Air Pressure Responses to Sudden Vocal Tract Pressure Bleeds During Production of Stop Consonants: New Evidence of Aeromechanical Regulation

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Abstract

Two studies were conducted to evaluate short-latency vocal tract air pressure responses to sudden pressure bleeds during production of voiceless bilabial stop consonants. It was hypothesized that the occurrence of respiratory reflexes would be indicated by distinct patterns of responses as a function of bleed magnitude. In Study 1, 19 adults produced syllable trains of /pʌ/ using a mouthpiece coupled to a computer-controlled perturbator. The device randomly created bleed apertures that ranged from 0 to 40 mm^2 during production of the 2nd or 4th syllable of an utterance. Although peak oral air pressure dropped in a linear manner across bleed apertures, it averaged 2 to 3 cm H_2O at the largest bleed. While slope of oral pressure also decreased in a linear trend, duration of the oral pressure pulse remained relatively constant. The patterns suggest that respiratory reflexes, if present, have little effect on oral air pressure levels. In Study 2, both oral and subglottal air pressure responses were monitored in 2 adults while bleed apertures of 20 and 40 mm^2 were randomly created. For 1 participant, peak oral air pressure dropped across bleed apertures, as in Study 1. Subglottal air pressure and slope, however, remained relatively stable. These patterns provide some support for the occurrence of respiratory reflexes to regulate subglottal air pressure. Overall, the studies indicate that the inherent physiologic processes of the respiratory system, which may involve reflexes, and passive aeromechanical resistance of the upper airway are capable of developing oral air pressure in the face of substantial pressure bleeds. Implications for understanding speech production and the characteristics of individuals with velopharyngeal dysfunction are discussed.

There is controversy regarding reflexive control of oral air pressure during production of stop consonants by normally speaking individuals under experimental conditions of unexpected pressure loss (e.g., Finnegan & Hoffman, 2000; Finnegan, Luschei, & Hoffman, 1999; Kim, Zajac, Warren, Mayo, & Essick, 1997; Zajac & Warren, 2000). While Kim et al. (1997) have suggested that rapid and nonvolitional respiratory responses may occur, Finnegan et al. (1999) have argued that passive aeromechanical processes are sufficient to maintain oral air pressure. We believe that the controversy may have arisen due to interpretation of both passive aeromechanical and active volitional responses as evidence of

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short-latency reflexes by Kim and colleagues. The following information is presented to (a) elucidate these points, (b) help the reader understand the evolution of the controversy, and (c) provide a theoretical background on the role of reflexes during speech production.

A Pressure Regulation Hypothesis

Warren and colleagues have theorized that the use of compensatory speech behaviors by individuals with velopharyngeal (VP) dysfunction reflects a primary motor control strategy to regulate vocal tract air pressure (e.g., Warren, 1986; Warren, Dalston, Morr, Hairfield, & Smith, 1989). Warren (1986) proposed that oral and/or subglottal air pressures are regulated by active changes in respiratory effort and/or airway resistance. Warren (1986) speculated, for example, that compensatory articulations such as glottal stops and pharyngeal fricatives serve to increase airway resistance and minimize pressure loss, even at the expense of perceptual accuracy. Warren (1986) suggested that compensatory articulations are learned responses that individuals often maintain even after surgical management of the VP mechanism. Warren (1986), however, also suggested that the respiratory structures might be controlled by a "possible feedback system" (p. 258) that regulates air pressure reflexively.

Warren et al. (1989) reported that even in the absence of compensatory articulations, individuals with VP dysfunction use respiratory and/or temporal adjustments to regulate oral air pressure. Using a model of the upper airway, Warren et al. noted that pressure fell approximately 10-fold when VP openings increased from 3 to 45 mm^2. In contrast, they reported only a 1.4-fold drop in pressure for speakers who exhibited a similar range in VP orifice areas. Warren et al. interpreted the difference between the passive model and speakers as evidence of active respiratory compensations that optimize oral air pressure levels. Warren et al. acknowledged, however, that some speakers with VP dysfunction might not need to actively increase respiratory effort because of high nasal airway resistance.

Two aspects of the Warren et al. (1989) study need to be emphasized. First, Warren and colleagues did not speculate whether the active respiratory responses that they proposed were learned and/or reflexive in nature. Given that individuals with VP dysfunction are assumed to learn compensatory articulations, it is likely that respiratory responses might also be learned. Second, Warren et al. used a constant flow source to drive the model of the upper airway. The significance of using constant flow is discussed below.

A Constant Pressure Hypothesis

In response to Warren (1986) and Warren et al. (1989), Moon, Folkins, Smith, and Luschei (1993) noted that the respiratory system functions as a constant pressure source (i.e., low output resistance relative to downstream resistance). As such, Moon et al. suggested that the system's "inherent physiologic characteristics" could maintain oral air pressure at reduced but usable levels even in the presence of VP deficits without the need for compensations. Moon et al. (1993) described "inherent physiologic characteristics" as the "musculoelastic" respiratory pump that provides a "pressurized stream of air to the vocal tract" (p. 55). In essence, this means that pressure is regulated by both passive elastic recoil and active muscular processes. Moon et al. supported their hypothesis of a constant pressure pump by presenting evidence from a model of the upper airway that used both constant pressure and flow as respiratory driving sources. Moon et al. reported that when a constant pressure source was used, air pressure dropped minimally while airflow increased as the size of simulated VP openings increased. The investigators noted that these findings were opposite from those obtained with a constant flow source but similar to findings associated with human speakers reported by Warren et al. (1989).

Although the model data presented by Moon et al. (1993) were rather convincing, their overall conclusion does not negate the possibility that speakers with VP dysfunction might use either reflexive and/or volitional responses to increase the output of the constant pressure respiratory pump. Indeed, Moon et al. acknowledged some discrepancies between data from their model and responses of human speakers. Moon et al. reported, for example, that when an inflated balloon was used as a constant pressure source to drive the model, duration of deflation decreased as orifice size increased. Warren et al. (1989), however, reported that duration of oral air pressure remained constant as VP orifice size increased in human speakers. As indicated by Moon et al. (1993), the stability of air pressure duration in humans with VP dysfunction as compared with a model might suggest the occurrence of active compensatory processes.

Experiments Using Sudden Pressure Bleeds

In a series of three experiments, Kim et al. (1997) reported psychophysical and physiological responses to sudden pressure bleeds during production of stop consonants by individuals with normal VP function. In the first experiment, Kim et al. used a motor-controlled valve connected to a mouthpiece to create unexpected bleed openings during production of the syllable /pa/. Kim et al. reported that most speakers began to perceptually detect a pressure loss when the bleed aperture exceeded 14 mm^2. In the second experiment, Kim et al. used the motor-controlled valve to create a bleed aperture of 45 mm^2 during production of the speech sample. Based on oral air pressure responses, Kim et al. (1997) described what they believed to be a short-latency compensatory response following the loss of resistance (see Figure 3, p. 852). In a third experiment, Kim et al. compared responses of speakers with a passive model that used a mechanically inflated balloon as a constant pressure source. Kim et al. reported that when oral air pressure was bled by opening the valve to 10 mm^2, the speakers showed little change in slope of the rise of pressure during the following syllable. In contrast, there was a substantial reduction in slope of pressure with the model. Because the model used a constant but passive pressure source, Kim et al. concluded that the speakers used active compensations to maintain pressure. Kim et al. suggested that the mechanism of active control might involve either the recruitment of expiratory muscles to increase pressure and/or inhibition of postinspiratory muscles to increase elastic recoil. As previously noted by Hixon (1987, p. 47), both of these mechanisms might be involved in the generation of "pulsatile" pressure variations associated with conversational speech.

In their third experiment, Kim et al. (1997) used a bleed aperture (10 mm^2) that they believed was below conscious perceptual awareness of the speakers. Because of this, they stated that the findings demonstrated that "the physiological response does not depend on individuals' volition" (p. 855). Although Kim et al. did not use the term *reflex,* the preceding statement implies reflexive behavior, especially given that the responses reported in the second experiment were short latency (27 ms on average). It must be noted, however, that in the third experiment, Kim et al. measured responses that occurred in the syllable that followed the start of the pressure bleed. It was still possible, therefore, that the participants detected the initial pressure loss either directly or indirectly and consciously compensated during the following syllable. While this would be consistent with active volitional responses, it might not involve reflexes.

Indeed, Finnegan et al. (1999) questioned the occurrence of respiratory reflexes to regulate oral air pressure during bleed conditions. Finnegan et al. attempted to determine the degree to which the respiratory system functions as an "ideal" (or constant) pressure source. An ideal pressure source was defined as how well tracheal pressure reflects alveolar pressure. To estimate alveolar pressure, they developed a method to calculate resistance of the lower airway during the sudden change in airway resistance associated with the release of a voiceless stop (see Figure 2, p. 1140). Finnegan et al. noted that this technique might be able

to shed light on the findings reported by Kim et al. (1997). Accordingly, Finnegan et al. (1999) used vocal tract resistance data from their study in combination with pressure and flow data provided by Kim et al. to show that peak oral air pressure during bleeds could be explained "in terms of the resistances of the system without any active respiratory response." Although Zajac and Warren (2000) acknowledged the validity of this approach, they questioned the analysis of limited data obtained from two different studies. Finnegan and Hoffman (2000) also acknowledged that combining data across studies was "problematic" and called for continued investigation of pressure regulation.

Pressure-Sensitive Reflexes During Speech Production

As indicated by Finnegan and Hoffman (2000), the investigation of pressure-sensitive reflexes might add to our overall understanding of speech production. Wyke (1983), for example, proposed that subglottal mechano-receptors that are sensitive to changes in air pressure reflexively stimulate laryngeal adductor muscles to help regulate vocal fold tension during phonation. Although Garrett and Luschei (1987) called this hypothesis "both attractive and plausible," they reported no obvious electromyography (EMG) evidence of laryngeal adductor activity in response to induced subglottal pressure changes in vocalizing cats. Newsom Davis and Sears (1970), however, reported evidence of respiratory reflexes in response to mechanical loads that were imposed during breathing and phonation in humans. When a load (positive pressure) was applied to the airway of a phonating participant, expiratory intercostal muscles showed a transient silent period (inhibition) followed by increased EMG activity. The latencies of these responses were 22 ms and 50 to 60 ms, respectively. Newsom Davis and Sears suggested that the excitatory phase was mediated by stretch receptors located within the intercostal muscles. Conversely, when a load was decreased during expiration without phonation, consistent inhibition of intercostal muscle activity occurred. Hixon (1987) suggested that similar reflexes might occur during speech production when articulatory activity of the vocal tract "causes very rapid loading and unloading of the respiratory pump" (p. 50). In the present study, we propose that the sudden loss of air pressure during stop production might be a stimulus that elicits respiratory reflexes to compensate for the loss. The pressure stimulus might be detected either directly by upper airway mechano-receptors or indirectly by thoracic stretch receptors.

Purpose of the Present Investigation

Two studies were designed to further investigate the nature of respiratory responses to sudden and unexpected pressure bleeds during production of voiceless bilabial stop consonants. The primary objective was to determine if evidence could be found to support the occurrence of short-latency reflexes. In the first study, a computer-controlled perturbator created bleed apertures ranging from 0 to 40 mm^2 during the rise of oral air pressure. Peak pressure during the bleed, slope of the rise in pressure during the bleed, and duration of the entire pressure pulse during the bleed were determined for a group of normally speaking adults. In the second study, both oral and subglottal air pressure responses of 2 adults were monitored while 20 and 40 mm^2 bleeds were imposed during production of stop consonants. It was hypothesized that the presence of short-latency respiratory reflexes would be reflected by distinct patterns of air pressure responses as a function of bleed magnitude. Specifically, it was expected that (a) peak oral and/or subglottal air pressure and slope of pressure would remain relatively stable across small bleed apertures, (b) peak pressure and slope would drop precipitously at some larger bleed aperture, and (c) duration of air pressure would remain relatively constant across all bleed apertures.

The above hypotheses were expected based on assumptions of regulation theory and evidence from previous studies. Clearly, stability of a response is a fundamental tenet of any regulated system. As noted by Warren (1986), however, there are obvious "limits to

respiratory compensation for palatal inadequacy" (p. 254). Kim et al. (1997) further indicated that the lack of stability does not necessarily imply the lack of regulation. Specifically, Kim et al. (1997) stated, "regulating systems are successful in *normal* [italics added] situations and attempt to maintain constancy in *abnormal* [italics added] situations" (p. 854). Accordingly, we predicted that respiratory reflexes, if extant, will fail to maintain stable pressure when bleed apertures exceed some critical point. We anticipated that this point might occur at an aperture in the range of 10 to 20 mm^2. This tentative prediction was based on previous findings that had suggested that (a) VP gaps up to 5 mm^2 might occur in some adult speakers with normal function (Zajac, 2000) and (b) perceptual symptoms of VP dysfunction begin to occur at openings of 10 mm^2 and typically always occur at openings greater than 20 mm^2 (Warren, 1986). The third hypothesis was based on previous findings that showed that (a) speakers with VP dysfunction tend to maintain constant duration of oral air pressure across increasing orifice area (Warren et al., 1989), (b) normal speakers tend to maintain constant duration of air pressure across increasing bleed magnitude (Kim et al., 1997), and (c) the duration of balloon deflation in model studies tends to decrease as a function of increasing bleed magnitude (Moon et al., 1993).

We further speculated that respiratory reflexes might operate in a manner similar to the "pulsatile solution" for linguistic stress described by Hixon (1987). Hixon (1987) proposed that the respiratory pump generated rapid increases in muscular pressure that were superimposed on the typical "background" levels of pressure required for an utterance. Citing findings from Netsell (1969), Hixon (1987) stated that "these abrupt changes in muscular pressure are of brief duration (about 75 to 150 milliseconds) and involve magnitudes of change in the neighborhood of 1 to 3 cm H_2O during normal-loudness speech" (p. 47). Clearly, both the duration and magnitude of pulsatile responses, if elicited through reflex activity, would be sufficient to regulate (or attempt to regulate) pressure during a sudden bleed.

Finally, the present studies differ from Kim et al. (1997) in several important aspects. First, we manipulated bleed apertures by using a series of electronically controlled miniature valves rather than a single motor-controlled valve. This modification provided increased control over the timing and precision of pressure venting. Second, while Kim et al. used two apertures (10 and 45 mm^2) to determine respiratory responses, we used six apertures, ranging from 5 to 40 mm^2. As indicated above, we anticipated that a wider range of apertures might better reflect the nature and/or limits of responses. A linear drop in pressure and/or slope of pressure with increasing aperture size, for example, might support a passive aeromechanical process, while relatively stable pressures with or without a precipitous drop might suggest active compensatory responses. Last, and most important, we created pressure bleeds and determined respiratory responses during the same syllable to reduce the possibility of volitional responses.

Study 1

Method

Participants—Participants consisted of 22 women (mean age = 22 years, *SD* = 3, range = 20 to 32) and 17 men (mean age = 24 years, *SD* = 7, range = 18 to 43). All participants presented with perceptually normal sounding speech and reported no history of speech, voice, hearing, or respiratory disorders. Informed consent was obtained from all participants.

Speech Sample—All participants produced 65 utterances consisting of 5 repetitions of the syllable /pʌ/ within a single breath group. Each speaker produced 325 syllables. Participants were instructed to use a rate of approximately 2 to 3 syllables per second and habitual loudness during production of all utterances.

Instrumentation—To create pressure bleeds during production of the speech sample, we developed a computer-controlled perturbator. The device consisted of a series of 144 fast-response miniature solenoid valves (1.37 mm port diameter) connected to a common manifold. The manifold consisted of a plastic tube approximately 14 in. long (¾ in. internal diameter) that was closed at one end. As illustrated in Figure 1, the valves were spatially configured along the length of the manifold in three sets of two parallel rows with 24 valves per row. All valves were coupled to the manifold by plastic tubing approximately 5 in. long (2.46 mm internal diameter). The open end of the manifold was connected in series to a heated pneumotachograph (Model 1; Fleisch) and a mouthpiece with an area of approximately 1 cm^2. Oral air pressure (P_o) was detected by a plastic catheter (2.46 mm internal diameter) inserted into the mouthpiece and connected to a pressure transducer (Model MTSAR-S1; Microtronics) referenced to atmosphere. Bleed airflow was detected by the pneumotachograph. The pressure transducer and pneumotachograph were calibrated with a manometer and rotometer at 6 cm H_2O and 250 ml/s, respectively. The pressure transducer was shown to be linear over the range of 0 to 24 cm H_2O while the pneumotachograph was linear over the range of 0 to 1000 ml/s. Pressure and airflow data were digitized directly to a computer using PERCI-SARS software (Version 3.21) and hardware (Microtronics) at a sampling rate of 1 kHz. The pressure and airflow signals were not low-pass filtered, to facilitate interpretation and measurement of pressure, as described below. A microphone was used to record the acoustic signal. The acoustic signal was low-pass filtered (5 kHz) and digitized (10 kHz) directly to the computer.

Bleed apertures were suddenly created during production of the speech sample by opening a predetermined number of valves to create effective areas of 5, 10, 15, 20, 30, and 40 mm^2. This range reflected VP orifice areas typically exhibited by speakers with repaired cleft palate (Warren, 1979, 1986). The number of valves required per aperture area was determined by steady-state airflow tests that created a pressure drop through the manifold and valves by delivering airflow at a rate of 250 ml/s through the pneumotachograph and measuring the pressure drop to atmosphere. Aperture areas were then calculated using the hydrokinetic equation (Warren & DuBois, 1964; Zajac & Yates, 1991). Table 1 lists the number of valves required to create each aperture area. As seen in the table, there was a nonlinear relationship between the number of valves and aperture areas. In addition, the maximum area obtained with all 144 valves was less than expected based on the nominal port size of the valves. These effects occurred due to added resistance introduced by (a) the length of the tubes connecting the valves to the manifold and (b) the spatial configuration of the tubes along the length of the manifold. Although there was a nonlinear relationship between the number of valves and aperture areas, the areas listed in Table 1 were reliably obtained during three repeated steady-state airflow tests. In addition, the reliability of the perturbator to create apertures was determined during the speech tasks, as reported below.

Procedure—The participants were instructed to produce the 65 utterances by closing their lips around the mouthpiece during /p/ and releasing their lips during the vowel. During several practice trials, the participants were monitored by an investigator and given feedback regarding rate or any unusual attempts to produce the speech sample (e.g., without closing the lips completely around the mouthpiece for /p/ and/or opening the lips for the following vowel). All participants were informed that they might sense a loss of pressure while producing /p/ during some of the utterances. They were instructed to continue to produce all five syllables of an utterance even if this occurred. During 30 utterances, the six bleed apertures (5, 10, 15, 20, 30, and 40 mm^2) were created five times during the second syllable. During another 30 utterances, the six bleed apertures were created five times during the fourth syllable. Five utterances were produced without any bleed apertures. The order of the 65 utterances was randomized for each participant. Although the speech task was not tiring,

all participants were given a rest of several minutes after producing approximately 30 utterances.

To create bleed apertures, the computer was programmed to open the valves when P_o H_2O for a duration of 300 ms. Preliminary testing of exceeded 2.5 cm different speakers indicated that these settings ensured that the bleed (a) started during the rise in pressure for /p/ (i.e., during the period of lip closure) and (b) ended after release of the stop. Although fairly quiet, the perturbator did produce audible clicks when the valves were opened and closed. Because this noise might have signaled the participants that a bleed had occurred, they were asked to wear headphones that played white noise into the ears. Each participant was instructed to adjust the dial on a noise generator until the intensity just masked the background noise of the perturbator. The actual level of the masking noise, however, was not monitored and may have created a Lombard effect for some participants. The extent to which this may have occurred is addressed below.

Because of the need to bleed pressure in the study, a mouthpiece was used during production of the speech sample, as described above. To determine the effect of using a mouthpiece on air pressure responses, two additional speakers who did not participate in the study produced /pʌ/ 15 times using a mouthpiece and 15 times using a small plastic catheter (1.67 mm internal diameter) placed behind the lips. The order of production was counterbalanced for the two speakers.

Measurement—Figure 2 illustrates the measurements obtained during production of /p/ in either the second or fourth syllable. PERCI-SARS software automatically determined peak P_o, bleed flow at peak P_o, and aperture area at peak P_o (point D, Figure 2). Aperture areas were obtained as an additional check on the reliability of the perturbator. Slope of P_o was determined from the point where the rise in pressure stabilized following the opening of the valves (point C, Figure 2) to peak P_o. This avoided the momentary pressure oscillations associated with the bleed (points B to C, Figure 2). During production of utterances without a pressure bleed, slope of P_o was determined from the point in which pressure exceeded an arbitrary threshold of 0.2 cm H_2O (point A, Figure 2) to peak P_o. Note that this measurement included a longer P_o segment than when a bleed occurred. The impact of this on the findings is discussed below. The duration of P_o for utterances both with and without pressure bleeds was determined from the point in which pressure exceeded an arbitrary threshold of 0.2 cm H_2O (point A, Figure 2) to the point in which pressure dropped below 0.2 cm H_2O during the release of /p/ (point E, Figure 2).

Statistical Analyses—Differences between the two speakers who produced the speech sample using both a mouthpiece and an oral catheter were evaluated by paired *t* tests. For each of the 39 participants, mean values of peak P_o, bleed aperture area, slope of P_o, and duration of P_o were calculated on the basis of production of the five utterances at each bleed aperture and syllable position. Group data were evaluated using three-factor (sex, aperture size, and syllable position) analysis of variance (ANOVA) procedures with repeated measures. Alpha levels were set at .05 for all statistical tests.

Results

Effects of the Mouthpiece—Neither speaker exhibited significant differences in oral air pressure responses when using a mouthpiece versus using a small-diameter oral catheter. For the first were speaker, means and standard deviations of peak P_o 4.8 cm H_2O (*SD* = 0.5) with the mouthpiece and 4.6 cm H_2O (*SD* = 0.3) with the oral catheter, paired *t*(14) = 0.98, *p* = .345. Mean duration in the rise of P_o was 93 ms (*SD* = 12) with the mouthpiece and 86 ms (*SD* = 19) with the oral catheter, paired *t*(14) = 1.21, *p* = .245. For the second speaker, mean

peak P_o was 7.8 cm H_2O (SD = 0.6) with the mouthpiece and 7.4 cm H_2O (SD = 0.3) with the oral catheter, paired t(14) = 1.79, p = .095). Mean duration in the rise of P_o was 145 ms (SD = 11) with the mouthpiece and 143 ms (SD = 12) with the oral catheter, paired t(14) = 0.65, p = .526. Slope of P_o was not determined because the duration measure effectively included the slope interval.

Reliability—Table 2 lists means, standard deviations, and 95% confidence intervals for bleed aperture areas, calculated during the speech sample for all participants. As indicated, calculated areas during speech were slightly larger than those determined by steady-state airflow tests for the 15- and 20-mm^2 apertures. Overall, however, the data indicate reliable function of the perturbator as shown by relatively small standard deviations and confidence intervals that do not overlap.

An estimate of intrajudge reliability was done by randomly selecting 2 participants and repeating the P_o measurements. All of the second syllables (n = 35) from 1 participant and all of the fourth syllables (n = 35) from the other participant were measured a second time. For peak P_o, exact agreement was obtained between the two sets of measurements. For slope of P_o, a correlation coefficient of .97 (p < .001) was obtained between the two sets of measurements. The mean difference was only 0.2 cm H_2O/s, paired t(67) = 0.53, p = .600. For the duration of the entire P_o pulse, a correlation coefficient of .98 (p < .001) was obtained between the two sets of measurements. The mean difference was only 1 ms, paired t(69) = 0.97, p = .337. High levels of reliability were obtained, given that (a) P_o was measured using an automatic peak detection feature of the computer software and (b) slope and duration were measured using mostly objective criteria, as illustrated in Figure 2.

Peak Oral Air Pressure—Figure 3 (top) illustrates means of peak P_o for all speakers during production of /p/ as a function of syllable position in the utterance (second or fourth syllable) and bleed aperture (0 to 40 mm^2). As illustrated, peak P_o during the no-bleed condition was approximately 2 cm H_2O higher than values typically reported for adult speakers (e.g., Netsell, Lotz, Peters, & Schulte, 1994; Zajac, 2000). Indeed, Zajac reported mean peak P_o of 5.9 cm H_2O (SD = 1.3) for adults who produced an identical speech sample. Although participants in the present study were instructed to use habitual loudness, some speakers may have increased respiratory effort as a result of the Lombard effect induced by self-determined levels of masking noise. If so, then patterns of air pressure responses also may have been altered. To safeguard against this possibility, speakers who exhibited peak P_o that was greater than 7.3 cm H_2O were eliminated from further analysis. This cutoff value was selected because it represents approximately 1 SD above mean P_o reported by Zajac (2000). Accordingly, the bottom of Figure 3 illustrates the responses of 19 participants (10 women and 9 men) whose peak P_o did not exceed the cutoff during the no-bleed condition. As illustrated, these speakers produced the second syllable with slightly but consistently higher P_o than the fourth syllable across all bleed apertures. There was also a systematic decrease in P_o for both syllables across all apertures. A regression analysis revealed that a linear equation accounted for 95% of the variance as a function of bleed aperture. Use of a second-order (quadratic) equation did not account for a substantially greater percentage of the variance.

A three-factor (sex, bleed aperture, and syllable position) ANOVA with repeated measures was used to analyze P_o data for the 19 participants. Because the sphericity assumption for bleed aperture was not met, a conservative F ratio was used to evaluate this factor (Max & Onghena, 1999). Also, a multivariate test, which did not require the sphericity assumption, was conducted. Results indicated a significant main effect of bleed aperture, conservative F(1, 17) = 264.52, p < .001, η^2 = .940, and a significant main effect of syllable position, F(1, 17) = 21.92, p < .001, η^2 = .563. The multivariate test confirmed the significant main effect

of bleed aperture, Hotelling's $F(6, 12) = 116.12$, $p < .001$, $d = 0.983$. There was no significant main effect of sex and no significant two- or three-way interactions. To highlight the effect of bleed aperture, Table 3 lists P_o, collapsed across sex and syllable position, as a function of aperture area for the 19 participants. The table also lists the cumulative change in P_o across aperture areas. Post hoc comparisons of pairwise differences in Table 3 were done using Tukey's honestly significant difference (HSD) tests (Kirk, 1982). Except for the difference between the 0- and 5-mm^2 apertures, all pairwise differences were significant.

Note that the pattern of responses for the entire group of speakers was quite similar to the pattern of the 19 speakers with typical P_o levels (see Figure 3). The linear nature of both patterns suggests that all speakers might have responded to the pressure bleeds in a similar manner.

Slope of Oral Air Pressure—Figure 4 illustrates means of the slope of P_o for the 19 speakers during production of /p/ as a function of syllable position in the utterance and bleed aperture. Slope measurements paralleled peak P_o in that (a) the second syllable was produced with slightly greater slope than the fourth syllable for all apertures except 5 mm^2 and (b) slope decreased systematically for both syllables across all apertures. As shown in Figure 4, slope was substantially greater during utterances produced without a bleed, because the entire pressure pulse, including the relatively rapid rise due to closing of the lips, was measured (Müller & Brown, 1980). A regression analysis of all data in Figure 4 revealed that a quadratic equation accounted for 90% of the variance as a function of bleed aperture. A regression analysis excluding the no-bleed condition, however, revealed that a linear equation accounted for 82% of the variance without a substantial increase using a higher order equation.

A three-factor (sex, bleed aperture, and syllable position) repeated measures ANOVA was used to analyze the slope data for the 19 participants illustrated in Figure 4. Again, the sphericity assumption for the bleed aperture factor was not met. Results indicated a significant main effect of bleed aperture, conservative $F(1, 17) = 37.18$, $p < .001$, $\eta^2 = .686$, and a significant main effect of syllable position, $F(1, 17) = 6.80$, $p = .018$, $\eta^2 = .286$. A multivariate test also confirmed the significant main effect of bleed aperture, Hotelling's $F(6, 12) = 22.79$, $p < .001$, $d = 0.919$). Similar to the ANOVA for peak P_o, there was no significant main effect of sex nor significant two-or three-way interactions. Post hoc comparisons using Tukey HSD tests revealed significant pairwise differences of slope between (a) 0 mm^2 and all other apertures, (b) 5 mm^2 and all other apertures except 10 mm^2, and (c) 10 mm^2 and 40 mm^2.

Duration of Oral Air Pressure—Figure 5 illustrates means and standard deviations (error bars) of the duration of P_o for the 19 speakers during production of /p/ as a function of syllable position in the utterance and bleed aperture. As illustrated, P_o in the fourth syllable tended to be slightly longer than in the second syllable, especially at the larger bleed apertures. Unlike measurements of peak P_o and slope, however, duration tended to remain constant across apertures.

A three-factor (sex, bleed aperture, and syllable position) repeated measures ANOVA was used to analyze the duration data for the 19 participants illustrated in Figure 5. Results indicated a significant main effect for syllable position, $F(1, 17) = 4.93$, $p = .040$, $\eta^2 = .225$. There were no significant main effects for sex or bleed aperture and no significant two- or three-way interactions. As indicated by the relatively small effect size, the mean difference between the second and fourth syllables across apertures was only 7 ms.

Discussion

It was hypothesized that the presence of respiratory reflexes would be reflected by distinct patterns of oral air pressure responses as a function of bleed magnitude. As indicated by the peak pressure and slope results, however, there was little evidence to indicate the occurrence of short-latency respiratory reflexes. Conversely, if reflexes did occur, there was little effect on oral air pressure levels.

If stability of a response is a fundamental tenet of a regulated system, then the present results indicate that peak oral air pressure during production of a stop consonant is poorly regulated during a sudden pressure bleed. Although the pressure drop from the no-bleed to the 5-mm^2 aperture was relatively small in the present study, the overall patterns of decline for both pressure and slope across all apertures do not reflect stability. Kim et al. (1997) reported that slope of oral air pressure of speakers remained stable after a 10-mm^2 bleed compared with a passive model. Kim et al. noted that slope would be expected to decease as a function of increasing bleed aperture in a passive system, essentially the findings of the present study. A possible explanation for these inconsistent findings might involve volitional responses. As previously noted, Kim et al. believed that a 10-mm^2 bleed was below perceptual awareness of the speakers. It is possible, however, that the speakers actually detected the pressure loss and responded volitionally. There is some evidence from the present study to support this speculation. As indicated in Table 3, an average drop in oral pressure of 1.0 cm H_2O occurred from the no-bleed condition to an aperture of 10 mm^2. Research has indicated that individuals can discriminate oral air pressure differences of this magnitude (e.g., Williams, Brown, & Turner, 1987). This finding indicates that the response of the speakers during the syllable after a bleed, as reported by Kim et al., may have been volitional. In the present study, a post hoc analysis did not reveal any evidence of volitional responses occurring in the syllable after a bleed. Note, however, that unlike the procedures used by Kim et al., duration of the bleed did not extend across consecutive syllables in the present study.

Because duration of the pressure pulse remained relatively constant across all bleed apertures, an argument could be made that respiratory reflexes occurred but failed to maintain stable oral air pressure levels. The failure of reflexes might precipitate volitional behaviors by speakers, especially during experiments that use sudden but extended bleeds (e.g., Kim et al., 1997). It is also possible that reflexes and volitional responses may occur together to achieve a goal of pressure regulation. As discussed below, this might be especially likely to occur during natural (i.e., unperturbed) stop production when changes in air pressure are expected due to previous learning. Finally, as indicated by Warren et al. (1989), oral pressure is not a true reflection of subglottal pressure during a bleed condition. It is possible, therefore, that evidence of respiratory reflexes might be revealed by subglottal pressure responses. Accordingly, a second, preliminary study was done that monitored both oral and subglottal air pressure responses of 2 speakers.

Study 2

Method

Participants—Two men participated in Study 2. Participant 1 also had participated in Study 1. The studies were separated by a period of 1 month. Participant 2 was 45 years of age, with no reported history of speech, voice, hearing, or respiratory disorders. Informed consent was obtained from both participants.

Speech Sample—The 2 participants produced 50 utterances, consisting of 7 repetitions of /pʌ/ within a single breath group. They were instructed to use a rate of approximately two to three syllables per second and habitual loudness. An extended-breath group (i.e., 7 vs. 5

syllables) was used on the basis of the results from Study 1. That study showed a small increase in the duration of the fourth pressure pulse, especially at larger bleed apertures. We speculated, therefore, that relative lung volume level might be a factor in eliciting respiratory reflexes. We acknowledge, however, that actual lung volume levels were not monitored.

Instrumentation and Procedure—The magnitude of bleeds induced by the perturbator was limited to the 20- and 40-mm^2 apertures, in addition to the no-bleed condition. This was done for the following reasons. First, peak pressure and slope results from Study 1 did not indicate a critical aperture size, as hypothesized. Second, as noted above, results from Study 1 indicated a small duration effect for the fourth syllable, especially at the larger bleeds. Third, because we monitored subglottal pressure (P_s) using an invasive technique as described below, we wanted to limit the duration of the study to reduce potential risk to the participants.

To detect P_s, a 26-gauge needle with an outer plastic cannula was inserted into the subglottic space through the cricothyroid membrane by an experienced head and neck surgeon (Mark C. Weissler). The needle was withdrawn, and the plastic cannula was connected to a pressure transducer. During 40 of the utterances, a bleed aperture of either 20 or 40 mm^2 was created during either the third, fourth, fifth, or sixth syllable. Each of the 2 apertures occurred during 5 utterances at each syllable. Ten utterances were produced without any bleed apertures. The order of the 50 utterances was randomized for each participant.

Measurement—Air pressure responses were determined for /p/ in the third, fourth, fifth, or sixth syllables of the utterances for the 2 participants, as illustrated in Figure 6. Peak P_o and associated P_s were determined at point D (Figure 6). As illustrated, the rise in P_o peaked slightly before P_s, owing to the effects of the bleed. Slope of P_s was determined from the point in which the rise in P_o stabilized after the bleed (point C) to peak P_o (point D, Figure 6). These were the same criteria used in Study 1. During nonbleeds, slope of P_s was determined from the point in which pressure exceeded a threshold of 2.5 cm H_2O (approximately point C, Figure 6) to peak P_o This threshold was selected because it coincided with the start of imposed bleeds and thus helped to equalize the duration of the bleed and nonbleed segments. As previously noted, we did not attempt to equalize slope segments in Study 1 because of the rapid rise in P_o associated with closing of the lips. In essence, although duration may have been equalized in Study 1, variability in the rise of P_o still might have occurred for some participants. Finally, because glottal voicing information was available, we defined the duration of P_s during /p/ from points A to E in Figure 6.

Statistical Analyses—Means and standard deviations were computed for each measure and participant. Because only 2 participants were studied, inferential statistics were not computed.

Results and Discussion

Limited data were obtained from Participant 1 due to frequent blockage of the P_s catheter that resulted in termination of the procedures after 25 utterances. Results, therefore, focus on Participant 2, who completed the entire protocol. Means and standard deviations of P_o, P_s, slope of P_s, and duration of P_s for Participant 2 are presented in Table 4. Means of both syllable position (based on 5 utterances) and grand means collapsed across syllables are listed. As indicated by the grand means, P_o dropped in a linear manner across bleed apertures. The decrease from 0 to 40 mm^2 was approximately 60%, in keeping with findings from Study 1. The grand means for P_s and slope of P_s, however, tended to remain relatively stable across bleed apertures. These means decreased from 0 to 40 mm^2 by only 7% and

14%, respectively. Duration of P_s also remained relatively constant across bleed apertures, in keeping with duration findings from Study 1.

For Participant 1, data were available only for the sixth syllable. Means and standard deviations were calculated based on three utterances at each bleed aperture. For P_o, means and standard deviations (in parentheses) were 6.9 (0.2), 4.6 (0.4), and 2.4 (0.7) cm H_2O, respectively, across bleed apertures. The decrease from 0 to 40 mm^2 was approximately 65%. For P_s, means and standard deviations were 7.0 (0.2), 5.9 (0.4), and 4.9 (1.1) cm H_2O, respectively, across bleed apertures. The decrease from 0 to 40 mm^2 was approximately 30%. These limited data from Participant 1 also indicate a tendency of relatively stable P_s, as compared with P_o.

The above results indicate the possible occurrence of respiratory reflexes to maintain P_s during a sudden bleed. It may still be argued, however, that normal respiratory processes (i.e., elastic recoil and/or muscular activity) in conjunction with passive resistance of the vocal tract accounted for the obtained pressure (e.g., Finnegan et al., 1999; Moon et al., 1993). As indicated by Finnegan et al. (1999), it is possible to calculate P_o during a bleed that would be developed due to the resistance of the upper airway. Figure 7 illustrates P_o, P_s, and bleed airflow obtained from Participant 2. Figure 7 shows the fourth, fifth, and sixth productions of /pʌ/ with a 40-mm^2 bleed imposed during production of the fifth (middle) syllable. As illustrated, P_o during the bleed (point B) was reduced as compared with the no-bleed conditions (points A and C). Finnegan et al. (1999) calculated expected P_o during the bleed by means of the equation, $P_o = P_a \times R_{lips}/(R_{lips} + R_{lx} + R_{law})$, where P_a is alveolar pressure, R_{lips} is resistance at the lips, R_{lx} is laryngeal resistance, and R_{law} is lower airway resistance. Because the sum of the terms R_{lips}, R_{lx}, and R_{law} represents total airway resistance (R_t), the equation can be rewritten as: $P_o = P_a \times R_{lips}/R_t$. From Figure 7, P_a was estimated as 5.4 cm H_2O by interpolation of P_o (horizontal dotted line) at point B. R_{lips} was calculated from P_o and bleed airflow at point B (2.4 cm H_2O/.517 l/s = 4.6 cm H_2O/l/s). R_t was calculated from estimated P_a and bleed airflow also at point B (5.4 cm H_2O/.517 l/s = 10.4 cm H_2O/l/s). Expected P_o, therefore, was $5.4 \times 4.6/10.4 = 2.4$ cm H_2O. Indeed, this value (rounded from 2.38 cm H_2O) accounts for all peak P_o exhibited by the speaker during the pressure bleed.

Note that the above analysis used estimated P_a from the no-bleed conditions and thus assumed that because respiratory drive (reflected by peak P_s) was relatively constant across the utterance, reflexes did not occur. Conversely, however, it might also be assumed that respiratory drive was constant because reflexes consistently occurred across the utterance. As suggested by Hixon (1987), respiratory reflexes might occur as a result of lip closure (increased load) to regulate the rise in P_s during a stop consonant. Indeed, as illustrated in Table 4 and Figure 7, slope of P_s, also a reflection of respiratory drive, was relatively constant across bleed and no-bleed conditions. As previously indicated by Newsom Davis and Sears (1970), respiratory reflexes that occur during speech production might be conditioned by "previous experience" (p. 712) or learning. Specifically, they suggested that excitatory reflexes might operate only in response to learned or "predictable" (p. 736) loads to prevent inappropriate responses. In the present study, predictable loads occurred when the speakers closed their lips around the mouthpiece, while the sudden pressure bleed was an unexpected event.

It is also possible that laryngeal responses may have occurred to regulate P_s, either in isolation or in conjunction with respiratory reflexes. Because P_s and bleed airflow were obtained, it was possible to calculated laryngeal resistance during bleeds. For Participant 2, laryngeal resistance was calculated at a flow rate of 400 ml/s for utterances produced with

the 20-and 40-mm^2 bleeds. Resistance decreased from the 20-to 40-mm^2 bleed, indicating that laryngeal responses did not occur.

Based on the above air pressure responses, we believe that the occurrence of respiratory reflexes cannot be entirely ruled out. We further acknowledge, however, that the ambiguity of the present findings is due to limitations in methodology. As noted by Kim et al. (1997, p. 855), "EMG data demonstrating increased respiratory muscle activity following venting would provide the strongest evidence for an active, regulated response." We agree that such research is warranted to determine the role of respiratory reflexes during stop production when pressure loads are both predictable (i.e., unperturbed in natural speech) and unexpected (i.e., perturbed under bleed conditions).

Clinical Implications—Individuals with VP dysfunction typically produce obstruent consonants with reduced oral air pressure (e.g., Dalston, Warren, Morr, & Smith, 1988; Laine, Warren, Dalston, Hairfield, & Morr, 1988). Many individuals, however, maintain minimum levels of oral air pressure at approximately 2 to 3 cm H$_2$O regardless of the extent of their VP deficit. Dalston et al. (1988), for example, reported that 50% of speakers with VP gaps greater than 20 mm^2 achieved oral pressure above 3 cm H$_2$O. Dalston et al. (1988) suggested that "at least some of the subjects investigated adopted an active means of maintaining pressure in the face of decreased airway resistance" (p. 216). Such means might include the use of increased respiratory drive and/or articulatory compensations (e.g., a nasal grimace). The results of Study 1, however, indicate that oral air pressures of this magnitude are readily obtained at bleed apertures up to 40 mm^2 by the inherent function of the respiratory system without the need for additional responses. Obviously, the use of increased respiratory drive and/or a nasal grimace may further increase oral air pressure levels.

Given the above, it appears that speakers with normal respiratory function are likely to achieve oral air pressures of at least 2 to 3 cm H$_2$O even in the presence of substantial VP openings. This occurs, as indicated by Warren (1986), due to the effects of nasal passage resistance. The resistance of the nasal cavity will begin to affect pressure when the cross-sectional area of the VP gap exceeds the cross-sectional area of the nasal cavity. In adult speakers, this occurs at approximately 40–50 mm^2 (Vig & Zajac, 1993). At this point, the nose becomes the flow-limiting structure of the VP-nasal passage continuum. As indicated above, if speakers use a nasal grimace, then airway resistance may increase even further.

Although the findings of Study 1 do not provide strong evidence for the occurrence and/or effectiveness of short-latency reflexes, volitional respiratory responses to conditions of either structural VP deficits and/or prolonged experimental pressure bleeds may be likely. Mayo, Warren, and Zajac (1998), for example, reported that speakers with repaired cleft palate and inadequate VP closure exhibited reduced but similar oral air pressure levels compared with speakers with cleft palate and adequate VP closure. Adequate closure was defined as VP areas less than 10 mm^2 during production of *papa*; inadequate closure was defined as areas greater than 10 mm^2. Mayo et al. reported only a 0.5-cm H$_2$O difference between the groups. Mayo et al. suggested that the inadequate speakers might have learned to increase pressure to satisfy the aerodynamic and/or acoustic requirements of speech production. Relative to experimental studies, Sapienza, Brown, Williams, Wharton, and Turner (1996) reported active respiratory adjustments in response to imposed oral air pressure bleeds in a single participant with congenital cleft palate. Openings of various sizes (10, 20, and 30 mm^2) were systematically placed in a palatal obturator worn by the participant. The investigators reported that while oral air pressure decreased as a function of increasing bleed magnitude, the participant increased both lung and rig cage volume excursions. Note also that oral air pressure did not fall below 3 cm H$_2$O, even at the largest bleed. Similarly, Huber and Stathopoulos (2003) reported respiratory compensations to

extended pressure bleeds in a group of normally speaking adults. These investigators demonstrated that speakers terminated lung volume excursions well below resting expiratory levels during conditions of oral air pressure bleeds as compared with a no-bleed condition.

Finally, note that Warren (1986) proposed a theory of pressure regulation to help explain the stigma of compensatory behaviors such as glottal stops that are often used by speakers with cleft palate. In essence, Warren's goal was to show that articulatory deviations might be tolerated by speakers to satisfy some other, perhaps more primary, speech requirement. In this context, note that some studies have reported altered temporal aspects of consonant production by individuals with cleft palate. Warren and Mackler (1968), for example, reported that the duration of oral constriction associated with voiceless stops was greater in speakers with repaired cleft palate than in speakers without cleft palate. They suggested that this was a compensatory response to improve speech intelligibility. Similarly, Forner (1983) reported that the acoustic stop gaps of children with VP dysfunction were significantly longer compared with control children. Forner suggested that individuals with VP dysfunction might require a longer period of time to impound pressure for release of a stop or affricative. Forner further suggested that segment lengthening in general might be due to a combination of factors, including abnormal respiratory drive and/or the physiologic inability to achieve articulatory targets. Regardless of the specific cause of segment lengthening reported in these studies, we believe that it is possible that some speakers with VP dysfunction may choose to use compensatory articulations such as glottal stops to preserve normal temporal patterns of stop production by taking advantage of the relatively stable subglottal pressure head. As indicated by Forner more than 20 years ago, additional research in this area is needed to help explain and manage speech anomalies associated with cleft palate.

Summary—Study 1 indicated that peak P_O during production of a stop consonant was poorly regulated during a sudden pressure bleed. Even so, speakers were able to develop oral air pressure of 2 to 3 cm H_2O during the largest bleeds. We suggest that this occurs due to the inherent physiologic processes of the respiratory system, which may include pressure-sensitive respiratory reflexes, and passive aeromechanical properties of the upper airway. Study 2 indicated that P_s during production of a stop consonant with or without a bleed might be regulated by respiratory reflexes. Due to the preliminary nature of Study 2, additional research is required.

Acknowledgments

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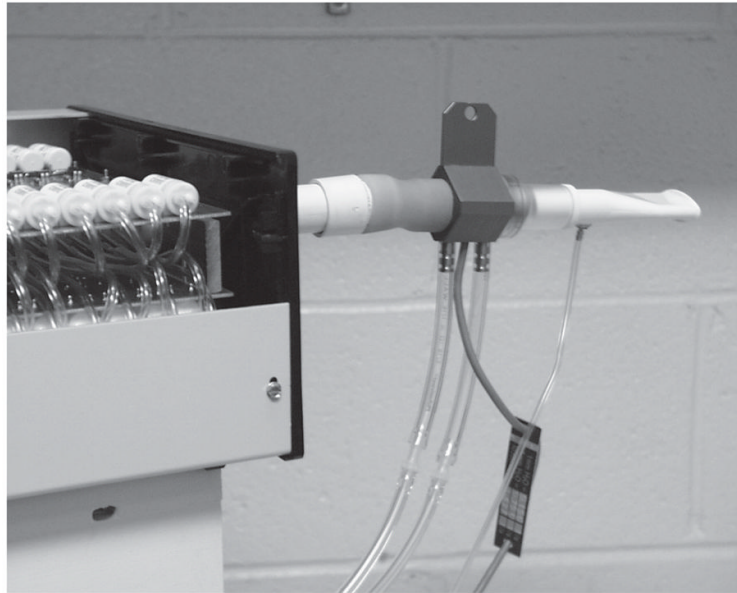
Figure 1.
Photograph of the computer-controlled perturbator with the cover removed. A total of 144 valves (all not in view) were coupled to a common manifold. A mouthpiece and heated pneumotachograph were connected to the open end of the manifold. The mouthpiece contained a catheter connected to a pressure transducer.
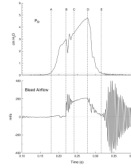
Figure 2.
Oral air pressure (P_O) measurements obtained during /p/: start of pressure (A), start of bleed airflow (B), end of transient pressure oscillations (C), peak pressure (D), end of pressure (E).
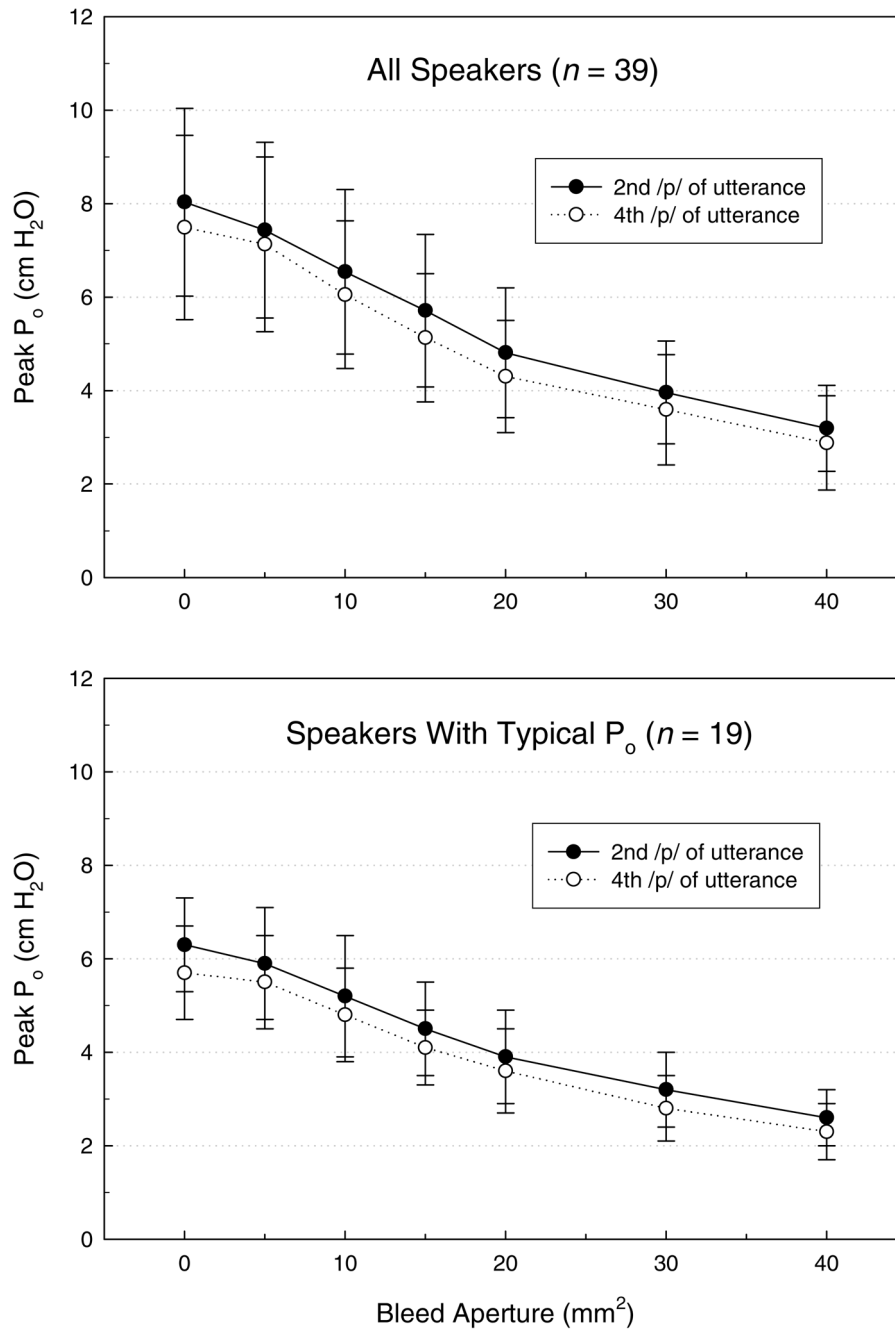
Figure 3.
(Top) Means and standard deviations (error bars) of peak oral air pressure (P_o) for all speakers during /p/, as a function of syllable position and bleed aperture area. (Bottom) Responses of speakers with typical P_o levels.
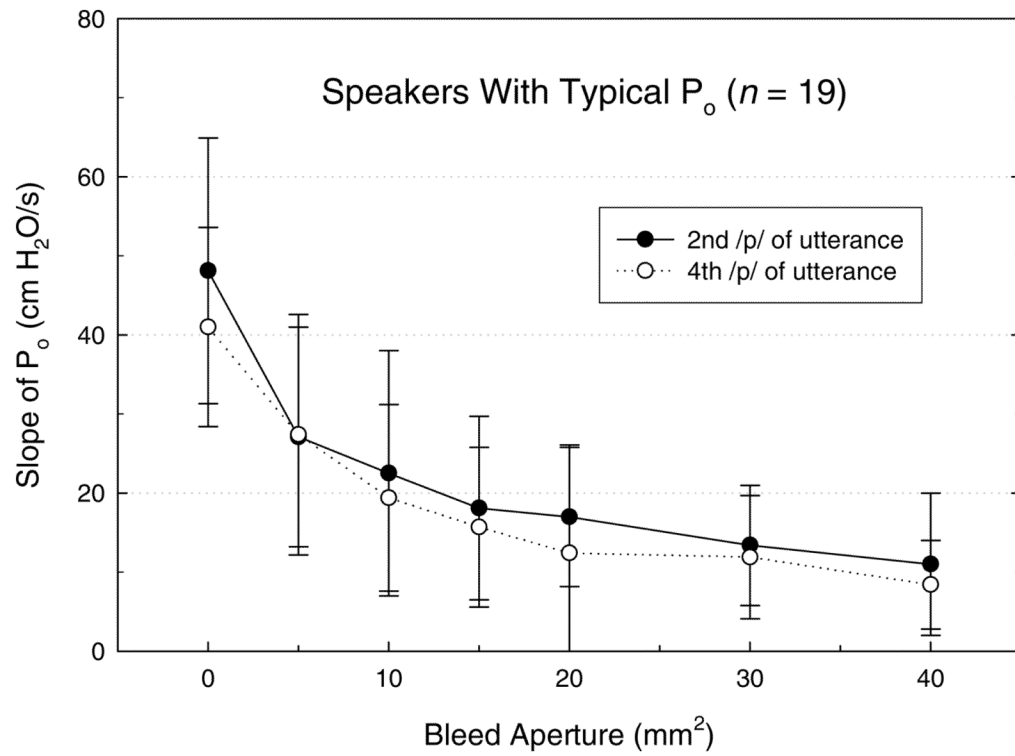
Figure 4.
Means and standard deviations (error bars) of slope of oral air pressure (P_o) for speakers
with typical P_o during /p/, as a function of syllable position and bleed aperture area.
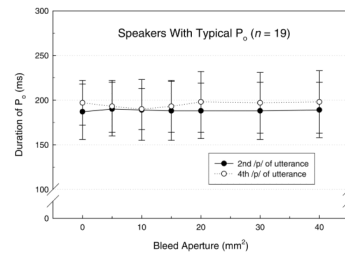
Figure 5.
Means and standard deviations (error bars) of duration of oral air pressure (P_o) for speakers with typical P_o during /p/, as a function of syllable position and bleed aperture area.
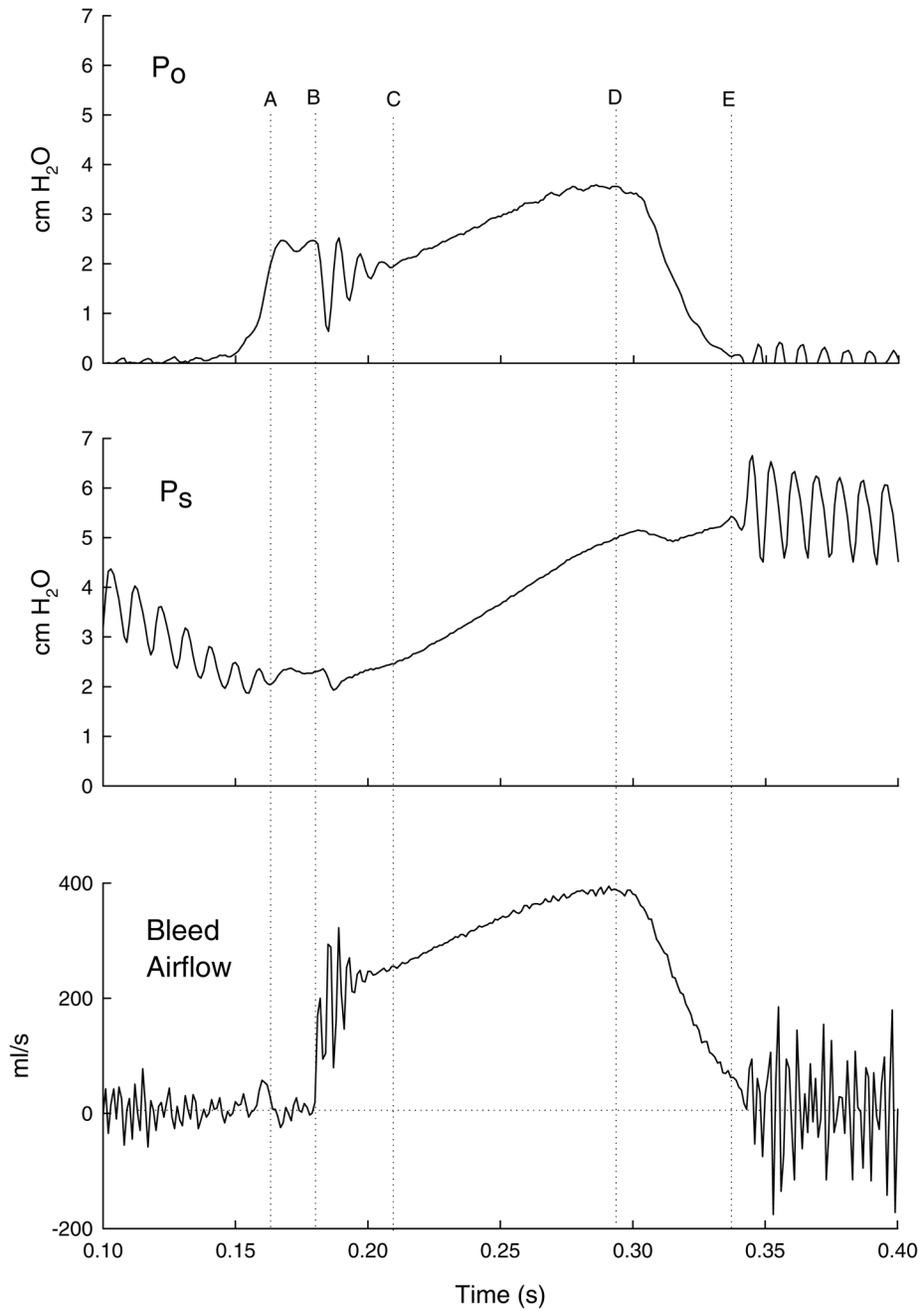
Figure 6.
Illustration of oral (P_O) and subglottal (P_S) air pressure measurements obtained during /p/:
start of P_S (A), start of bleed airflow (B), end of transient P_O oscillations (C), peak P_O (D),
end of P_S (E).
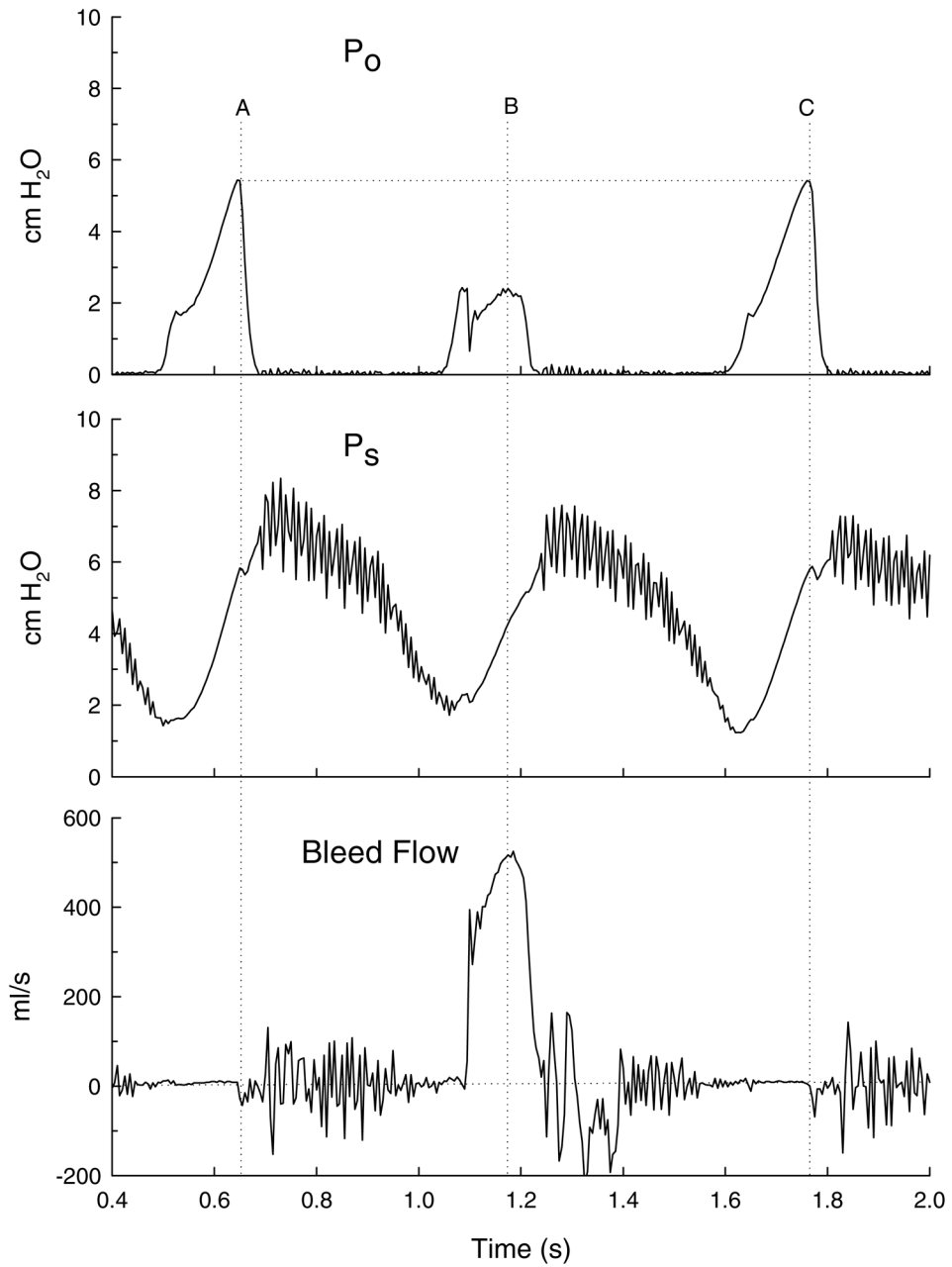
Figure 7.
Illustration of oral air pressure (P_o), subglottal air pressure (P_s), and bleed airflow during production of /p/ with a 40-mm^2 bleed: peak P_o during no bleed (A), peak P_o during bleed (B), peak P_o during no bleed (C).

Table 1

Relationship between number of bleed valves and effective aperture area of the perturbator during steady-state airflow calibration at 250 ml/s.

No. valves	Aperture area (mm^2)
12	5
30	10
48	15
72	20
102	30
144	40

Table 2

Mean bleed aperture areas, calculated during the speech sample for all participants.

	Aperture area during speech (mm^2)					
	Second syllable			Fourth syllable		
Bleed aperture area	*M*	*SD*	**CI**	*M*	*SD*	**CI**
5	4.8	0.4	4.7–4.9	4.7	0.4	4.6–4.9
10	11.0	0.6	10.8–11.2	10.9	0.9	10.6–11.2
15	16.9	0.8	16.6–17.2	16.7	0.7	16.4–16.9
20	23.5	1.9	22.8–24.1	23.4	1.7	22.8–24.0
30	30.8	2.2	30.0–31.5	30.1	2.0	29.4–30.7
40	39.3	3.2	38.2–40.3	37.5	3.2	36.5–38.6

Note. CI = 95% confidence interval. Bleed aperture area (mm^2) was determined by steady-state airflow calibration.

Table 3

Cumulative change in oral air pressure (P_O) as a function of increasing bleed area for speakers with typical P_O levels ($n = 19$).

Bleed area (mm^2)	P_o (cm H$_2$O) [a]	Cumulative change in P_o	
		cm H$_2$O	%
0	6.0	0.0	0.0
5	5.7	−0.3	−5.0
10	5.0	−1.0	−16.7
15	4.3	−1.7	−28.3
20	3.7	−2.3	−38.3
30	3.0	−3.0	−50.0
40	2.4	−3.6	−60.0

[a]Pressure collapsed across syllable position.

Table 4

Means of oral (P_O) and subglottal (P_S) air pressure responses of Participant 2, as a function of bleed aperture and syllable position in the utterance.

Aperture/syllable no.	P_o		P_s		Slope P_s		Duration P_s	
	M	SD	M	SD	M	SD	M	SD
0 mm²								
3	5.5	0.7	5.7	0.7	42.0	11.1	168	11
4	5.6	0.4	5.7	0.4	37.8	5.4	178	10
5	5.5	0.3	5.6	0.4	33.1	7.2	172	13
6	5.3	0.6	5.5	0.6	35.1	8.7	165	9
Grand M	5.5		5.6		37.0		171	
20 mm²								
3	3.6	0.2	5.2	0.2	37.2	2.6	188	11
4[a]	3.7	0.1	5.2	0.3	33.2	2.7	178	14
5	3.8	0.4	5.2	0.5	34.6	11.6	170	9
6	3.6	0.5	5.0	0.6	32.1	9.3	179	9
Grand M	3.7		5.2		34.3		179	
40 mm²								
3	2.4	0.2	4.7	0.3	36.8	4.0	183	20
4[a]	2.5	0.3	4.6	0.5	26.5	5.0	163	12
5	2.5	0.1	4.4	0.2	33.7	5.7	184	14
6	2.4	0.3	4.6	0.6	29.7	3.9	183	9
Grand M	2.4		4.6		31.7		178	

Note. Means Means Means and standard deviations for P_O and P_S are in cm H_2O. Means and standard deviations for slope of P_S are in cm H_2O/s. Means and standard deviations for duration of P_S are in ms.

[a] Means based on four utterances.