested in sharing expe-
riences and in learning about new data that
may be available on these issues. Perhaps
discussions and forums in such publica-
tions as The Cleft Palate Journal will stim-
ulate research, provide answers to some of
our many questions, and result in im-
proved care for our patients.

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