Velopharyngeal Nomenclature: Incompetence, Inadequacy, Insufficiency, and Dysfunction

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Different usages of the terms velopharyngeal incompetence, velopharyngeal inadequacy, velopharyngeal insufficiency, and velopharyngeal dysfunction that are commonly found in the literature are reviewed. It is advocated that one should not attempt to use any of the terms to distinguish between neuromotor or structural causes for behavioral dysfunction. Although the terms can be used as synonyms, this is not always the case. When terms are used differently, it is important to make the specific usage clear from context.

KEY WORDS: *velopharyngeal incompetence, velopharyngeal inadequacy, velopharyngeal insufficiency, velopharyngeal dysfunction.*

Several investigators have noted ambiguity in the terminology used to describe velopharyngeal function during speech (Trost, 1981; Loney and Bloem, 1987). As discussed by Folkins (1985), "velopharyngeal incompetence" has been used with at least four meanings. If an individual is described as velopharyngeally incompetent, it implies one of the following: (1) the velopharyngeal structures cannot produce full closure of the port, (2) the velopharyngeal system is structurally inadequate for production of good speech, (3) the structure of the velopharyngeal system or its neuromotor control is inadequate for production of good speech, or (4) an individual's speech is perceived as showing characteristics associated with disorders of the velopharyngeal system.

In addition to concern for the potential confusion among the popular usages of "velopharyngeal incompetence," Loney and Bloem (1987) reported that there are multiple meanings in the literature for "velopharyngeal inadequacy" and "velopharyngeal insufficiency." They note that the three terms are often used interchangeably, but in other instances they are used with one term representing a general category of all velopharyngeal malfunctions and the other two as delimiting categories within the general term. The subcategories usually relate to different presumed causes of behavioral problems, such as limits on structure or neuromotor function. Unfortunately, there is not even consistency regarding which of the three terms is used to designate the general behavioral category and which are used to specify the underlying pathology. In fact, Loney and Bloem (1987) use a fourth term, "velopharyngeal dysfunction," as the general behavioral category that includes all types of presumed causes.

Kuehn and Dalston (in press) take the approach that we might apply the terms differently on the basis of etymologic perspectives. They state: "The term 'insufficiency' connotes an anatomic deficit, 'incompetency' suggests a physiological aberration, and 'inadequacy' could imply either an anatomic or physiologic problem or both." Such a system has the advantage that it provides a rationale for making distinctions, and knowledge of etymology may help one remember and distinguish among meanings. However, word meanings change as people use them differently, and therefore etymologic guidance may sometimes lead to inaccurate conclusions.

Loney and Bloem (1987) point out that "having three terms used in a redundant and contradictory manner hinders efficient scientific discourse, especially when the nomenclature is

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Preparation of this paper was aided by support from PHS Research Grants NS-07555-20 from the National Institute of Communicative Disorders and Stroke and DE-00853 from the National Institute of Dental Research.

used by many members of a multidisciplinary team.'' They suggest that everyone adopt the following usages of the terms:

Velopharyngeal insufficiency: Any malfunctioning that results in imperfect closure of the velopharyngeal apparatus. Velopharyngeal insufficiency includes both velopharyngeal incompetence and velopharyngeal inadequacy.

(a) *Velopharyngeal incompetence:* Imperfect closure of the velopharyngeal apparatus that is caused by a defect in neuromuscular functioning rather than a deficit of tissue.

(b) *Velopharyngeal inadequacy:* Imperfect closure of the velopharyngeal apparatus that is caused by a deficit of tissue.

I concur that the chances of misunderstanding are reduced when there is only one meaning for a scientific or clinical term. However, many words have more than one definition. If one is careful to make a single usage clear from the context, there will not be a confusion or a loss of information. Multiple meanings hinder discourse only to the extent that they require one to be careful that the context makes only one meaning possible.

My concern for the terminology of velopharyngeal disorders is not that multiple meanings lead to loss of information but that terms used in theoretical constructs must have referents that can be clearly specified and measured with operational definitions. There are many treatises on the role of word meaning in philosophical theory (Chappell, 1964), basic science (Kuhn, 1962), interpersonal perspectives (Hofstadter, 1985), and even clinical science (Johnson, 1946, 1956). One of the basic themes of this work on semantics is that our theories, and the data used to support them, are inextricably linked to the terminology used to express them. We are always extrapolating from an abstract concept to a quantitative measurement and then reversing the process by using measurements to define new abstract concepts. What appears to be an abstract concept on one level soon forms a framework for quantitative measurements within a larger theoretical perspective. Often, it is an appreciation for the assumptions inherent in moving between theory and measurement that allows us to enhance the power of our theories. Conversely, a failure to understand the consequences of leaping from a quantitative measurement to an abstract concept is often a barrier to effective development of theory.

Loney and Bloem (1987) review the literature and do an excellent job of developing definitions that fit commonly accepted theory. However, their definitions point out assumptions in common theory that may act to inhibit further insight. For example, they use concepts such as ''imperfect closure,'' ''a defect in neuromuscular functioning,'' and ''a deficit of tissue.'' They imply that one can define ''perfect'' and ''imperfect'' velopharyngeal closure and that there is a rationale to determine when the amount of velopharyngeal tissue is deficient and when it is just reduced. They imply that we can tell when neuromotor control is so limited in an individual that it is defective.

A second concern with the definitions proposed by Loney and Bloem (1987) is that they assume that an important diagnostic decision for patients manifesting velopharyngeal dysfunction is captured by a dichotomy between neuromotor causes and reduced tissue causes. As discussed by Folkins (1985), speech performance in many individuals is dependent on a variety of interacting motor and structural factors. Posing a gross diagnostic distinction between the individual with a structural problem and one with a neuromotor problem obscures the need to assess the many different factors determining how the neuromotor and structural processes interact in understanding the velopharyngeal behavior of each speaker.

A third concern is that the definitions cited earlier depend on quantitative measurements that we do not usually have. We do not typically measure the size and shape of the velopharyngeal structures, and there are no common systems for quantifying the ''amount of tissue.'' A tissue deficit might also depend on the location and composition of various structures. Similar issues could be raised with ''a defect in neuromuscular functioning.'' We do not have ways to measure neuromuscular processes of the velopharyngeal system that are independent of variation in anatomic structure. Even if we did, one might conceive of a situation in which there was poor neuromotor control, but the limits would still allow perceptually normal speech if there were a large amount of velopharyngeal tissue. In this case, there would be no neuromuscular defect. If, however, the same poor neuromuscular control were present in a speaker with an amount of tissue that was slightly, but not substantially, smaller than typical, and perceptually defective speech were present, would there be a tissue deficit or a neuromuscular defect?

Although it may be difficult to determine what is meant by ''imperfect closure'' (Folkins, 1985), it seems intuitive that one should be able to develop operational definitions to measure it. One could claim that imperfect closure means that tasks involving closure of the velopharyn-

geal port are performed with imperfect behavior. However, "imperfect closure" then becomes a synonym for the other terms for dysfunction. The list then includes "imperfect" as well as, "inadequate," "insufficient," and "incompetent." One has only added another label for velopharyngeal dysfunction rather than help to define it.

One could assume that "imperfect closure" does not mean that a value judgment about perfection or imperfection is made; instead, it means that maximum closure of the port is measured and the minimum areas are dichotomized into "perfect" (meaning complete closure is possible) and "imperfect" (meaning complete closure is not possible). Complete closure could be designated as any minimum area between 0 and 20 mm^2 (Warren and Devereau, 1966). However, in theory an individual could have velopharyngeal closure that is less than complete, but not a behavioral dysfunction; or alternatively, a speaker could be able to reach complete closure, yet still produce poor speech that is related to velopharyngeal dysfunction. As stated below, complete closure of the velopharyngeal port is not theoretically necessary in avoiding hypernasal speech and, conversely, the perceptual adequacy of speech is not related directly to the extent of velopharyngeal closure.

The filtering characteristics of the nasal tract are dependent not on the velopharyngeal port size but on the relative acoustic impedance of the nasal channel to the oral channel. Furthermore, for frequencies below 4,000 Hz, the overall cross-sectional size of the vocal tract, which determines its impedance does not determine the resonant frequencies. It is the variations in cross-sectional area that determine the resonant frequencies, not the absolute size of the constrictions. Fant (1960) shows the changes in formant patterns as extent of minimum oral constriction is varied. As long as variations in area are maintained in the mouth, there are many possible levels of oral impedance that may be usable during speech. A structural inability to reduce the nasal impedance can be counteracted by using relatively open gestures for the oral articulations.

This perspective was stated 20 years ago in a discussion of cleft palate speech by Curtis (1968). He reviewed theory and data from normal speakers showing that acceptable formant frequencies can be generated by a considerable variety of vocal tract configurations. Although Curtis admitted that he was speculating, he wrote: "It seems probable that cleft subjects may adjust their vocal cavity configurations in some manner which keeps the location of the formant resonances at or near their normal frequency placement." Evidence showing that such maneuvers are motorically possible is provided by Kent (1966), who used appliances to produce controlled circular orifices in the velopharyngeal port in six young subjects with cleft palate. After a practice period, some subjects showed no hypernasality for open vowels even at an orifice size of 113 mm^2. Kent (1966) used cinefluorography to document that the speakers systematically opened the lips, jaw, and tongue more in order to interact with the increases in velopharyngeal port size. However, at present we do not know the extent to which speakers with a repaired palatal cleft learn atypical movements that improve their speech (Karnell et al, 1985) or the factors that influence their ability to learn such maneuvers (Folkins, 1985).

It is sometimes stated that speakers who cannot come within 20 mm^2 of complete velopharyngeal closure are likely to have hypernasal speech (Warren and Devereux, 1966). However, if a speaker were able to learn (perhaps unusual) motor control strategies, allowing oral sounds to be made with relatively low lip and tongue positions, nasal resonance would not be too great even at velopharyngeal port openings well above 20 mm^2. If speakers with structures not allowing complete velopharyngeal closure, but closure just above 20 mm^2, produce perceptually inadequate speech, is this because of a tissue deficit or a neuromotor defect? Obviously, the poor speech is related to an interaction between structure and function and it is not useful to label either as a separate causal factor.

Theoretically, it is not necessary to produce closure of the velopharyngeal port to build up typical levels of intraoral air pressure during speech. One only needs to use the respiratory and laryngeal systems to increase air flow into the mouth to counter the air escaping through the nasal shunt. Unfortunately, as flow through the nose becomes very rapid, turbulence increases, and if turbulence increases enough, nasal emission will become perceptible—and audible nasal emission is perceptually aberrant. Again, the problem is not necessarily a structural inability to close the velopharyngeal port, it can also be considered a problem of making frication or plosion with nasal flows that avoid audible emission.

Repp (1982, 1983, 1984) and Parker et al (1986) have shown that many combinations of acoustic cues in synthesized speech provide similar perceptual responses. Specifically, Repp (1983, 1984) has shown that there are trading relations among the duration of silence, noise-burst duration, and noise-burst amplitude

in speech samples involving plosives. Because other acoustic variables can be manipulated, high levels of noise-burst amplitude, and thus high intraoral air pressures, are not necessary for perceptually adequate plosives. Although it is not known to what extent speakers employ such strategies, in theory the perceptual effects of low noise levels can be overcome by increasing the duration of the noise or of the silence prior to the noise burst.

If speakers who cannot reach reasonable closure of the velopharyngeal port do not produce good obstruents without audible nasal emission, the problem is again not necessarily due to a tissue deficit. One could just as easily say that their problem is an inability to develop neuromotor processes that achieve good speech with low oral air pressures. Evaluation of the interaction between structural and motor factors should play a central role in the assessment of speech. Again, it may be counterproductive to rely on scientific or diagnostic definitions that divide patients into discrete categories unless the criteria used in categorization have theoretical utility.

In conclusion, I feel that Loney and Bloem (1987) have helped to identify some of the confusion that may exist in the literature. In many cases, the terms velopharyngeal incompetence, velopharyngeal inadequacy, velopharyngeal insufficiency, and velopharyngeal dysfunction are used with different meanings, and ambiguities may be present in some contexts. However, it is not clear that we have the quantitative ability or the theoretical need to separate speakers with poor speech that is related to neuromotor causes from those with structural causes and to use these categories as distinctions among terms.

Perhaps the simplest approach would be to use the terms velopharyngeal incompetence, velopharyngeal inadequacy, velopharyngeal insufficiency, and even velopharyngeal dysfunction as synonyms. It is my preference that they be used for perceptual judgments about the adequacy of speech. However, any of the terms could be used as a substitute for describing anatomic or physiologic measurements as long as the context makes the specific measurement clear, and only one meaning is given to a word at a time. If different terms are used to specify general categories for distinguishing among individuals, it cannot be expected that such categories can substitute for measurement of the many different structural and neuromotor factors that interact during speech production for any individual member of this heterogeneous population of speakers with cleft palate.

Acknowledgment. I thank Virginia A. Hinton, David P. Kuehn, Jerald B. Moon, Hughlett L. Morris, and Duane R. Van Demark for reading and commenting on a preliminary draft of this manuscript.

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