

Controlling changes in vocal tract resistance

By: Donald W. Warren, Anne Putnam Rochet, Rodger M. Dalston, and Robert Mayo

Warren, D.W., Rochet, A.P., Dalston, R.M., and [Mayo, R.](#) (1992). Controlling changes in vocal tract resistance. *Journal of the Acoustical Society of America*, 91(5), 2947-2953. DOI:10.1121/1.402930

Made available courtesy of the Acoustical Society of America: <http://dx.doi.org/10.1121/1.402930>

*****Note: Figures may be missing from this format of the document**

Abstract:

There is some evidence that speech aerodynamics follows the rules of a regulating system. The purpose of the present study was to assess how the speech system manages perturbations that produce "errors" within the system. Three experimental approaches were used to evaluate the physiological responses to an imposed change in airway resistance. The first involved subjects with varying degrees of velopharyngeal inadequacy. The second and third approaches involved noncleft subjects whose airway was perturbed by bleed valves and bite blocks during consonant productions. The pressure-flow technique was used to measure aerodynamic variables associated with the production of test consonants. The results of this study provide additional evidence that the speech system actively responds to perturbations in ways that tend to minimize a change in consonant speech pressures. The degree of success in stabilizing pressures appears to reflect the capability of the system to use whatever articulatory and respiratory responses are available.

Article:

INTRODUCTION

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 that operate 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 (Brobek, 1965). A system is said to be regulated if structures respond to change and by their activity preserve or attempt to preserve some level of constancy. That is, the purpose of a regulating system is to maintain a certain parameter at an acceptable 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. It should also be noted that whenever the term control is used, it implies that the brain receives information, processes it, and then directs the control activity (Warren, 1986).

The concept of a speech regulating system implies that mechanisms exist to detect "errors" such as a loss of resistance. A detection system has not been identified in speech, although 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). Similarly, laryngeal receptors sensing pressure, airflow, and muscle contractions have also been described (Sant'Ambrogio *et al.*, 1983). Recent studies by England and Bartlett (1982) demonstrate that the larynx controls respiratory flow in man by varying the degree of glottal adduction. 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 stretch receptors in which the respiratory muscles during expiration compensated for changes in upper airway resistance. In a recent study, Warren *et al.* (1991) demonstrated that airway resistance is precisely controlled during breathing. Postural changes apparently moderate any imposed increase in breathing load. Elice and Warren (1991) reported that individuals can detect a change in resistance of about 0.7-cm H₂O 1/s. Thus there appears to be a detection system for breathing that may, in some fashion, operate in

speech as well. Malecot (1966, 1970) and Williams *et al.* (1987) provided some evidence that such a system exists. Whether the detection system actually senses pressure or some correlate of pressure remains to be seen. Malecot (1966, 1970), Muller and Brown (1980), Wyke (1981), and Williams *et al.* (1987) have indicated that aerodynamic monitoring may be used to direct the activity of speech structures.

Individuals with velopharyngeal inadequacy present a unique opportunity to study the dynamics of a pressure regulating system. Velopharyngeal inadequacy represents a decrease in vocal tract resistance and introduces an error that requires compensatory responses to moderate a potential drop in intraoral pressure. We have reported that, even at inadequacies greater than 0.20 cm², many cleft palate individuals are able to maintain speech pressures above 3-cm H₂O during non-nasal consonant productions (Dalston *et al.* 1988; Warren *et al.*, 1989). Several possible compensatory strategies may be involved. Some cleft palate speakers may attempt to maintain intraoral pressure by increasing respiratory effort. Others may use articulatory compensations to maintain intraoral pressure. Some may not have to employ compensatory strategies because their nasal airway resistance is high.

The drive to maintain aerodynamic integrity has also been observed in normal speakers under experimental conditions. Putnam *et al.* (1986) introduced an air leak into the oral cavity using a bleed valve. They found that respiratory airflow was increased and, as a consequence, intraoral pressures were maintained above 4-cm H₂O. In addition, bite-block studies indicate that the drive to maintain speech pressures in the presence of an air leak may be even stronger than the need to preserve acoustic accuracy (Warren *et al.*, 1984). These findings led us to postulate that the respiratory and articulatory structures are constrained to act together toward the common goal of maintaining an adequate level of pressure for consonants.

The purpose of the present study was to assess the control responses to changes in vocal tract resistance. Specifically, we wanted to perturb the system in ways that invoked different management or control responses. This approach would allow us to evaluate the system's capability to utilize available articulatory and respiratory adjustments and to compare the success of various outcomes.

I. METHOD

Three experimental approaches were used in this study. The first involved 141 subjects with varying degrees of velopharyngeal closure. The differences in closure represented changes in velopharyngeal resistance during consonant productions. The pressure-flow technique (Warren and Dubois, 1964; Warren, 1982; Warren *et al.*, 1985) was used to measure pressures and airflow associated with velopharyngeal closure. The age range of the subjects was 5 years, 8 months to 58.0 years with a mean age of 18.0. The only criterion employed during subject selection was that the patients had to have the intellectual capacity needed to perform the tasks required of them.

Briefly, the pressure drop across the velopharyngeal orifice (oral pressure minus nasal pressure) was measured by placing one catheter within the mouth and another in one nostril. The nasal catheter was secured by a cork that blocked the nostril, creating a stagnant column of air. Both catheters measured static air pressures and transmitted these pressures to pressure transducers. Nasal airflow was measured by a heated pneumotachograph connected by plastic tubing to the subject's other nostril. The area of the constriction was then calculated from the equation $A = \dot{V} / k(2\Delta P / d)^{1/2}$, where A is the area of the orifice, \dot{V} is the nasal airflow, $k = 0.65$, ΔP is the oral-nasal pressure, and d is the density of air. A commercially available software program (P-Scope, Microtronics, Inc., Carrboro NC 27510) was used to obtain average pulse pressure, peak pressure, average airflow, peak airflow, velopharyngeal orifice area, and velopharyngeal resistance values.

The subjects were asked to produce a series of the bilabial plosive consonant /p/ within the carrier word "hamper." The nasal-plosive blend /mp/ was used to stress the palatal mechanism. This phonetic combination also more nearly approximates the degree of closure that occurs during continuous speech (Warren, 1979). Aerodynamic variables measured during the production of /mp/ were calculated from a series of five

utterances for each subject. The subjects were then divided into four groups according to velopharyngeal orifice size. Fifty subjects had adequate closure ($< 0.05 \text{ cm}^2$); 38 had adequate-borderline closure ($0.05\text{-}0.09 \text{ cm}^2$); 22 had borderline-inadequate closure ($0.10\text{-}0.19 \text{ cm}^2$); and 31 had inadequate closure ($> 0.20 \text{ cm}^2$). Categorization according to those area criteria is, in part, perceptually based and, in part, aerodynamically based (Warren, 1964, 1979, 1982). That is, velopharyngeal areas less than 0.05 cm^2 were considered adequate because normal, noncleft speakers do not manifest areas greater than this value (Warren, 1964). Conversely, the definition of inadequate closure was based on aerodynamic data demonstrating the oral-nasal differential pressure during speech is very low at areas equal to or greater than 0.20 cm^2 (Warren and Dubois, 1964). In addition, unpublished clinical observations have led us to conclude that speakers with velopharyngeal areas in the inadequate range invariably manifest hypernasality and/or nasal emission. Finally, the intermediate categories of adequate/ borderline closure ($0.05\text{-}0.09 \text{ cm}^2$) and borderline/inadequate closure ($0.10\text{-}0.19 \text{ cm}^2$) are somewhat more arbitrary. However, there is both aerodynamic and perceptual evidence to support such groupings (Warren, 1979).

Audiometric testing was conducted on 110 of the 141 subjects investigated. The 31 patients on whom testing was not performed were individuals who had a negative otologic history and normal audiograms at each of their last two team evaluations. Of the 110 patients on whom testing was performed, all but two had no more than a mild conductive hearing loss in the better ear. For this reason, audiologic status was not considered an important determinant of subject performance.

The second approach involved ten normal adult male subjects who produced /pa/ under conditions in which a translabial device released intraoral pressure during /P/. Openings ranged from 0.0 to 0.28 cm^2 in eight steps. Eight interchangeable plastic cylinders served as bleed plugs: one was solid (i.e., "no bleed" condition), and seven were invariably patent with orifice areas from 0.045 to 0.283 cm^2 .

The protocol for this condition required subjects to produce the syllable /p/ seven times at a rate of 1.5/s on one expiratory breath. This was done twice with each translabial bleed plug in place, and the entire protocol was performed a second time: For both performances of the entire protocol, subjects always began with the pressure release device in the unperturbed (i.e., "no bleed") condition. Presentation of the various patent bleed plugs was randomized thereafter for every performance by each subject. Oral pressures and oral airflow through the bleed valve were recorded using the pressure-flow instrumentation.

In the third condition, the same ten subjects produced /si/ under several bite-block openings that increased vertical dimensions during /s/ from 0, 2, 4, and 6 mm. The protocol for the bite blocks required subjects to produce the syllable /si/ seven times at a rate of 1.5/s on the expiratory breath. This was done twice with each bite block, and the entire protocol was performed a second time. For both performances of the entire protocol, subjects always began with the unperturbed (i.e., "no block") condition. Presentation of the various bite blocks was randomized thereafter for every performance by each subject. The pressure-flow technique was used to record oral pressures and oral airflow variables as described earlier.

Descriptive statistics were initially run in order to check for homogeneity of variance of the data from subjects with varying degrees of velopharyngeal adequacy. There was a discrepancy in homogeneity in three of the variables namely, airflow rate, average airflow, and average resistance. A log transformation was performed on average flow and average resistance to correct for this. The airflow data were ranked to fulfill homogeneity. A one-way ANOVA was then used to analyze the data that were grouped by categories of velopharyngeal orifice size. In the bleed-valve and bite-block studies, a two-way ANOVA was used to analyze the data.

II. RESULTS

A. Average pressure pulse

Figure 1 illustrates the average pulse pressure across the three experimental conditions. The average pulse pressure (pressure over pulse duration) for the subjects with velopharyngeal inadequacy fell from 3.0-cm

H₂O for the adequate group to between 2.6- and 2.4-cm H₂O for the other groups. The drop in average pulse pressure was only 0.6-cm H₂O as mean orifice size increased from 0.02 to 0.55 cm². A one-way ANOVA revealed that the overall change in pressure across groups was not significant [$F(3, 137) = 2.65; p = 0.0513$]. Thus despite the large change in velopharyngeal orifice size across groups, the average pressure change was very small (Table I). The drop in average pressure was somewhat larger in the bleed-valve experiment with pressure falling from 4.5- to 3.1-cm H₂O, a drop of 1.4-cm H₂O which was statistically significant [$F(7, 63) = 13.48; P 0.0001$]. On the other hand, the bite-block study resulted in virtually no change in the average pressure pulse across bite openings [$F(3, 27) = 0.11; p 0.95$].

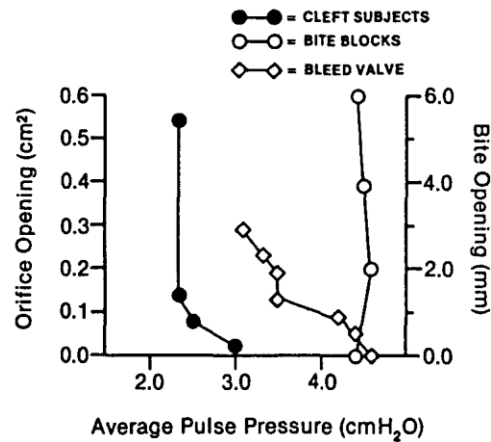


FIG. 1. Average pulse pressures for degrees of velopharyngeal closure, bite blocks, and bleed valves. The average pulse pressure represents pressure over the duration of the pulse.

Table II lists the average airflow rates across openings for each condition. Average airflow rate increased significantly as degree of opening increased [$F(3, 137) = 229.7; p = 0.0001$]. The bleed-valve findings were similar [$F(7, 63) = 220; p = 0.0001$]. On the other hand, average airflow rate did not change across the various bite-block openings.

TABLE I. Average pressures (cm H₂O) and standard errors (s.e.) across conditions.

Condition	Amount of opening	Pressure (cm H ₂ O) ± s.e.
Cleft (cm ²)	0.017 ± 0.002	3.0 ± 0.18
	0.08 ± 0.007	2.6 ± 0.16
	0.14 ± 0.007	2.4 ± 0.17
	0.55 ± 0.038	2.4 ± 0.21
Bleed valve (cm ²)	0.0	4.5 ± 0.40
	0.045	4.2 ± 0.51
	0.910	4.0 ± 0.48
	0.132	3.4 ± 0.40
	0.159	3.7 ± 0.38
	0.196	3.5 ± 0.36
Bite block (cm)	0.238	3.3 ± 0.45
	0.283	3.1 ± 0.39
	0.0	4.6 ± 0.49
	0.2	4.5 ± 0.59
Bite block (cm)	0.4	4.5 ± 0.42
	0.6	4.4 ± 0.51

TABLE II. Average airflow (cc/s) and standard errors (s.e.) across conditions.

Condition	Amount of opening	Airflow (cc/s) \pm s.e.
Cleft (cm ²)	0.017 \pm 0.002	58 \pm 3.9
	0.08 \pm 0.007	108 \pm 7.1
	0.14 \pm 0.007	162 \pm 13.5
	0.55 \pm 0.038	233 \pm 20.5
Bleed valve (cm ²)	0.0	233 \pm 24.2
	0.045	344 \pm 30.4
	0.091	371 \pm 36.6
	0.132	390 \pm 34.8
	0.159	448 \pm 39.4
	0.196	446 \pm 29.8
	0.238	447 \pm 34.2
Bite block (cm)	0.0	504 \pm 86.6
	0.2	524 \pm 111.8
	0.4	596 \pm 152
	0.6	609 \pm 141.6

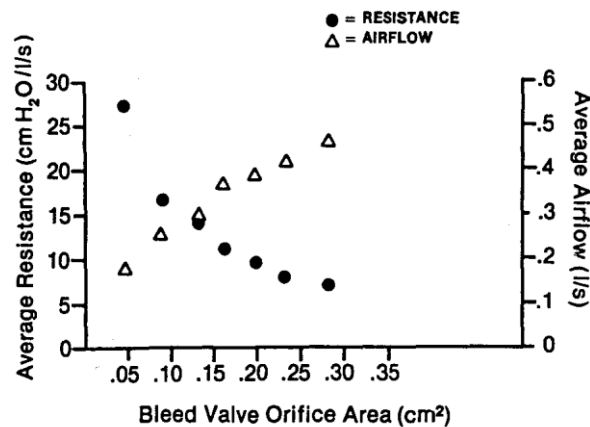


FIG. 3. Average resistance and average airflow across conditions for the bleed-valve experiment.

B. Resistance

Figure 2 presents data on average velopharyngeal resistance and airflow rates for subjects by degrees of adequacy. As expected, resistance dropped sharply from adequate closure to borderline-inadequate closure and then stabilized somewhat. At about 0.2 cm² of opening the increase in airflow rate moderated the fall in resistance. For example, as velopharyngeal orifice area increased from 0.017 to 0.14 cm², a difference of 0.12 cm², resistance fell about 60-cm H₂O 1/s. As orifice area increased from 0.14 to 0.55 cm², resistance fell only about 3-cm H₂O 1/s. A one-way ANOVA was run, and the drop in resistance was statistically significant [$F(3, 137) = 38.7; p = 0.0001$].

Results from the perturbation studies involving the normal adult subjects were notably different from those reported above. Figure 3 illustrates the relationship among average resistance, average oral airflow rate and bleed-valve orifice area. In this experimental condition, resistance fell more rapidly than in the condition involving subjects with velopharyngeal inadequacy. For example, resistance at 0.28 cm² is about 8-cm H₂O 1/s compared to about 16-cm H₂O 1/s in the human subjects. On the other hand, average airflow rate was about twice as high. A two-way ANOVA was run on the resistance values, and the overall decrease was statistically significant [$F(7, 63) = 21.3; p = 0.0001$]. Average airflow rate was also significantly increased [$F(7, 63) = 21.99; p = 0.0001$] with the values for the bleed-valve condition being about twice as great as the cleft condition.

The data from the bite-block experiment are shown in Fig. 4. In marked contrast to the preceding experiments, the increase in vertical opening imposed by the various bite blocks was associated with minimal change in both oral port resistance and oral airflow rate. It should be noted that the magnitude of resistance in the unperturbed system is much higher for /p/ because /s/ is normally produced with an oral port opening of 0.05 cm². Nevertheless, resistance dropped only about 2-cm H₂O 1/s when bite-block openings increased from 0 to 6 mm. No statistically significant differences were noted across bite-block conditions.

III. DISCUSSION

This study provides additional evidence that the human system actively responds to perturbations of vocal tract resistance in ways that tend to minimize the potential drop in pressures associated with consonant productions. As hypothesized previously (Warren, 1986), the articulatory structures represent a set of variable resistors that maintain vocal tract resistance during phonation. Thus structural movements create the resistances that are required to maintain speech pressures. The regulatory system also includes a variable energy source in the form of controlled respiratory airflow that provides further adjustments of vocal tract resistance.

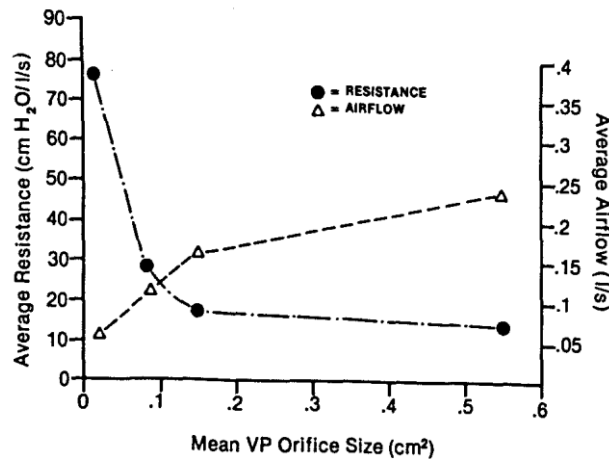


FIG. 2. Average resistance and airflow for the subjects with velopharyngeal inadequacy. Average resistance is the resistance of the velopharyngeal sphincter over the duration of the pressure pulse.

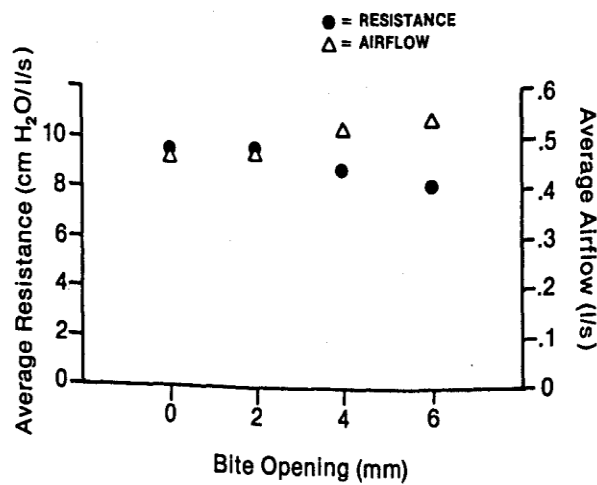


FIG. 4. Average resistance and average airflow across conditions of bite opening for the bite-block experiment.

The differences noted among the three experimental conditions apparently reflect the capability of the speech system to successfully utilize available articulatory and respiratory adjustments to stabilize the aerodynamic environment during consonant productions. The bleed-valve experiment represented a condition under which subjects could compensate only by adjusting respiratory drive. Although pressure did fall across conditions, the magnitude of change was considerably less than what has been reported in an equivalent but passive model system (Warren *et al.*, 1989). The difference reflects the success of respiratory compensation. As Putnam *et al.* (1986) demonstrated earlier, the human system is not a constant flow system, and increased respiratory effort moderates the fall in pressure. This was confirmed in the current study.

Subjects with velopharyngeal inadequacy compensated to a greater degree than the bleed-valve subjects. The average drop in pressure was only 0.4 cm-H₂O compared to 1.6-cm H₂O for the bleed-valve subjects. The reason for the difference may be attributed to the additional resistance provided by the nose in subjects with velopharyngeal inadequacy. In a previous study Warren *et al.* (1990) demonstrated that the ability to achieve adequate consonantal pressures depends upon total upper airway resistance, and the nose usually provides about two-thirds of that resistance when the velopharyngeal mechanism is inadequate. Thus the level of respiratory effort required to minimize pressure loss was less in the cleft group because of the upstream nasal resistance—a circumstance not available to the subjects in the bleed-valve experiment. That is, the bleed valve opened to the atmosphere rather than the nose. Using respiratory compensation alone, the latter subjects were not as successful in regulating speech pressures.

The bite-block experiment serves as the best example of a successful outcome in terms of compensation. Pressure did not change, and resistance was maintained across conditions of bite opening. In this experiment, subjects were free to use respiratory and lingual articulatory compensations in a coordinated fashion to minimize the impact of imposed perturbations. The results reported here and elsewhere (Warren *et al.*, 1984; Putnam *et al.*, 1986) suggest that subjects use lingual compensations to prevent a fall in pressure under such circumstances, even though such maneuvers may sometimes occur at the expense of speech accuracy (Warren *et al.*, 1984).

One might argue that pressure maintenance is not under strict regulation/control since pressure did fall somewhat in each of the human studies reported above. However, human regulation systems are not always successful in accomplishing their tasks. The essence of a regulating system is that attempts are made to maintain constancy even though constancy may not always be achieved. In two previous studies (Dalston *et al.*, 1988; Warren *et al.*, 1989), we compared the active human regulating system to a simple model and found that intraoral pressures dropped about seven to tenfold in the passive model under simulated experimental conditions.

Although the term constancy is usually used to denote the goal of a regulatory system, it should be noted that regulatory systems have flexibility that allows them to meet changing demands. The cardiovascular system serves as an appropriate example. Blood pressure is usually regulated at a mean pressure of 100 mm Hg. However, during moderate exercise, the demands of the system elevate the mean pressure to 120-130 mm Hg. This is somewhat comparable to increasing intensity level in speech—a higher pressure is required under more demanding circumstances. A change in the barostat raises the level at which blood pressure is regulated, but this should not be construed as meaning the system is not regulated.

It might also be argued that the maintenance of pressure is merely required so that speakers can generate the acoustic power necessary to generate perceptually acceptable pressure consonants. Netsell (1990), for example, suggests an acoustic regulator hypothesis as an alternative to the pressure regulating hypothesis. He proposes that a speaker's attempt to maintain an appropriate level of pressure is for acoustic purposes rather than pressure per se. Thus pressure regulation would reflect the fact that pressure is a necessary requisite to achieve the primary goal of impounding air for pressure-consonant productions. This possibility has yet to be tested, but there is some information that suggests that this may not be the case.

Warren *et al.* (1984) reported studying the speech of normal individuals under auditory masking and varying conditions of imposed anterior open bite. They found that these subjects tended to maintain oral port sizes appropriate for the production of fricative consonants even though the perceptual quality of these phonemes deteriorated noticeably. However, the fact that listeners perceived a deterioration in speech during the experimentally induced anterior open-bite conditions suggests one or more of the following: (1) pressure maintenance took precedence over perceptual accuracy; (2) pressure maintenance does not have a one-to-one relationship with perceived speech normality; (3) pressure maintenance was necessary but not sufficient for production of accurate speech, and speakers were able to maintain pressure but not able to produce all the requisites for acceptable speech; (4) speakers may adopt a minimum competency strategy leading to adjustments that do not fully compensate for experimentally imposed perturbations; or (5) the speakers perceived that their own speech remained acceptable under the various experimental conditions.

A recent study by Moon and Folkins (1989) provides further insight into the question of acoustic regulation versus pressure regulation. They assessed the relative importance of auditory feedback in the regulation of intraoral pressure and reported some preliminary findings. Normal speaking subjects were forced into a choice between aerodynamic versus auditory feedback. Manipulation of the acoustic signal heard by the subjects resulted in adjustments of peak pressure and/or duration. However, the changes in pressure were not of the magnitude expected for the large adjustment in the acoustic signal. They found that the system is somewhat resistant to changes in speech pressures.

Interaction between the acoustic and aerodynamic systems is highly probable. Most regulating systems are multi-factorial, and outcomes often reflect the need to maintain more than one parameter at an optimal level. For example, in respiration, the system is managed so that CO₂ concentrations, pH level, and peak upper airway breathing pressures are kept within a certain limited range despite changes in the environment or in level of activity. Information concerning the chemical environment of the blood, the status of the airway, and other factors is integrated by the brain and appropriate control responses made. Sometimes, these responses represent compromises for the system.

It should also be noted that a subject who may monitor or attempt to regulate pressure during speech production may be doing so because of associations established much earlier between pressure and other, perhaps more fundamental, variables such as perceived speech quality or acoustics. It is quite possible that such associations may be formed and strengthened during the very early stages of speech development. Therefore, the fact that a given variable (e.g., pressure) may be regulated after speech development is completed does not rule out the original and primary importance of some other associated variable. Congenital deaf speech clearly exemplifies the consequences of inadequate acoustic feedback upon speech development and speech production. An assessment of the aerodynamic patterns of deaf speakers is a direction that should yield important new information on aerodynamic and acoustic interaction. Cain *et al.* (1989) found that congenitally deaf but intelligible speakers have basically normal pressure patterns despite articulatory errors related to their auditory deficits. Brown and Goldberg (1990) reported similar findings, but only when speech was intelligible.

There are still many gaps in our understanding of a regulating system as it pertains to speech. The detection system, an essential component to identify "errors," is only now beginning to receive some attention (Miller and Brown, 1980; Wyke, 1981; Williams *et al.*, 1987) although Malécot presented some data on sensitivity of receptors in 1966. The specific variable being detected is not entirely clear. The regulated parameter is not necessarily the same variable that is detected (Brobeck, 1965). The system could respond to some correlate or function related to that variable. The minimal level of pressure required for adequate consonant production also remains unknown. For example, a pressure value of 3-cm H₂O has been routinely adopted as an operational definition of adequate consonant pressure (Dalston *et al.*, 1988; Warren *et al.*, 1989). The validity of this definition has not been tested rigorously, even though it is based upon longterm observations of data generated from aerodynamic studies. The relationship between consonant pressure and the perceptual acceptability of its acoustic analog also must be investigated. The linguistic influence on the control of

speech respiration also has received little attention. Conrad *et al.* (1983) reported that linguistic factors such as the structural organization of textual material exert a strong influence on the timing of inspiration and the size of inspiratory amplitude when speaking under normal conditions. The potential influences of linguistic factors on pressure, flow, and vocal tract resistance remain to be determined.

Finally, as Netsell (1990) correctly indicated, intraoral pressure is a reflection of subglottal pressure as well as articulatory movements of the larynx and upper airway. Studies of the speech regulation system have purposely limited the speech sample to pressure consonants in which intraoral pressure would be expected to reflect subglottal pressure. Vowels, for example, have not been used. Thus a major test of the regulating system hypothesis would be to determine whether subglottal pressures rather than intraoral pressures remain fairly constant under experimental conditions that perturb the system during continuous speech.

ACKNOWLEDGMENT

- Brobeck, J. R. (1965). "Exchange, Control, and Regulation," in *Physiological Controls and Regulation*, edited by J. R. Brobeck (W. B. Saunders, Philadelphia).
- Brouillette, R. T., and Thach, B. T. (1980). "Control of Genioglossus Muscle Inspiratory Activity," *J. Appl. Physiol.* 49,801-808.
- Brown, W. S., and Goldberg, D. M. (1990). "An Acoustic Study of the Intelligible Utterances of Hearing-Impaired Speakers," *Folia Phoniatr.* 42,230-283.
- Cain, M., Seaver, E., Jackson, P., and Sandridge, S. (1989). "Aerodynamic and Nasometric Assessment of Velopharyngeal Functioning in Hearing-Impaired Speakers," *ASHA* 31,158.
- Cohen, M. I. (1975). "Phrenic and Recurrent Laryngeal Discharge Patterns and the Hering-Breuer Reflex," *Am. J. Physiol.* 228,1489-1496.
- Conrad, B., Thalacker, S., and Schönle, P. (1983). "Speech Respiration as an Indicator of Integrative Contextual Processing," *Folia Phoniatr.* 35, 220-225.
- Dalston, R. M., Warren, D. W., Morr, K. E., and Smith, L. R. (1988). "Intraoral Pressure and its Relationship to Velopharyngeal Inadequacy," *Cleft Palate J.* 25,210-217.
- Elice, C. E., and Warren, D. W. (1991). "Perception of Nasal Airway Resistance," *J. Dent. Res.* 70, 341.
- England, S. J., and Bartlett, D. (1982). "Changes in Respiratory Movements of Human Vocal Cords During Hyperpnea," *J. Appl. Physiol.* 52, 780-785.
- Malecot, A. (1966). "The Effectiveness of Intraoral Air Pressure Pulse Parameters in Distinguishing between Stop Cognates," *Phonetica* 14, 6581.
- Malecot, A. (1970). "The Lenis-fortis Opposition: Its Physiological Parameters," *J. Acoust. Soc. Am.* 47, 1588-1592.
- McBride, B., and Whitelaw, W. A. (1981). "A Physiological Stimulus to Upper Airway Receptors in Humans," *J. Appl. Physiol.* 51, 1189-1197.
- Moon, J. B., and Folkins, J. W. (1989). "Regulation of Intra-oral Air Pressure: Role of Auditory Feedback," *ASHA* 31,114.
- Muller, E. M., and Brown, W. S. (1980). "Variations in the Supraglottal Air Pressure Waveform and Their Articulatory Interpretation," in *Speech and Language: Advances in Basic Research and Practice*, edited by N. J. Lass (Academic, New York).
- Netsell, R. (1990). "Commentary," *Cleft Palate J.* 27,59-60.
- Putnam, A. H. B., Shelton, R. L., and Kastner, C. V. (1986). "Intraoral Air Pressure and Oral Airflow under Different Bleed and Bite-Block Conditions," *J. Speech Hear. Res.* 29,37-49.
- Remmers, J. E., and Bartlett, D. (1977). "Reflex Control of Expiratory Airflow and Duration," *J. Appl. Physiol.* 42,80-87.
- Sant'Ambrogio, G. (1982). "Information Arising from the Tracheobronchial Tree in Mammals," *Physiol. Rev.* 62,531-569.
- Sant'Ambrogio, G., Matthew, O. P., Fisher, J. T., and Sant'Ambrogio, F. B. (1983). "Laryngeal Receptors Responding to Transmural Pressure, Airflow and Local Muscle Activity," *Resp. Physiol.* 54, 317-330.
- Warren, D. W. (1964). "Velopharyngeal Orifice Size and Upper Pharyngeal Pressure-Flow Patterns in Cleft Palate Speech: A Preliminary Study," *J. Plast. Reconstr. Surg.* 34, 15-26.

- Warren, D. W. (1979). "Perci: A Method for Rating Palatal Efficiency, *Cleft Palate J.* 16, 279-285.
- Warren, D. W. (1982). "Aerodynamics of Speech," in *Speech, Language and Hearing*, edited by N. J. Lass (W. B. Saunders, Philadelphia).
- Warren, D. W. (1986). "Compensatory Speech Behaviors in Cleft Palate: A Regulation/Control Phenomenon?," *Cleft Palate J.* 23, 251-260.
- Warren, D. W., Allen, G., and King, H. A. (1984). "Physiologic and Perceptual Effects of Induced Anterior Open Bite," *Folia Phoniatr.* 36, 164-173.
- Warren, D. W., Dalston, R. M., and Dalston, E. T. (1990). "Maintaining Speech Pressures in the Presence of Velopharyngeal Impairment," *Cleft Palate J.* 27, 53-58.
- Warren, D. W., Dalston, R. M., Morr, K. E., Hairfield, W. M., and Smith, L. R. (1989). "The Speech Regulating System: Temporal and Aerodynamic Responses to Velopharyngeal Inadequacy," *J. Speech Hear. Res.* 32, 566-575.
- Warren, D. W., Dalston, R. M., Trier, W. C., and Holder, M. B. (1985). "A Pressure-Flow Technique for Quantifying Temporal Patterns of Palatopharyngeal Closure," *Cleft Palate J.* 22, 11-19.
- Warren, D. W., and Dubois, A. B. (1964). "A Pressure-Flow Technique for Measuring Velopharyngeal Orifice Area during Continuous Speech," *Cleft Palate J.* 1, 52-71.
- Warren, D. W., Hairfield, W. M., and Dalston, E. T. (1991). "Nasal Airway Impairment: The Oral Response in Cleft Palate Patients," *Am. J. Orthod.* 99, 346-353.
- Williams, W. N., Brown, W. S., and Turner, G. E. (1987). "Intraoral Air Pressure Discrimination by Normal-speaking Subjects," *Folia Phoniatr.* 39, 196-203.
- Wyke, B. (1981). "Neuromuscular Control Systems in Voice Production," in *Vocal Fold Physiology: Contemporary Research and Clinical Issues*, edited by D. Bless (College-Hill, San Diego).