

# Compensatory Speech Behaviors in Individuals with Cleft Palate: A Regulation/Control Phenomenon?

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**The articulation errors associated with velopharyngeal incompetence are of particular interest to clinicians and researchers because the problems they pose usually remain after surgical repair and the reasons they appear may have important implications for speech-motor control. The intriguing question of why individuals develop and maintain such detrimental behaviors has been the focus of our laboratory's attention over the past 25 years. The purpose of this paper is to present a hypothesis dealing with compensatory speech behaviors in cleft palate, provide some data in support of this hypothesis, and project future directions for research. The hypothesis, that speech aerodynamics conform to patterns characteristic of a regulating system, proposes that compensatory behaviors in cleft palate are manifestations of regulation and control strategies.**

Cleft palate speech is usually characterized by two major distortions, one of resonance and the other of articulation (McWilliams, 1954; Spriestersbach, 1955; Spriestersbach et al, 1961; VanDemark, 1979; Riski, 1979). Both relate to an inability to attain adequate velopharyngeal closure (Shelton et al, 1973; Pitzner and Morris 1966; Brooks et al, 1965; Shelton et al 1964). The articulation errors associated with velopharyngeal incompetence are of particular interest to clinicians and researchers because the problems they pose usually remain after surgical repair and the reasons they appear may have important implications for speech-motor control. It is paradoxical that although velopharyngeal incompetence stimulates compensatory speech behaviors, such responses tend to further undermine rather than enhance speech performance.

The intriguing question of why individuals develop and maintain such detrimental behaviors has been the focus of our laboratory's attention over the past 25 years. The purpose of this paper is to present a hypothesis dealing with compensatory speech behaviors in cleft palate, provide some data in support of this hypothesis, project future directions of our research, and possibly stimulate others to pursue similar lines of inquiry.

## BACKGROUND

Physiologists have long observed that the human body maintains a degree of constancy or "homeostasis" for its many systems. Circulation, respiration, acid-base balance, body temperature, and food intake are examples of bodily functions operating under rules that tend to preserve physiological balance. These systems have common features that are fundamental to homeostasis. The essential characteristics involve (1) regulation for the purpose of stability and (2) control mechanisms to achieve relatively steady state conditions (Brobeck, 1965).

For example, in the cardiovascular system, blood pressure is regulated while heart rate, stroke volume, and blood flow are controlled. A system is said to be regulated if structures respond to change and by their activity preserve some level of constancy. That is, the purpose of

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a regulating system is to maintain a certain parameter at a generally steady level. The control process is the means by which this is accomplished. The term control is sometimes used interchangeably with regulation, but there are valid reasons for distinguishing between the two terms. Control describes the process of management. Thus, in cardiovascular regulation, control of heart rate, stroke volume, and peripheral blood flow is necessary to maintain an appropriate amount of blood within the arterial system and, thereby, regulate blood pressure. It should also be noted that whenever the terms *control mechanisms*, *responses*, *behaviors*, or *strategies* are used, it implies that the brain receives information, processes it, and then directs the control activity.

Recent studies in our laboratory suggest that compensatory speech behaviors associated with cleft palate may be strategies developed for the purpose of satisfying the special requirements of a speech regulating system (Warren et al, 1980; Warren et al, 1981; Warren et al, 1984). Again, it is important to reiterate that this paper presents a hypothesis, replete with uncertainties, but certain to provoke discussion and, hopefully, interest. The hypothesis that speech conforms to patterns characteristic of a regulating system proposes that compensatory behaviors in cleft palate are manifestations of regulation and control strategies.

### SUPPORTING EVIDENCE

In a typical regulating system (e.g., cardiovascular, temperature, body fluids, respiration), a physiological parameter is said to be regulated if it remains relatively constant under different conditions. Response mechanisms or controls tend to keep such parameters constant. The respiratory system is especially relevant since speech production represents modified respiratory performance. Although the regulated parameter in breathing is probably CO<sub>2</sub> concentration, maintaining breathing pressures by controlling airway resistance and airflow influences gas exchange and provides a stable CO<sub>2</sub> environment.

There is evidence that resistance in the respiratory airway is controlled for breathing purposes (Remmers and Bartlett, 1977). During expiration, the nasal valve is about 10 percent smaller than during inspiration (Hairfield et al, 1986). The glottis adducts more strongly during the expiratory phase of breathing as well (England and Bartlett, 1982). The additional resistances during expiration serve as an expiratory brake, providing longer intervals for gas exchange at the alveoli (Gautier et al, 1973). Individuals

appear to perceive differences in airway resistance during breathing and respond accordingly. For example, we mouthbreathe when our nasal airway seems obstructed. Hinton et al, (1986) compared the nasal and oral pressures of normal subjects during breathing with pressures measured in subjects diagnosed as having impaired nasal airways. They found that breathing pressures in both groups are similar. Upper airway pressures are maintained at a fairly constant level by opening or closing the mouth.

Similarly, runners mouthbreathe because they perceive an increase in nasal resistance as airflow rate is increased. Since resistance is flow dependent, opening the mouth reduces nasal airflow and upper airway resistance.

Certain responses of the speech articulators are similar to those of the respiratory structures, and there appears to be control of resistances along the vocal tract during speech, somewhat paralleling the previously noted controls within the respiratory tract during breathing. The vocal tract is a tube containing oral, pharyngeal, and glottal structures capable of producing a variety of constrictions (Fig. 1). The production of speech requires a relatively constant subglottal pressure, which serves as the energy source (Ladefoged, 1962, 1968; Mead et al, 1968; Netsell, 1969). Intraoral pressures for consonants also appear to be fairly constant within subjects and seem to vary only according to consonant type or context (Brown and McGlone, 1969).

Specifically, for speech aerodynamics, subglottal pressure is kept relatively constant by checking or enhancing elastic forces and by compensating for sudden changes in respiratory load that occur with opening and closing of the upper airway. Precise control over the movement of upper airway structures and airflow tend to keep subglottal pressure regulated (Warren, 1982) (Fig. 2). Similar responses provide relatively stable intraoral pressures among consonants. Although position of an articulatory structure will vary according to phonetic context, overall resistance across the vocal tract remains fairly stable (Fig. 3). Thus, in normal speech, vocal fold resistance for a vowel is approximately

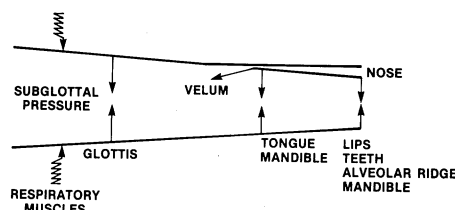


FIGURE 1 Vocal tract structures are capable of producing a variety of constrictions.

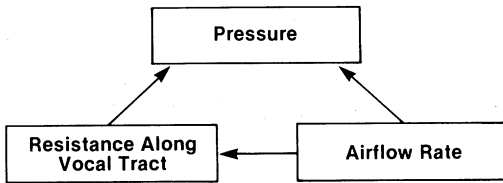


FIGURE 2 Pressure is regulated at an appropriate level by controlling movements of articulatory structures and airflow rate. Control over movements provides the necessary amount of resistance to maintain pressures.

40 to 80 cm H<sub>2</sub>O/L/S (Smitheran and Hixon, 1981; Warren, 1982) but only 25 cm H<sub>2</sub>O/L/S for voiced fricatives. However, oral port resistance for fricatives is approximately 75 cm H<sub>2</sub>O/L/S, thus providing an overall resistance within the airway similar in magnitude to that observed for vowels (Warren, 1982).

Another example is glottal resistance for voiceless fricatives and plosives. The resistance for voiceless fricatives is approximately 10 cm H<sub>2</sub>O/L/S compared to about 6 cm H<sub>2</sub>O/L/S for voiceless plosives. On the other hand, resistance is infinite for the plosive at the oral port and about 75 cm H<sub>2</sub>O/L/S for fricatives. Such interrelationships suggest that resistances of the speech structures may be synchronously controlled, in a manner analogous to respiratory control (Warren, 1982). A regulating system would prevent a rapid fall in subglottal pressure and provide flexible, local energy sources throughout the vocal tract. The important point is that the same structures that modify airway resistance during breathing are also used as articulatory structures during speech. Sequenced breathing behavior (England and Bartlett, 1982; Warren, Hairfield et al, 1985) presumably is programmed prior to birth and, therefore, is available for later use in speech.

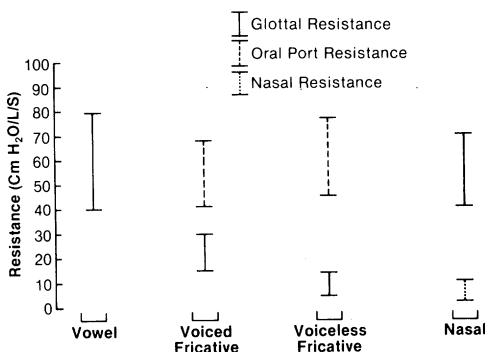


FIGURE 3 Resistance across the vocal tract is controlled to maintain pressures across consonant types. Approximate ranges are illustrated.

## SENSORS

In any regulating system, there must be a relationship among input, processing, and output. Mechanisms to detect or identify change are necessary components if control factors are to operate. The presence of sensory receptors must be established, their location identified, and their sensitivity determined. The physiological detection system does not have to respond to the variable being regulated; it can respond to some correlate or function related to it (Brobeck, 1965).

Respiratory receptors have been found in the trachea (Sant'Ambrogio, 1982), in the larynx (Sant'Ambrogio et al, 1983), and in the nasopharynx of man (McBride and Whitelaw, 1981). Intrathoracic receptors signal both volume and flow; extrathoracic receptors signal flow and upper airway resistance (Sant'Ambrogio, 1982). Similarly, laryngeal receptors sensing pressure, airflow, and contraction of muscles have also been described (Sant'Ambrogio, 1982). There is also evidence that muscles in the upper airway play a functional role in instantaneous control of airflow and compensation for changes in airway resistance during breathing (Cohen, 1975; Brouillette and Thach, 1980). Remmers and Bartlett (1977) observed a "tracking" behavior involving extrathoracic tracheal stretch receptors in which the respiratory muscles during expiration responded to compensate for changes in upper airway resistance. The expiratory discharge of the receptors was determined by the relationship between upper airway resistance and instantaneous flow. This provides information useful for coordinating the activity of expiratory flow controllers. If this afferent information were compared centrally with that derived from intrathoracic pulmonary receptors, the relative magnitude of upper airway resistance could be estimated. Such information might allow adjustment of laryngeal resistance in response to changes in supralaryngeal pressures. Recent studies by England and Bartlett (1982) demonstrate that the larynx activity controls respiratory flow in man by varying the degree of glottal adduction. Thus, there appears to be a detection system for breathing that may, in some fashion, operate in speech as well.

## CLEFT PALATE STUDIES: DEFINING THE MAGNITUDE OF ERROR

Individuals with velopharyngeal incompetence provide a unique opportunity to study the dynamics of a speech regulating system. Varying degrees of incompetence represent different mag-

nitudes of error. In our earlier studies we defined velopharyngeal function in physiological terms (Warren, 1979). A velar opening greater than  $0.2 \text{ cm}^2$  during non-nasal consonant productions is not adequate for normal speech (Warren, 1975). Individuals with an orifice opening of this magnitude may be unable to impound sufficient intraoral air pressure *unless* the nasal cavity is somewhat obstructed, respiratory effort is increased, or other strategies are employed. Similarly, nasal emission of air is usually excessive and audible, and resonance is hypernasal. An incompetence of this magnitude represents a significant error in a regulating system.

According to our physiologic definition, competence has a limit which is below  $0.2 \text{ cm}^2$ . Except in extremely rare instances, when the opening is less than  $0.05 \text{ cm}^2$ , voice quality is within normal limits, and any nasal emission present is inaudible. Speech performance is determined by accuracy of articulation rather than by palatal closure. This magnitude of opening represents an insignificant error in most instances.

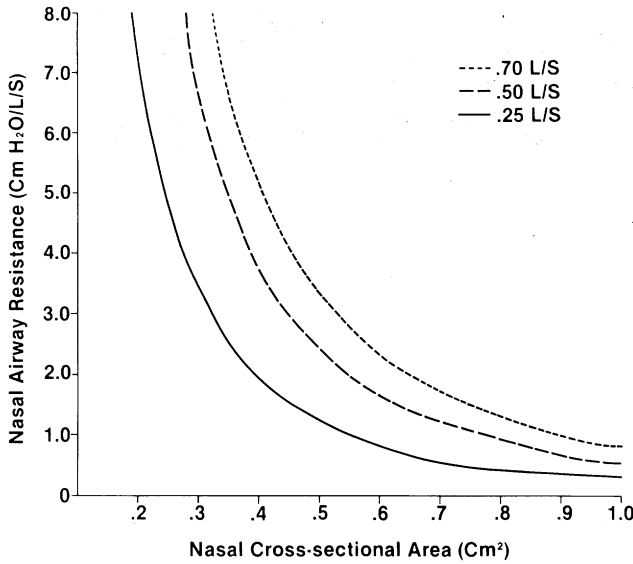
Openings between  $0.05$  and  $0.10 \text{ cm}^2$  are usually small enough not to interfere with an individual's ability to impound intraoral pressure. However, some nasal emission will occur, and it may be audible. If the nasal airway is obstructed, turbulence will produce audible airflow that will be most noticeable during fricative productions, since respiratory effort is increased (Warren, 1979). Resonance will be within normal limits or slightly hypernasal, providing articulatory performance is reasonably correct. However, there will be individuals who speak with an overclosed oral airway (i.e., teeth close together) who will sound moderately hypernasal as a result of this small degree of opening (Warren, 1979). Under these circumstances, oral airway impedance may be high enough to shunt a greater amount of acoustic energy into the nasal cavity, producing hypernasal speech. Thus, an error in this range, altogether small, may result in compensatory behaviors.

Openings between  $0.10$  and  $0.20 \text{ cm}^2$  represent borderline incompetence. Again, the term borderline represents a physiological determination based on the respiratory requirements of speech. Its acoustic analog may differ slightly. That is, in most instances, the listener will recognize slight to moderate audible nasal emission and hypernasality. However, there will be infrequent instances where speakers will sound normal in spite of borderline velopharyngeal inadequacy (Warren, 1979). Generally, an error of this magnitude is significant, and compensatory responses or stigmata of cleft palate speech would be expected.

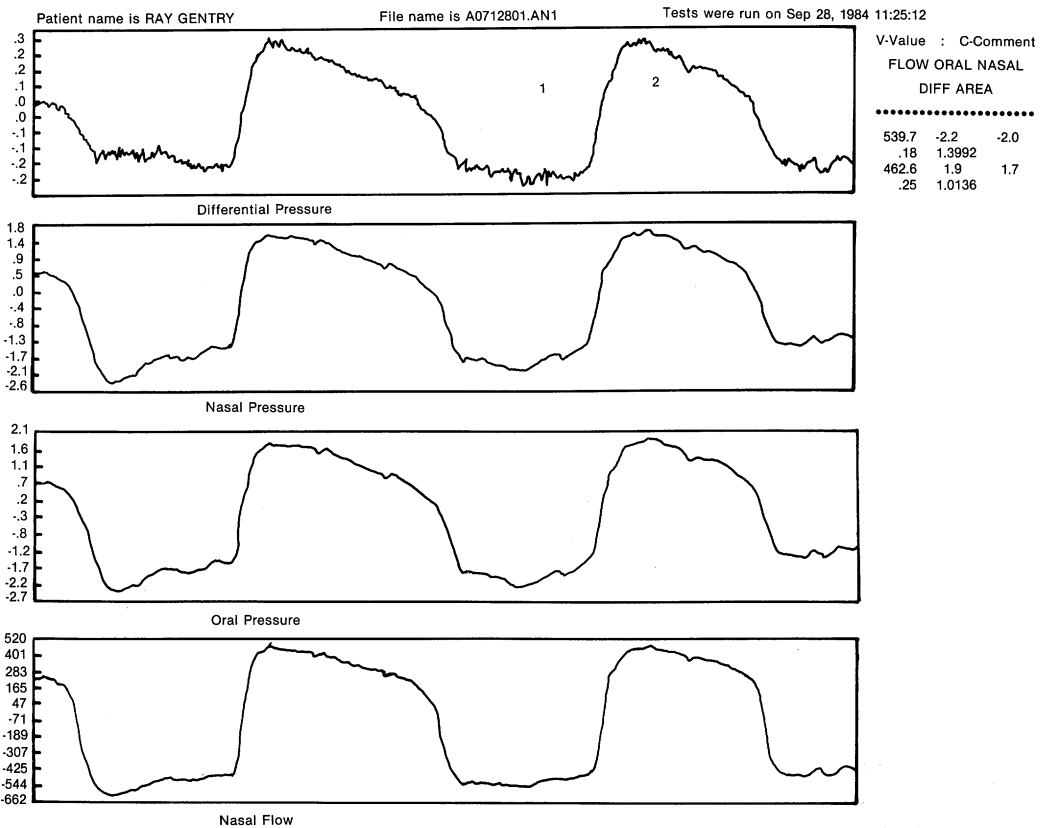
We have identified several compensatory strategies used by cleft palate speakers (Warren and Ryon, 1967; Warren and Mackler, 1968; Warren, Wood et al, 1969). Individuals with palatal incompetence use greater respiratory effort or air volumes during speech. Their volumes are approximately twice those of normal speakers. The two factors responsible, i.e., airflow rate and duration of production, are both increased in cleft palate speakers. We have also found (Warren, Duany et al, 1969) that nasal resistance is considerably higher in the cleft population. This means that cleft palate speakers can compensate for palatal inadequacy by increasing respiratory effort and the amount of increase would be determined by the degree of resistance.

Furthermore, our studies also demonstrated compensatory changes in speech timing and alterations in tongue carriage (Warren and Mackler, 1968; Claypoole et al, 1974; Warren, Dalston et al, 1985). It is apparent that the level of intelligibility attained by cleft speakers is determined to a great extent by the manner in which the various articulatory structures of the vocal tract react to the incompetence rather than by the specific degree of incompetence or error present.

These findings indicate that most cleft palate speakers attempt to regulate intraoral pressure by increasing airflow rate. However, there are limits to respiratory compensation for palatal inadequacy. Airflow rate increases only to a level of about 600 to 800 cc per second. Intraoral pressure is usually maintained above  $3.0 \text{ cm H}_2\text{O}$  in about 80 percent of the cleft population (Warren and Hinton, 1983). Many cleft speakers develop other strategies as well. The nasal grimace is another example of response to a velar deficit. Physiologically, the grimace is a manifestation of nasal valve constriction. The valve is in the region between the junction of the upper and lateral cartilages and the pyriform aperture just beyond the anterior ends of the inferior turbinates. As noted earlier, during normal breathing, the valve is closed more during expiration than it is during inspiration, and the nasal airway is approximately 10 percent smaller during the expiratory phase of breathing. In the cleft population, the nasal airway and the nasal valve assume an even more dynamic role in maintaining upper airway resistance (Warren, Hairfield et al, 1985). The nose is approximately 25 percent smaller in the cleft palate population, presumably because of maxillary growth deficits and nasal abnormalities. This smaller dimension provides greater airway resistance during speech. If one considers the relationship between cross-sectional area and airway resistance (Fig. 4), this added resistance maintains an intraoral pressure



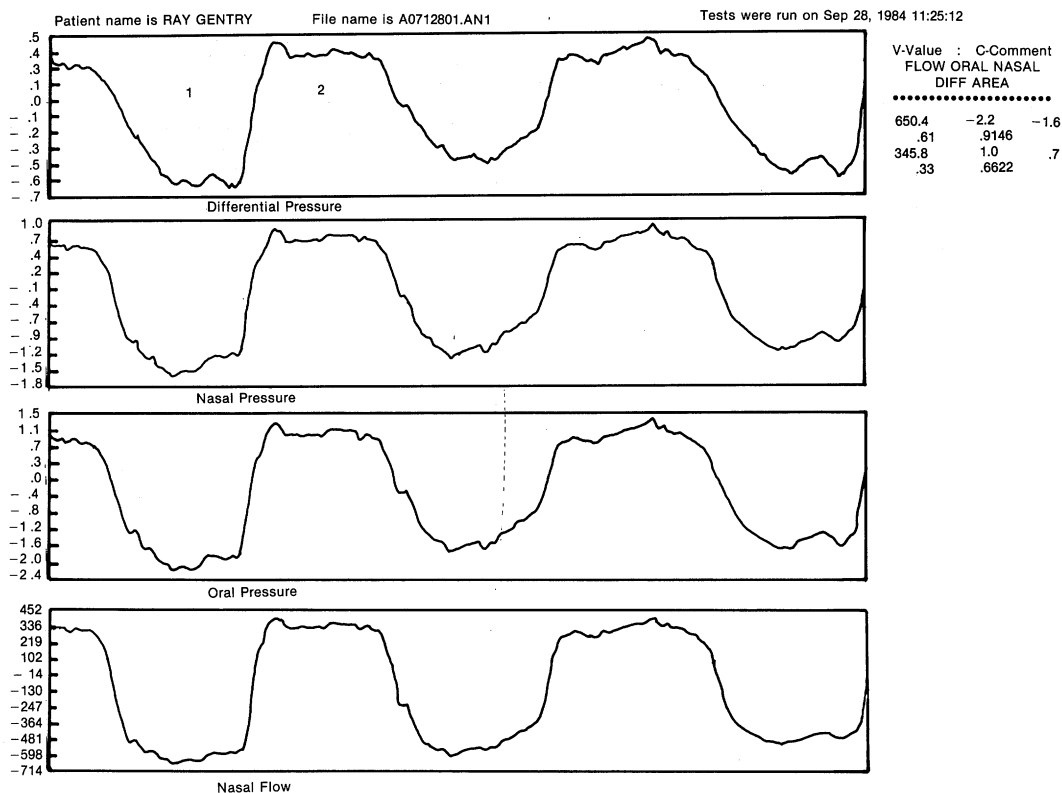
**FIGURE 4** The relationship between nasal cross-sectional area and nasal airway resistance at different rates of airflow.



**FIGURE 5** Record of an individual breathing with a speech appliance in place. Top line illustrates oro-nasal differential pressures during inspiration and expiration. Second line represents nasal pressures; third line is oral pressures and bottom line is nasal airflow. Data on right include values of nasal airflow, oral pressure, nasal pressure, differential pressure, and nasal airway size. During inspiration, nasal size is 1.4 cm<sup>2</sup> and during expiration, it is 1.0 cm<sup>2</sup>. Data points 1 and 2 illustrate where measurements are made.

of about 2 cm H<sub>2</sub>O or 3 cm H<sub>2</sub>O, depending upon the increase in airflow rate. Furthermore, there is evidence that the nasal valve actively responds to a velar deficit in some instances. Figure 5 illustrates the breathing pattern of an individual

with a speech appliance. The nasal cross-sectional area is 1.4 cm<sup>2</sup> during inspiration and 1.0 cm<sup>2</sup> during expiration. This shows the usual area differences within the inspiratory/expiratory cycle. Figure 6 illustrates that when the appliance



**FIGURE 6** The same subject as Figure 5 with speech appliance removed. Note that inspiratory size is  $0.91 \text{ cm}^2$  and expiratory size is  $0.66 \text{ cm}^2$ . The additional resistance provided by a smaller area compensates for loss of velar resistance when the appliance is removed.

ance is suddenly removed, the nasal airway becomes significantly smaller in size during expiration. That is, during inspiration the nasal cross-sectional size is  $0.92 \text{ cm}^2$ , but during expiration the nasal valve closes to  $0.66 \text{ cm}^2$ . Thus, the nasal valve responds with greater resistance when the speech appliance is removed and velar resistance decreases.

### BITE BLOCK AND BLEED STUDIES

Systemic study of a regulating system involves experiments that identify and describe the mechanisms of control. One approach is to introduce an error into the normal system and determine the mechanisms of response. We have used bite blocks that provide unnatural jaw openings as a means for assessing responses to sudden change. Insertion of a bite block forces a speaker into compensatory maneuvers that can be evaluated quantitatively (Warren et al, 1980; Warren et al, 1981; Warren et al, 1984). The pressure-flow technique developed in our laboratory was used to assess changes in anterior port size during fricative productions. Bite blocks producing 1, 3, and 6 mm of increased vertical dimension were inserted in random order without time for adaptation. Subjects were asked to

phonate a variety of test sounds, phrases, and, in one study, (Warren et al, 1984) the rainbow passage. Measurements of oral port size were made during fricative productions. Airflow rates and oral pressures were also recorded, and, in some of the studies, subjects were judged perceptually by trained and untrained listeners. The results of the studies were quite similar, and the data clearly demonstrate the remarkable adaptive behaviors of the speech structures. Although vertical dimension was increased up to 6 mm, oral port cross-sectional opening for fricatives showed little or no change. Whenever port size did increase slightly, airflow rate also increased, and intraoral pressure was maintained. These findings may be interpreted to support the regulation/control hypothesis in the following way. The sudden increase in vertical dimension that produced an anterior open bite was almost instantaneously compensated for by changes in anterior tongue tip placement. This change in tongue placement allows oral port size to remain nearly the same during fricative productions. Thus, resistance at the oral port is also maintained at an adequate level to regulate pressure. Slight adjustments by increasing airflow rate compensated for slight increases in port size. Since resistance is flow dependent, airflow changes fine-tune intraoral pressures.

Another interesting finding was the increased frequency of misarticulations with each increase in vertical dimension, in spite of the fact that port size did not change significantly (Warren et al, 1980). In contrast, when auditory masking was added, the frequency of /s/ distortions increased dramatically with 1 mm of vertical opening and remained at that high level in spite of additional increases in vertical dimension (Warren et al, 1984). Although sound distortions decreased in time in the unmasked group, they did not in the masked group. These studies suggest that structural responses to sudden change successfully maintain a normal aerodynamic environment for speech, but the speech outcome, as judged by listeners, is compromised. Impaired ability to monitor self-generated sounds under open-bite conditions further impedes the adaptive response mechanism. This seems consistent with the fact that continuous speech involves anticipation and adjustment to the articulatory position of every phoneme spoken. Without auditory feedback, /s/ sounds, where the 'margin of error' may be small even in the presence of normal hearing, would be increasingly misarticulated, especially when situated in phonetic contexts requiring extensive movement of articulators.

#### NATURALLY OCCURRING OPEN BITE STUDIES

Similar studies were performed on individuals with naturally occurring open bites, where there was no need for bite blocks (Klechak et al, 1976). Again, individuals manipulated their tongues and maintained an aerodynamic equilibrium. Whenever this was less successful, and oral port size increased for fricatives, a larger increase in airflow was observed. Pressures in almost all instances were maintained at an adequate level. The only individuals who had difficulty achieving adequate oral pressures were those with naturally occurring open bites greater than 6 mm. In these instances, it was almost physically impossible to manipulate the tongue to the extent needed to provide enough oral port resistance to prevent pressure from dropping below 3 cm H<sub>2</sub>O. In those rare occasions of gross open bites, pressures were in the 2 cm H<sub>2</sub>O range, and airflow rates increased to maximal levels of 700 to 800 cc per second (Warren et al, 1981).

#### STUDIES FROM OTHER LABORATORIES

Putnam et al (1986) recently published some data on compensatory responses to bite block and bleed valve maneuvers. Their findings viewed in terms of regulation/control theory are supportive of our hypothesis. When bite blocks were

inserted, intraoral pressures were maintained at an appropriate level, and airflow rate increased. Presumably, as in other bite block studies, the tongue was manipulated to close the induced open bite, and these maneuvers resulted in maintenance of adequate oral port resistance. The increased airflow observed in their study also would have augmented port resistance.

Another segment of this interesting study involved the use of bleed tubes of different diameters during plosive consonant productions. The bleed device prevented lingual maneuvers as a compensatory response. The investigators found that airflow rate increased in a linear fashion as bleed tube size increased. Although pressure fell with increased opening, it was always maintained above 4 cm H<sub>2</sub>O.

Putnam et al (1986) also observed another characteristic of aeromechanical integrity. Airflow on the postconsonantal vowels exhibited remarkably stable patterns across bleed and block conditions. The investigators reported that "this implies some accommodating adjustments in laryngeal airway resistance to normalize vowel flow in spite of the aeromechanical perturbations introduced during the preceeding /p/ and /s/ segments." "Such data lend credence to the attractive but inherently elusive hypothesis that vocal tract pressures and flows are somehow monitored and airway resistances regulated." Similarly, Shelton and Blank (1981) measured oral air pressure and nasal airflow during stop /p/ and fricative /f/ consonant productions in subjects with oronasal fistulas. Their data support the hypothesis that adequate intraoral pressure can be maintained in the presence of some oral air leaks.

Further support of pressure regulation, even in the absence of a respiratory source, is found in a study by Brown and McGlone (1979). They observed that during a Valsalva maneuver oral pressures were maintained at normal speech levels.

#### IMPLICATIONS OF A REGULATING SYSTEM

If speech aerodynamics do conform to the principles of a regulating system, new explanations for the maladaptive behaviors observed in cleft palate are possible. Aerodynamic performance rather than acoustic accuracy would receive priority in the speech-motor control program. In fact, in many of the studies cited earlier, compensatory responses to errors in the system usually met the criteria for aerodynamic stability at the expense of speech performance.

Figure 7 suggests possible control responses to velopharyngeal incompetence. Greater respiratory muscle activity results in an increased airflow rate. Since resistance is flow dependent, velar resistance would increase and, as a conse-

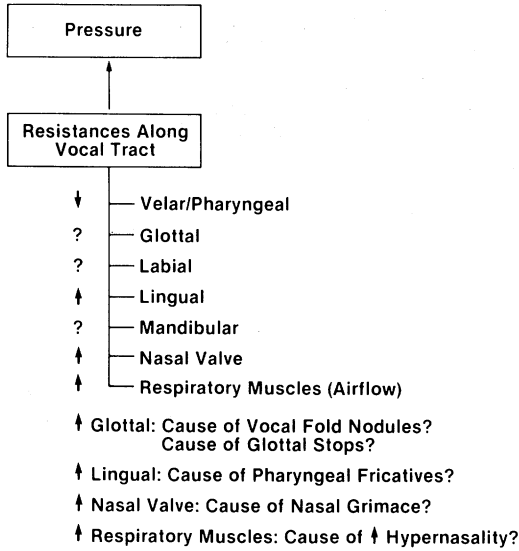


FIGURE 7 Proposed possible effects of velar resistance deficit resulting from velopharyngeal incompetence.

quence, greater intraoral pressure would be achieved. Individuals with high nasal resistance would require less airflow to maintain intraoral pressure. Additionally, nasal resistance can be increased by constricting the nasal valve. This is the physiologic manifestation of the nasal grimace and could increase nasal resistance by 10 to 30 percent. As discussed earlier, the nasal valve normally constricts more during expiration than during inspiration, and this reflex associated with breathing is also available for speech function.

High tongue carriage and the pharyngeal fricatives would also increase vocal tract resistance in the presence of a velar deficit. Similarly, the glottis normally serves as an expiratory brake during breathing and adducts more during expiration than during inspiration. The glottal stop could be a more forceful manifestation of this reflex. The well known Passavant's pad activity of the posterior pharyngeal wall may also be an airway response to a loss of resistance. The anterior movement of the muscle decreases cross-sectional size of the airway and, therefore, would increase airway resistance. The point to be emphasized is that a speech regulating system would be dedicated to maintaining speech pressures, and many of the compensatory behaviors in cleft palate appear to fit the description of control responses.

Although the hypothesis that speech aerodynamics follows regulation/control principles may be novel, it is very much in line with modern theories of speech-motor control as described by Kelso et al, 1983, and Kelso and Tuller, 1984. Speech-motor control theory is framed in such terms as "motor equivalence"

and "coordinative structures." In terms of segmental speech activities, the postulate is that functionally and anatomically distinct parts of the speech system are constrained to act together toward a common goal. Within this framework, the respiratory and articulatory systems during obstruent productions are hypothesized to form a coordinative structure whose goal is to regulate speech pressures.

Figure 8 illustrates a possible feedback system for regulating speech pressures. Although pressure would vary somewhat according to consonant type, the system would be driven to maintain an adequate level of pressure for consonant productions. The system proposed is very similar to pressure regulation in cardiovascular dynamics. It is purposefully enigmatic to convey the uncertainty of many of the components of this "black box" arrangement.

FUTURE RESEARCH

Systemic study of a regulating system involves experiments that identify and describe the

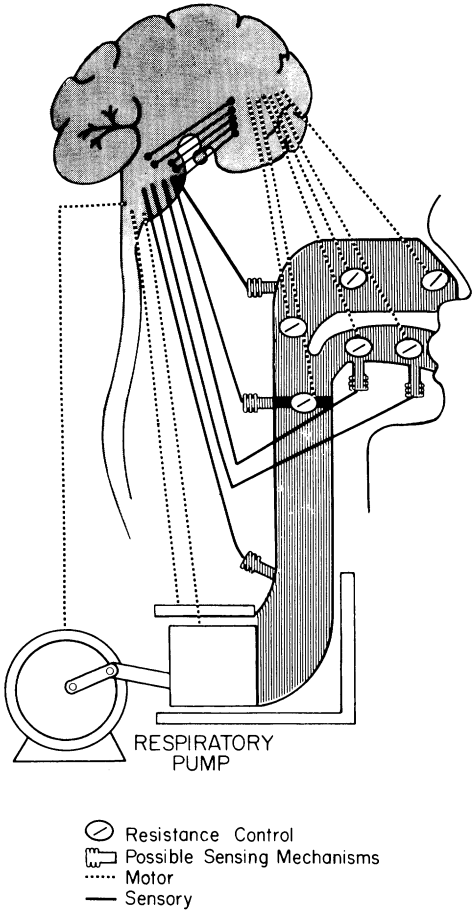


FIGURE 8 Theoretical representation of a possible feedback system to regulate speech pressures. Possible sensors send error messages to resistance controllers via the brain.



mechanisms of control. Subjects with velopharyngeal incompetency provide an opportunity to assess how the system is maintained when an error is introduced. Similarly, the use of bite blocks and bleed valves provides the means to assess responses in normal individuals when an intact system is suddenly perturbed.

The stigmata of cleft palate speech would be explained if we find that compensatory attempts to maintain a normal aerodynamic environment in the presence of velopharyngeal incompetence actually undermine acoustic performance. If data from our studies demonstrate that the speech structures are programmed for regulation/control purposes, then another method for "resetting" the brain's "speech computer" may be possible. That is, the data obtained in these studies may provide the information necessary to eventually develop an entirely new approach for modifying the abnormal speech patterns associated with cleft palate.

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