TEMPORAL CHARACTERISTICS OF ALVEOLAR STOP CONSONANTS PRODUCED BY CHILDREN WITH VARYING LEVELS OF VELOPHARYNGEAL DYSFUNCTION

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A thesis submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Master of Science in the Department of Allied Health Sciences, Division of Speech and Hearing Sciences.

Chapel Hill 2006

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ABSTRACT

MEREDITH GAYLORD: Temporal Characteristics of Alveolar Stop Consonants Produced by Children with Varying Levels of Velopharyngeal Dysfunction (Under the direction of David Zajac)

Children with cleft palate and velopharyngeal dysfunction (VPD) often present with disorders of articulation, resonance, and phonation. Although limited, previous research has shown that children with VPD, as reflected by hypernasality, prolong stop segment durations. A respiratory drive hypothesis was proposed to account for these findings. As a test of this hypothesis, the current study proposed relationships between velopharyngeal closing time and both voice onset time (VOT) and stop gap duration for the phonemes /t/ and /d/. Digital audio recordings were obtained from 20 children with cleft palate who produced the syllables /t₀/ and /d₀/. Pressure-flow measurements were used to determine velopharyngeal closing durations from the word "hamper." Results indicated a tendency (p > .05) for VOT to decrease as velopharyngeal closing time increased. Results also showed tendencies for children with alveolar clefts to have different segment durations (p > .05). Clinical implications are discussed.

ACKNOWLEDGEMENTS

I would like to thank everyone at the UNC Craniofacial Center for their support and encouragement, especially my thesis advisor, David Zajac, who inspired me to take on this project and who encouraged me the entire way through.

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INTRODUCTION

Previous research has shown higher occurrences of phonatory and articulatory disorders in persons with cleft palate than in the non-cleft palate population. Phonation disorders may occur as a result of vocal fold hyperfunction, and previous research suggests that this hyperfunction may be an attempt to compensate laryngeally for velopharyngeal incompetence. Warren (1986) hypothesized that this hyperfunction was a compensatory strategy resulting from the inability of the person with cleft palate to regulate pressure in his/her oral and nasal cavities. Forner (1983) found that certain speech segments of children with cleft palate tended to be longer than in children without cleft palate. She hypothesized that the prolongation of speech segments in children with clefts might result from an overdriving of the respiratory system in an attempt to compensate for dampened sound energy as well as the physiological inability to achieve articulatory targets. Forner's research implied that children with velopharyngeal dysfunction (VPD), as reflected by perceptual measures, may over-exert their respiratory and laryngeal systems, leading to prolonged speech segments. No research, however, has explored physiologic measures of velopharyngeal function in conjunction with the duration of discrete acoustical features of speech (e.g., stop gap duration and voice onset time). Research examining speech segment durations in relation to velopharyngeal function may help to explain a) compensatory strategies used by children with VPD, and b) speech intelligibility differences among children with cleft palate. The present study examined the relationships among various degrees of VPD in children with cleft palate and stop gap duration and voice onset time (VOT). The following hypotheses

were proposed: 1) As the duration of velopharyngeal closing time increases, the stop gap duration for /t/ will also increase. 2) As the duration of velopharyngeal closing time increases, VOT for /t/ will also increase. These hypotheses are consistent with Forner's (1983) speculation that increased respiratory drive in conjunction with VPD will lead to prolonged speech segments.

REVIEW OF THE LITERATURE

Speech Characteristics Associated with Velopharyngeal Dysfunction. Speakers with improper valving of their velopharyngeal mechanism can exhibit disorders of phonation, resonance, and articulation. Most cases of velopharyngeal dysfunction (VPD) result from clefts of the palate; however, some cases may be secondary to craniofacial anomalies associated with genetic syndromes or neuromuscular disorders. Still other causes of VPD may result from congenital palatopharyngeal incompetence (e.g., congenitally short palate, reduced palatal bulk, deep pharynx and/or malinsertion of the levator muscles) (McWilliams et al., 1990). Regardless of the cause, improper velopharyngeal valving can lead to disruptions of oral air pressures needed for speech and can result in a variety of both obligatory and compensatory speech disorders. Disordered resonance in the form of hypernasality, hyponasality, cul-de-sac resonance or some combination is one of the most common obligatory characteristics of improper velopharyngeal valving. Hypernasality refers to speech characterized by excessive resonance in the nasal cavity. Hypernasality results when the nasal cavity is inappropriately coupled with the oral cavity during the production of vowels (McWilliams et al., 1990). Conversely, hyponasality refers to speech characterized by too little sound resonating in the nasal cavity and can result from a small nasal passage or from surgical procedures that may block airflow to the nose such as a pharyngeal flap (McWilliams et al., 1990). When sound enters the nasal passage, but is prevented from exiting because of a nasal obstruction, cul-de-sac resonance (i.e., a muffled speech quality) may occur (McWilliams et al., 1990).

Nasal air emission can be another obligatory characteristic of speakers with cleft palate. This phenomenon most commonly occurs during the production of pressure consonants rather than vowels (McWilliams et al., 1990). Nasal air emission can be audible, inaudible or turbulent, and can result both from VPD and/or an oral-nasal fistula. Some nasal air emission may be learned (i.e., compensatory) and phoneme specific, most commonly associated with sibilant sounds (Kummer, 2001).

Articulation errors in speakers with VPD can be either obligatory or compensatory in nature. Obligatory articulation errors occur as a direct result of the velopharyngeal structural anomaly and include weak pressure consonants (Sussman, 1992) as well as substitutions of nasal consonants for oral stops (e.g., /m/ for /b/). Compensatory articulation errors (e.g., glottal stops) refer to sounds that are learned and habituated in the presence of poor velopharyngeal structure and function (Phillips and Kent, 1984). Warren (1986) suggested that compensatory speech behaviors are used to maintain a normal aerodynamic environment in the presence of a disordered velopharyngeal mechanism. Typically, children with VPD compensate by producing sounds further back in the oral, pharyngeal, or glottal cavities. Glottal stops, pharyngeal and palatal fricatives, pharyngeal stops and mid-dorsum palatal stops are some compensatory behaviors reported in the literature (McWilliams et al., 1990; Trost, 1981). In addition to VPD, children with cleft lip and palate often present with abnormal dentition, oral-nasal fistula, and/or unrepaired clefts of the alveolar ridge (McWilliams et al., 1990). Depending upon the severity of these conditions, some children may have difficulty producing stop-plosive sounds that require the tongue tip to articulate with the alveolar ridge. Sibilant sounds that require a precise channeling of airflow through the oral cavity may also be affected by abnormal oral structures.

<u>Voice Disorders and Compensatory Strategies.</u> Though less understood, many studies also show a high prevalence of voice problems in speakers with velopharyngeal dysfunction (VPD). Some information regarding vocal problems and cleft palate and/or VPD is available in the literature; however, results vary among studies regarding the frequency and nature of these problems. Voice problems refer to disordered phonation at the level of the larynx and can include hoarseness, breathiness, low volume, and/or abnormal pith (McWilliams et al., 1990). Though the larynx is the primary structure for voice production, this system also requires the integration of the respiratory system and the oral and nasal branches of the vocal tract. Due to the integrated nature of the speech system, problems at the level of the velum may affect the functioning of the larynx.

Brooks and Shelton (1963) found that among 76 children with cleft palate, 10 percent showed evidence of hoarseness. In contrast, a survey study by Takagi et al. (1965) showed that of 616 patients seen at the Lancaster Cleft Palate Clinic, only 0.6 percent had a voice disorder at the time of evaluation. Marks et al. (1971) stated that differences in prevalence rates may be attributed to age of sample, varying definitions for voice disorders, and lack of standardized rating systems. Marks et al. found that of 102 cleft and non-cleft subjects between 6 and 22 years old, 34% had voice quality ratings that deviated from normal according to a laryngeal voice quality rating scale.

McWilliams et al. (1969) used laryngoscopic evaluation to study 32 children with both cleft palate and hoarseness. These investigators found that 84% of the children had vocal fold problems. The most common problem was bilateral vocal fold nodules that occurred in 71.9 percent of the subjects. Of those children, 59 percent had borderline velopharyngeal valving. In a follow-up study, McWilliams et al. (1973) found that surgical

management of borderline velopharyngeal closure did seem to eliminate nodules in some subjects. In a more recent study of voice disorders in children and adults with VPD, D'Antonio (1988) found that 41% of subjects had either laryngeal abnormalities, abnormal voice characteristics or both. This study examined a group of 85 patients with VPD with or without cleft palate between the ages of three and fifty-two years old. The results showed no statistically significant difference in the incidence of abnormal laryngeal or voice findings in the patients with clefts as compared to the ones without clefts. The most frequent vocal symptoms were harshness, abnormal pitch, and reduced loudness. In contrast to earlier studies (e.g. Brooks and Shelton, 1963) D'Antonio did not rely solely on perceptual characteristics of vocal function.

Several hypotheses have been offered in the literature to explain differences in the laryngeal functioning of children with and without velopharyngeal dysfunction (VPD). Warren (1986) hypothesized that children with VPD exaggerate laryngeal gestures to compensate for a decrease in oral pressure. In an attempt to maintain the required pressures for speech, Warren suggested that children with cleft palate hyper-adduct their vocal folds in order to control airflow and provide the resistance needed for speech. In essence, because children with VPD cannot create adequate oral pressure for many sounds, they compensate by creating this pressure at the level of the larynx. This theory of laryngeal tension (i.e., hyperfunction) may lead to perceptual characteristics of disordered phonation such as harshness and/or strain.

Forner (1983) suggested that children with VPD may use increased respiratory drive compared to children with normal velopharyngeal structures. Her research included 15 children with cleft palate and 15 controls between five and six years old. Of the participants

with cleft palate, 12 had cleft lip and palate (CL/P) and three had cleft palate only (CPO). Relative to participant selection, any child with substitution or omission errors was excluded from the study. A seven-point equal appearing interval scale was used to rate the conversational speech of both cleft and non-cleft children for hypernasality and intelligibility. As dependent measures, Fornerexamined segment durations of voiceless stops and affricates. These segments included stop gap and voice onset time in single words and nasal and non-nasal sentences. Results showed significantly longer total sentence durations for sentences containing nasal and non-nasal sounds for the experimental group as well as longer voice onset times for participants (n=5) who were rated the highest for hypernasality and unintelligibility (i.e., received a 4-7 on the seven-point equal appearing interval scale). It should be noted that participants, based upon inclusion criteria, were not highly unintelligible. Forner's (1983) intelligibility ratings, therefore, were only relative in nature.

Forner (1983) hypothesized that children with repaired or unrepaired cleft palate increase their respiratory drive both to compensate for reduced intensity (i.e., from oral-nasal coupling) and to reach articulatory targets in the presence of a disordered or previously disordered speech mechanism. Forner further noted that "overdriving" of the respiratory mechanism may result in a "stress-like emphasis on all utterances." Although Forner did not explicitly identify abnormal use of the respiratory mechanism as a cause of voice disorders, her findings have clear implications relative to VPD and vocal characteristics.

In a study of vocal quality characteristics in children with cleft palate, Van Lierde et al. (2004) also noted that when VPD occurs, vocal intensity is reduced due to damping of acoustics by the nasal cavity. Consistent with Warren's (1986) theory of laryngeal tension

mentioned above, Van Lierde et al. suspected that children use laryngeal tension (i.e., increased vocal fold adduction) as one way of increasing loudness.

<u>Voice Onset Time</u>. Voice onset time (VOT) has been used as a measure of consonant voicing in many studies across various ages and populations (Klatt, 1975; Zlatin and Koenigsknecht, 1976; Forner, 1983). VOT refers to the duration between the release of a complete articulatory constriction and the onset of phonation (Lisker and Abramson, 1967). In her study examining acoustic characteristics of children with cleft palate, Forner (1983) found that VOT was significantlylonger for voiceless plosives in children with cleft palate who were both unintelligible and hypernasal compared with normal controls. Overall, she found the prolongation of segments (i.e., stop gap duration) to be a consistent characteristic in the group with cleft palate. Forner hypothesized that this "segment lengthening" may be due in part to overdriving of the speech mechanism. That is, increased respiratory drive may lead to utterances that are over-emphasized and, therefore, prolonged. She also speculated that segment prolongation may be a compensation for reduced loudness. Forner's (1983) findings support Warren and Mackler's (1968) hypothesis that speakers with cleft palate may prolong speech segments such as VOT to provide more pronounced acoustic cues for the listener.

In normal speakers, contrasts between voiced-voiceless stops are determined by VOT clues. For example, /t/ is primarily distinguished from /d/ based upon a delay of voicing for /t/ that occurs during the release phase of that sound. VOT is also associated with place of stop production. For example, VOT is longer for /k/ as compared to /t/ due to a smaller volume of the vocal tract between the vocal folds and the point of articulatory occlusion (Weismer, 1980).

Baken (1987) suggests that VOT is a variable that "summarizes a very complex and extremely important aspect of articulator-laryngeal coordination" (p. 375). It is a measure that is well-documented in the literature, especially relative to VOT change in speech acquisition. For example, Zlatin and Koenigsknecht (1976) found that VOT values exhibited a developmental pattern of change among two-year olds, ten-year olds, and adults. Results showed that two- and six-year old children primarily fell within the short lag range (i.e., short VOT) for voiced stops while adults presented with short lead times (i.e., pre-voicing). At the time of speech onset, young children present with a unimodal distribution of VOT meaning that all phonemes resemble voiced phonemes (i.e., short lead or short lag times). By the age of six, voiceless stops become more distinct from the voiced cognates. This results in less VOT overlap between voiced and voiceless phonemes (Baken, 1987). The bimodal VOT distribution for voiced and voiceless phonemes reaches adult-like maturity between the ages of eight and eleven (Kent, 1976).

<u>Pressure-Flow Analysis</u>. Because speech relies on the build-up of air-pressure as well as airflow, aerodynamic measurements within the oral and nasal cavities can provide objective information about the structure and function of the speech mechanism (Zajac, 2001). Pressure-flow is a non-invasive aerodynamic procedure first described by Warren and DuBois (1964) to assess velopharyngeal function. Zajac (2001) describes pressure-flow as a procedure that can determine oral pressure levels, rates of nasal air emission, estimates of velopharyngeal orifice size during consonant production, timing aspects of velopharyngeal function, and patency of nasal airways. By determining differential oral-nasal pressures during stop consonants, an estimation can be made about the size of velopharygeal opening.

Pressure-flow instrumentation uses small-bore catheters to determine oral and nasal pressures and a tube to obtain nasal air flow during speech. As a child speaks, oral pressure is detected by a catheter placed in the mouth while nasal pressure is detected by a catheter secured by a cork or foam plug placed in the nostril. A flow tube inserted in the opposite nostril is used to detect nasal airflow. The oral and nasal catheters are connected to pressure transducers which convert the air pressure into electrical signals for further processing (Zajac, 2001). The nasal flow tube is connected to a pneumotachograph. This device determines the rate of airflow by measuring the differential pressure drop of the air when channeled through a bundle of small tubes. Oral air pressure, nasal air pressure, and rate of nasal airflow are analyzed with commercially available software.

Zajac (2000) used pressure-flow analysis to determine normal velopharyngeal orifice areas for children and adults producing /p/ during the word "hamper" by examining the differential pressures in the oral and nasal cavities. Results showed that normal speakers between 6 and 12 years of age had velopharyngeal orifice areas ranging from 0.0 to 1.6 mm² at the word level (repetitions of "hamper") and from 0.0 to 2.5 mm² at the sentence level ("hamper" embedded in a phrase). Zajac and Mayo (1996) also used pressure-flow analysis to examine timing aspects of the velopharyngeal mechanism in normal adults. Timing measurements of nasal airflow between the /m/ and /p/ segments of the word "hamper" showed that adult males had a mean velopharyngeal closing time of 78 ms and adult females had a mean velopharyngeal closing time of 82 ms (see Figure 1).

Dotevall et al. (2001) also used nasal airflow timing measures to examine dynamic aspects of velopharyngeal function in children with and without cleft palate. The study examined aspects of nasal flow during the transition between nasal consonants and oral

consonants (e.g., /mp/) in Swedish. Results showed that nasal airflow declination from a nasal consonant to a stop was slower in the subjects with cleft palate in comparison to the non-cleft controls. That is, the amount of time it took for peak airflow to decline to 5% of baseline airflow during the nasal-stop transition was significantly longer in the children with cleft palate. In addition, the rate of airflow was higher during the nasal-stop transition for the cleft palate group. These results suggest that analysis of nasal airflow during the velopharyngeal closing phase associated with a nasal-plosive sequence can reveal important timing aspects of the velopharyngeal mechanism. Based upon these findings, the present study used velopharyngeal closing time determined from the word "hamper" as a primary index of velopharyngeal function. Velopharyngeal orifice area was also used as an index to explore some secondary questions.

<u>Statement of Purpose.</u> Although limited, previous research suggests that children with velopharyngeal dysfunction (VPD) may prolong sound segments. The current study attempted to overcome limitations noted in previous research. Forner (1983) defined velopharyngeal dysfunction by perceptual characteristics and her conclusions were based on this perceptual data. The purpose of the current study was to explore the respiratory drive hypothesis as it relates to children with cleft palate and varying levels of VPD as determined by objective measures. Specifically, the relationships between VPDas indexed by velopharyngeal closing time and segment durations of the phonemes /t/ and /d/ were determined. The following hypotheses were proposed: 1) As the duration of velopharyngeal closing time increases, the stop gap duration for /t/ will also increase. 2) As the duration of velopharyngeal closing time increases, voice onset time (VOT) for /t/ will also increase.

METHODS

<u>Participants.</u> Nine females and 11 males (n=20) between the ages of 5 and 11 years participated in this study. The mean age was 7.8 years (SD=1.7). Subjects were recruited during scheduled clinical speech evaluations at the UNC-CH Craniofacial Center. These children were seen for speech evaluations either as part of regular team care for repaired cleft lip and/or palate and/or by referral for suspected velopharyngeal dysfunction. During the speech evaluation, all children were audio-tape recorded to document speech articulation errors. In addition, all children were evaluated by pressure-flow procedures (see below).

Participants were recruited based upon medical chart review and findings from the clinical speech evaluation. To be included in the study, all subjects a) were between the ages of 5 and 11 years, b) passed a pure tone hearing screening (see below), and c) spoke English as their first language. Participants' perceived hypernasality and nasalair emission ranged from within normal limits to severe based on subjective clinical perceptual evaluation. Children with normal perceptual characteristics were not excluded because the focus of the study was on physiologic function of the velopharyngeal mechanism as described below. Children were excluded, however, if they presented with compensatory errors such as glottal stop substitutions associated with the speech sample (see below). Participants were further excluded if they presented with a) known genetic syndromes, b) neuromuscular motor disorders, c) sensorineural hearing loss, or d) pulmonary and/or airway problems. All subjects passed a pure tone hearing screen in a sound-attenuated both at 25dB HL for the frequencies at 1, 2, and 4 kHz in the better ear. Parents of children who met the above criteria

were presented with a written description of the study and verbal consent for the child to participate was obtained. In addition, the study was explained to the child and verbal assent was obtained. The study was reviewed and approved by the UNC-CH Biomedical IRB.

Of the 20 participants, 13 had cleft lip and palate (CL/P) and 7 had cleft palate only (CPO). Of the 13 children with CL/P, 12 had unrepaired alveolar clefts and one had alveolar bone grafting completed during the previous year. Four children had secondary pharyngeal flaps and one had a sphincter-pharyngoplasty; 15 children had no secondary procedures. Two of the children had an oral-nasal fistula (ONF). Table 1 summarizes the number of participants relative to sex, age, type of cleft, ONF, secondary surgical procedures, and perceptual characteristics of speech.

Speech Sample and Instrumentation. Participants were audio-tape recorded in a soundattenuated booth to minimize background noise during the recording of speech. The investigator and child were seated at a small table and the child's speech was recorded with a miniature head-mounted condenser microphone (AKG Model C420) and portable DAT recorder at a sampling rate of 44.1 kHz (Tascam, Model DA-PI). The investigator explained the speech sampling procedure according to the testing script (see Appendix). Two 3x5 inch index cards with the sentences "Say ta again" and "Say da again" were used to elicit target productions. A neutral vowel was chosen for the target syllable to minimize tongue movement and co-articulatory effects. Before recording began, the child was given the opportunity to practice each sentence after a model by the investigator while shown the corresponding card. Once recording began, the child was shown the first card (/tx/) five times with an approximately 3 second pause between each production. The child was then shown

the second card $(/d_A/)$ five times with an approximately 3 second pause between productions.

The procedure was then repeated one more time so that each child produced a total of ten $/t_{\rm A}/$

phrases and ten $/d_{\Lambda}$ phrases. Half of the children produced $/t_{\Lambda}$ first and half produced $/d_{\Lambda}$

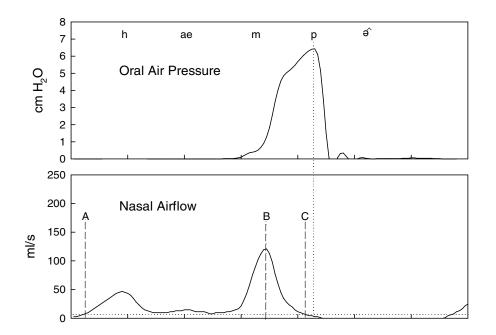
first.

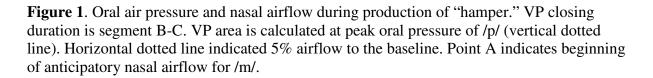
SPEAKER	SEX	AGE	CLEFT TYPE	ALVEOLAR CLEFT	ONF	SECONDARY SURGERY	_	EPTUAL TERISTICS
							NE	NR
1	Μ	9	CL/P	Y	Ν	Y*	WNL	Mixed
2	F	5	CL/P	Y	Ν	Ν	VNE	WNL
3	Μ	7	CL/P	Y	Ν	Ν	ANE	Hyper
4	Μ	8	СРО	N	Ν	Y*	ANE	Mixed
5	F	9	CL/P	Y	Ν	Y*	WNL	Нуро
6	Μ	10	CL/P	Y	Ν	Ν	VNE	Hyper
7	F	7	СРО	N	Ν	Ν	WNL	Hyper
8	Μ	11	СРО	N	Ν	Ν	WNL	WNL
9	F	6	СРО	N	Ν	Ν	VNE	WNL
10	Μ	7	CL/P	Y	Ν	Ν	WNL	Hyper
11	F	11	CL/P	Y	Ν	Ν	WNL	Нуро
12	М	9	CL/P	Y	Ν	Ν	ANE	Mixed
13	F	5	CL/P	Y	Y	Ν	VNE	Hyper
14	F	8	CL/P	Y	Ν	Ν	VNE	WNL
15	М	8	CL/P	Y	Y	Ν	ANE	WNL
16	М	7	CL/P	Y	Ν	Ν	VNE	Hyper
17	Μ	6	CL/P	Y	Ν	Ν	WNL	Нуро
18	F	8	СРО	Ν	Ν	Y*	ANE	Hyper
19	Μ	7	CPO	Ν	Ν	Y±	ANE	Hyper
20	F	7	CPO	Ν	N	Ν	WNL	Нуро

Table 1. Summary of participants' sex, age, cleft, oral-nasal fistula, surgery, and perceptual characteristics.

Notes: *=PHARYNGEAL FLAP ±=SPHINCTER PHARYNGOPLASTY ONF= ORAL-NASAL FISTULA CL/P= CLEFT LIP AND PALATE CPO=CLEFT PALATE ONLY NE=NASAL EMISSION NR=NASAL RESONANCE WNL=WITHIN NORMAL LIMITS ANE=AUDIBLE NASAL AIR EMISSION

VNE=VISIBLE NASAL AIR EMISSION HYPER=HYPERNASALITY HYPO=HYPONASALITY <u>Pressure-Flow Assessment</u>. As part of a clinical pressure-flow evaluation, each child produced the word "hamper" approximately 5 times to determine velopharyngeal (VP) orifice size and VP closing duration. VP closing duration was defined as the time from peak nasal airflow associated with the /m/ segment of "hamper" to 5% of baseline airflow (see Figure 1). VP orifice measurements were obtained during peak oral air pressure associated with the /p/ segments (see Figure 1). VP orifice areas were determined using the hydrokinetic equation (Warren and DuBois, 1964). Based on Bernoulli's principle, this equation calculates the size of the VP opening based upon simultaneous measures of the differential pressure across the orifice and the rate of airflow (Zajac, 2001). Figure 1 illustrates the points at peak nasal airflow and peak oral pressure that correspond to the /m/ and /p/ segments, respectively, in a normal adult male.





Acoustical Data Analysis. The stop gap duration and voice onset time (VOT) were determined for the syllables $/t_A/$ and $/d_A/$ using the Computerized Speech Lab (Kay Elemetrics, Model 4400). Both a spectrogram and waveform were used to analyze the sound segment durations. In reference to spectrographic analysis, VOT was defined as the time from the onset of the release-burst to the first vertical striation representing phonation (Lisker and Abramson, 1964, 1967). Voicing lead time or prevoicing was recorded when observed. The stop gap was defined as the time from the closed phase of the consonant to the beginning of the release burst of the stop. To facilitate these measurements, voice period marks were enabled on the waveform as part of the CSL software. In reference to the waveform analysis, the stop gap was represented by a flat line (i.e., no glottal pulsing) except in the cases of nasal frication or stop gap voicing. In those cases, irregular frication or continuous glottal pulsing was evident on the waveform. VOT was recorded as the first consistent glottal pulse following the stop gap. Total syllable duration for $/d_{\Lambda}/$ and $/t_{\Lambda}/$ was recorded to calculate a normalized stop gap and VOT. Stop gap and VOT were normalized by calculating a ratio between the segment interval and the total syllable(s) duration. It was anticipated that normalized measures would account for differences in speaking rate, if extant, that might affect segment durations.

Finally, although the focus of the study was on acoustic measurements of the targeted speech samples, an informal assessment of the perceptual acceptability of /t/ and /d/ for each child was obtained. The investigator rated each of the ten productions of /t/ and /d/ as either "acceptable" or "unacceptable" representations of the target phonemes. It was anticipated that this information might shed light on factors underlying speech acceptability for these phonemes.

Statistical Analysis. The following descriptive statistics were calculated for each participant based upon 10 productions of the target speech sample: means and standard deviations (SD) for the stop gap and VOT for /t_n/ and for the stop gap and VOT for /d_n/. In addition, means and SDs were calculated for normalized stop gap data for /t/ and /d/. Means and SDs for VP area and VP closing duration from "hamper" were calculated for each child based upon 3-6 productions. To evaluate the hypotheses, Pearson product moment corelations were calculated to examine relationships between a) VP closing time and the stop gap duration, and b) VP closing time and VOT. As a secondary analysis, differences between children with and without alveolar clefts fostop gap duration were evaluated by means of independent t-tests. Significance levels were established at 0.05 for all statistical tests.

<u>Reliability</u>. Intra-observer reliability of the author's measurements of stop gap durations and VOTs was estimated by randomly selecting two participants and re-measuring all segment durations. This resulted in a total of 40 repeated measurements for both stop gap duration and VOT (i.e., 2 participants x 10 productions x 2 syllables). Inter-observer reliability was estimated by having a second investigator (the thesis advisor) independently measure the stop gap duration and VOTs of the two selected participants.

Pearson product moment correlations between the author's repeated measurements of stop gap duration and voice onset time (VOT) were .999 (p<.001) and .956 (p<.001), respectively. The mean difference between repeated measurements for stop gap duration was less than 1 ms while the mean difference between repeated measurements for VOT was 2 ms. Pearson correlations between the author's and a second investigator's measurements of stop gap durations and VOT were .995 (p<.001) and .939 (p<.001), respectively. The mean difference between investigators for stop gap duration was 5 ms while the mean difference

between investigators for VOT was 6 ms. These findings indicate satisfactory intra- and inter-observer reliability.

RESULTS

To determine if normalized data were comparable with absolute (i.e., nonnormalized) data, correlations were calculated between the normalized stop gap for /t/ and /d/ and the absolute measures for both phonemes. The correlation between the normalized /t/ stop gap and the non-normalized /t/ stop gap was 0.951 (p<.001). The correlation between the normalized /d/ stop gap and the non-normalized /d/ stop gap was 0.948 (p<.001). Given that the normalized stop gaps were highly correlated with the non-normalized stop gaps for both /t/ and /d/, it was assumed that speaking rate was not a significant factor affecting segment durations. Because of this, all results reported below are based on the absolute (i.e., non-normalized) segment durations. Also, it should be noted that due to negative voice onset times (VOT), meaningful normalized VOTs could not be computed for some children.

<u>Descriptive Statistics.</u> Table 2 summarizes participant means, standard deviations (SD), minimum, and maximum values for the stop gap and VOT of /t/ and /d/. The mean overall value for the /t/ stop gap was 142.4 ms (SD=76.4). The minimum value for the /t/ stop gap was 63.9 ms and the maximum /t/ stop gap value was 323.3 ms. The mean overall value for /t/ VOT was 69.0 ms (SD=28.2). The minimum /t/ VOT value was -23.3 ms and the maximum /t/ VOT value was 107.4 ms.

The mean overall value for the /d/ stop gap was 153.7 ms (SD=70.7). The minimum value for the /d/ stop gap was 57.6 ms and the maximum /d/ stop gap value was 347.8 ms.

The mean overall value for /d/ VOT was -13.32 ms (SD=44.5). The minimum /d/ VOT value was -107.8 ms and the maximum /d/ VOT value was 26.3 ms.

Table 2 Summary of descriptive statistics for stop gap and voice onset time for /t/and /d/ inms.

	STOP GAP	VOT
/t/		
MEAN	142.4	69.0
SD	76.4	28.2
MIN	63.9	-23.3
MAX	323.3	107.4
	STOP GAP	VOT
/d/		
MEAN	153.7	-13.2
SD	70.7	44.5
MIN	57.6	-107.8
MAX	347.8	26.3

SUBJECTS (N=20)

Notes: VOT=VOICE ONSET TIME SD=STANDARD DEVIATION MIN=MINIMUM VALUE MAX=MAXIMUM VALUE

Table 3 summarizes the participant means, SDs, minimum, and maximum values for

VP area and VP closing time. The mean VP area value was 5.1 mm² (SD=8.6). The

minimum VP area value was 0.1 mm² and the maximum value for VP area value was 30.2

mm². The mean VP closing time value was 104.1 ms (SD=76.4). The minimum value for VP closing time was 47.0 ms and the maximum value for VP closing time was 176.0 ms.

Table 3. Summary of descriptive statistics for velopharyngeal area and velopharyngeal closing time for each participant.

SUBJECTS (N=20)		
	VP AREA (mm ²)	VP CLOSING TIME (ms)
MEAN	5.1	104.1
SD	8.6	76.4
MIN	0.1	47.0
MAX	30.2	176.0

Notes: VP=VELOPHARYNGEAL SD=STANDARD DEVIATION MIN=MINIMUM VALUE MAX=MAXIMUM VALUE

<u>Correlational Analysis</u>. Relative to the hypotheses of interest, two correlations were computed using VP closing time against a) stop gap duration of /t/ and b) VOT for /t/. The correlation between the stop gap of /t/ and VP closing time was -0.037 (p=.878). The correlation between VOT of /t/ and VP closing time was -.377 (p=.101). Neither of the correlations mentioned above were statistically significant. Figures 2-3 illustrate these relationships.

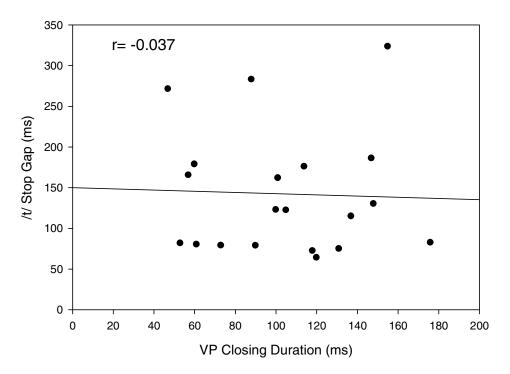


Figure 2. Scatter plot with regression line for velopharyngeal closing duration and stop gap of /t/ for all children (n=20).

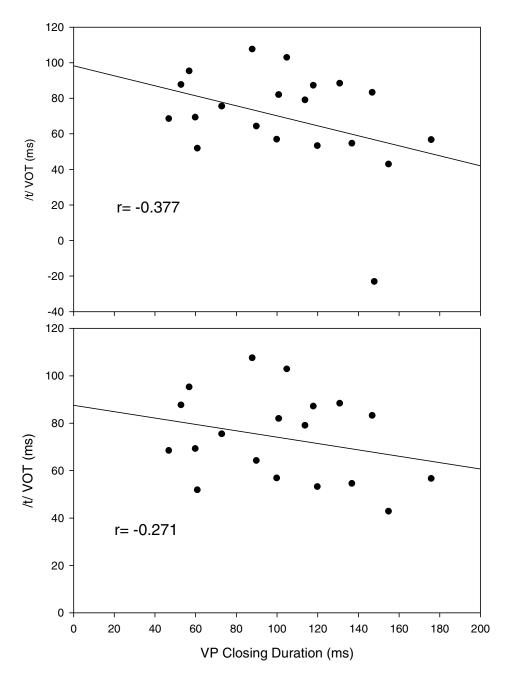


Figure 3. Top: Scatter plot with regression line for velopharyngeal closing duration and voice onset time of /t/ for all children (n=20). Bottom: Same as top but with one outlier omitted (n=19).

An additional analysis was performed that included only children with

velopharyngeal (VP) openings greater than 2.0 mm² (n=7). Correlations using only these subjects were computed between VP closing time and the stop gap duration and VOT for /t/. Although the correlation between VP closing time and the stop gap of /t/ was not strong or significant, the correlation for VP closing time and VOT of /t/ was -.558 (p=.193). Figure 4 illustrates the relationship between VP closing time for those participants with VP area greater then 2.0 mm² and VOT of /t/.

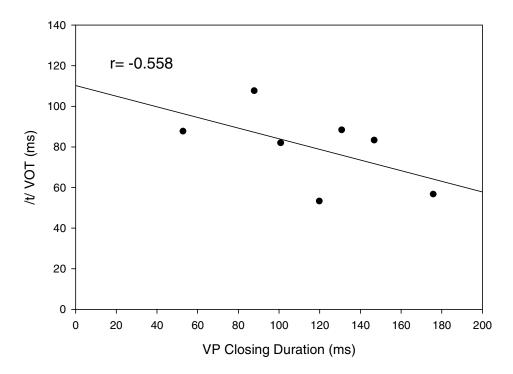


Figure 4. Scatter plot with regression line for velopharyngeal closing duration and voice onset time of /t/ for all children with velopharyngeal openings greater than 2.0 mm² (n=7).

<u>Perceptual Findings</u>. Table 4 below summarizes for each child the percentage of productions of /t/ and /d/ rated as "acceptable" by the investigator. Also in Table 4, mean VP areas and voice onset times (VOT) are listed for each subject. Based on data in Table 4, there appears to be no consistent relationship s among VP area, VOT for /t/ and /d/, and "acceptable" or "unacceptable" perceptual ratings.

Table 4. Summary of velopharyngeal area as well as percent of "acceptable" productions and mean voice onset time in ms of /t/ and /d/ for each participant.

PARTICIPANT	VP AREA (mm ²)	PROD	EPTABLE UCTIONS		VOT (ms)
		/t/	/d/	/t/	/d/
1	0.8	0	0	102.7	23.5
2	0.5	100	100	68.3	24.1
3	0.6	20	10	78.9	-85.1
4	3.9	100	90	83.1	-32.2
5	0.9	100	100	56.7	26.3
6	0.5	100	100	95.1	2.8
7	3.1	100	100	53.1	15.1
8	0.2	100	100	51.7	-93.8
9	1.6	100	100	87	15.1
10	0.1	100	100	69.1	23
11	0.1	100	100	64.1	21.8
12	10.2	100	100	107.4	-61
13	1.1	10	10	54.4	6.3
14	30.2	100	100	88.2	-107.8
15	2.1	100	100	56.5	-49.8
16	22	100	100	81.8	18.7
17	1.6	80	90	42.7	-42.9
18	2	90	100	-23.3	15.8
19	19.4	100	30	87.5	-9.2
20	0.6	100	100	75.3	22.9

Notes: VOT = VOICE ONSET TIME VP= VELOPHARYNGEAL <u>Secondary Analysis</u>. In addition to the primary hypotheses, differences between children with alveolar clefts (either unrepaired or repaired) and without alveolar clefts were determined for the stop gap durations of /t/ and /d/. Children with alveolar clefts often present with increased articulation errors compared to those without alveolar clefts. This analysis was performed, therefore, to determine if differences existed between the subgroups of children. Table 5 presents means and SDs of /t/ and /d/ stop gap durations for the children based upon alveolar cleft status.

	ALVEOLAR CLEFT	NONE
NUMBER OF PARTICIPANTS	13	7
/t/ stop gap		
MEAN	165.7	99.0
SD	81.1	43.9
/d/ stop gap		
MEAN	175.0	114.3
SD	76.1	38.5

Table 5. Summary of descriptive statistics for stop gap duration in ms for /t/ and /d/ in children with and without alveolar clefts.

Notes: SD=STANDARD DEVIATION

The differences between children with and without alveolar clefts were evaluated by means of two independent t-tests. Adjusting the alpha level for two t-tests (i.e., 0.025), the differences between children with and without alveolar clefts for both stop gaps approached significance. The p value for the /t/ stop gap was 0.028 and the p value for the /d/ stop gap

was 0.029. Finally, It should be noted that the difference in VP area between children with alveolar clefts (mean= 5.4 mm^2 , SD=9.6) and without alveolar clefts (mean= 4.4 mm^2 , SD=6.7) was not significant (p=.783).

DISCUSSION

The purpose of this study was to explore the relationships between velopharyngeal (VP) closing time in children with varying levels of velopharyngeal dysfunction (VPD) and sound segment durations for the phonemes /t/ and /d/. The proposed hypotheses were as follows: 1) As VP closing time increases, the stop gap duration for /t/ will also increase, 2) As VP closing time increases, voice onset time (VOT) for /t/ will also increase.

Previous research showed that speech segment durations may be longer in children with cleft palate than in normal children. However, findings from the present study showed a tendency towards shorter speech segment durations. Though not significant, a negative correlation was found between VP closing time and /t/ VOT. That is, as VP closing time increased, VOT decreased. No correlation was found between VP closing time and /t/ stop gap duration. These findings were unexpected given that Forner (1983) reported lengthened segment durations for both stop gap and VOT for voiceless plosives and affricates in children with cleft palate.

One explanation for these conflicting results might be that Forner's (1983) study did not include objective measures of VP function (i.e., pressure-flow analysis). Rather, her results were based strictly on perceived hypernasality and unintelligibility. In addition, Forner reported that speech segment durations were most affected in children who exhibited moderate to severe hypernasality and unintelligibility. By design, children with varying degrees of velopharyngeal dysfunction and perceptual characteristics were included in the present study. Of interest, however, is the additional analysis of children with relatively large VP gaps as discussed below.

Another explanation may be related to the perceptual objectives of children with velopharyngeal dysfunction (VPD). Rather than increasing respiratory drive and prolonging sound segments to increase intelligibility as Forner (1983) had speculated, children with cleft palate in the present study may have actually decreased their respiratory effort in order to camouflage undesirable speech characteristics such as hypernasality and/or nasal air emission. As indicated by McWilliams et al. (1990), some children may present with reduced loudness or "soft-voice syndrome" as a compensatory strategy to decrease the effects of perceived hypernasality. Indeed, Bzoch (1979) found that of 1000 cleft palate patients, 323 had a "weak and aspirate voice," suggesting a compensatory strategy to mask hypernasality. Reduced respiratory drive and loudness, therefore, may have resulted in obligatory reductions of sound segments such as the stop gap duration and VOT.

Conversely, children in the present study might have actively attempted to shorten stop segments to reduce the temporal window for listeners to perceive nasal air emission. For example, spectral noise – most likely resulting from nasal air escape – was often noted during spectrographic analysis of the stop gaps for /t/ and /d/ utterances. Children with velopharyngeal dysfunction, therefore, may be likely to reduce the duration of the stop gap in order to avoid prolonged nasal air emission which can be a perceptually distracting speech quality.

One last explanation for the difference in findings might be due to the relatively small number of participants with moderate to severe velopharyngeal dysfunction. Only a small proportion of the children (n=7) exhibited velopharyngeal areas greater than 2.0 mm^2 . This

means that most of the participants probably had minimal air escape through the velopharyngeal port. The additional analysis that included only children with velopharyngeal openings greater than 2.0 mm² showed a moderately strong negative correlation (-.558) between velopharyngeal closing time and VOT for /t/. At hough the correlation was not significant, it provides some evidence that larger velopharyngeal openings may be associated with reduced speech segment durations. Obviously, additional research should be done to determine if such relationships are due to obligatory and/or compensatory responses of speakers.

<u>Perceptual Data</u>. Based on a single listener, results yielded no apparent differences between VOTs for /t/ and /d/ and the children's ability to produce "acceptable" examples of the targets. That is, VOT did not appear to have a significant effect on productions of the target phonemes. Obviously, other factors such as hypernasality, nasal air emission, and/or articulation errors commonly found in the speech of children with cleft palate may have contributed to the perceptual findings.

<u>Influence of Nasalization on Voicing</u>. Eight of the 20 subjects had some percentage of voicing during the stop gap for /d/. Of the 8 subjects who did voice some portion of the stop gap, 5 had velopharyngeal areas greater than 2.0 mm². These findings suggest that nasalization facilitates voicing. Indeed, Bell-Berti (1980) has suggested that normal speakers may facilitate vocal fold vibration during production of voiced stops by allowing leakage of airflow through the velopharyngeal port in order to maintain a trans-glottal pressure difference necessary for voicing. In addition, Bundy and Zajac (in press) recently found that

some normal adult speakers consistently produced nasal airflow during voiced stop gaps. Children with cleft palate may have little control over the amount of air leakage due to velopharyngeal dysfunction, and the subsequent nasal airflow may actually facilitate voicing during the stop gap. Indeed, it has been reported that children with cleft palate have little difficultly acquiring voiced stops and affricates. Voiceless consonants, however, appear to be misarticulated more frequently than the voiced cognates in these children (McWilliams, 1958; Spriesterbach et al., 1956). Increased rates of nasal airflow may be an important factor in the facilitation of voicing during the stop gap.

Differences Among Subgroups Based on Cleft Type. As part of a secondary analysis, participants in the study were divided into two subgroups based upon cleft type: those with alveolar clefts and those without alveolar clefts. Comparison of these subgroups showed that participants with alveolar clefts had longer stop gaps than those without alveolar clefts. One possible explanation for lengthened stop gap duration in the alveolar cleft group may be related to decreased tactile sensation at the alveolar ridge. Children with alveolar clefts may also present with maxillofacial defects as a result of both palatal surgery and subsequent restricted growth and development (McWilliams et al., 1990). Defects can include dental malocclusions, high palatal vaults, and collapsed palatal arches. It is well known that children with clefts and dental malocclusions have more difficulty producing intelligible speech than those with normal oral structures (Kummer, 2001; McWilliams et al., 1990). The combination of structural anomalies and reduced tactile feedback, therefore, may lead to prolonged alveolar stop gap segments. As an added difficulty, children with alveolar clefts will most likely experience more changes in their oral structures due to surgeries and

orthodontic treatments than children without alveolar clefts. The child with an alveolar cleft, therefore, might have more difficultly reaching correct articulatory targets within a changing and possibly unstable oral structure than the child without an alveolar cleft.

A recent study reported some support for the hypothesis that lack of tactile sensation at the alveolar ridge may lead to longer speech segment durations. Aasland et al. (2006) used acoustical, perceptual, and electropalatography (EPG) analyses to observe the effects of a palatal perturbation on the production of /s/. Normal adult speakers produced /asa/ wearing a thick (6 mm) palatal EPG appliance, a thin palatal EPG appliance, and no palatal appliance. The appliance covered both the hard palate and alveolar ridge. Results showed that after a short period of intense target-specific practice, new strategies were learned for producing /s/ with the thick appliance in place. In fact, new compensatory strategies actually caused negative after-effects on the production of /s/ without the appliance. That is, the production of /s/ without an appliance had changed acoustically, perceptually, and electropalatographically after practice with an appliance. In addition, longer durations were recorded for the production of /s/ in both the thick and thin palatal conditions as compared to no appliance. These results suggest that lack of tactile sensation and feedback at the alveolar ridge may increase the duration of some speech segments.

<u>Limitations.</u> Limitations of the present study include the number of participants and participant characteristics. Due to the relatively small number of participants (n=20), results likely did not reach significance. For example, t-tests run on the same group differences but with an increased number of participants would most likely have reached statistical significance. In addition to sample size, most participants in this study presented with mild

cases of velopharyngeal dysfunction. Of the 20 participants, 13 had velopharyngeal orifice areas less than 2.0 mm². Normal velopharyngeal orifice areas for this age group during production of "hamper" are between 0.0 and 1.6 mm² (Zajac, 2000). As previously mentioned, an additional analysis for participants with areas greater than 2.0 mm² showed a stronger tendency for increased velopharyngeal closing time to be associated with decreased stop gap duration. Future research will need to examine differences between mild and more severe cases of velopharyngeal dysfunction using larger sample sizes.

<u>Implications.</u> Although correlations in the present study were not statistically significant, the results still suggest important clinical relevance. Present results show that children with velopharyngeal dysfunction may shorten speech segments in an attempt to camouflage or mask specific speech characteristics including hypernasality and/or nasal air emission. If so, then speech intelligibility may be affected. Additional studies that include larger numbers of both speakers and listeners are required to determine the effects, if any, of altered segment duration on speech intelligibility.

The present research also suggests that differences in cleft type may affect the length of speech segment durations. If children with alveolar clefts do have a tendency to lengthen sound segments, then they may be more likely to be judged as severe on perceptual characteristics such as audible nasal air emission. In addition, it has often been reported that children with alveolar clefts are more susceptible to misarticulations than children with cleft palate only (Morley, 1970; McWilliams and Musgrave, 1977; Fletcher, 1978). If future research confirms that prolonged sound segments influence either perceptual characteristics

or speech intelligibility, then this may suggest the need for earlier and more aggressive repair of alveolar clefts.

APPENDIX

Testing Script

Thank you for agreeing to help us with our research study. The first thing that we will need to do is to make sure that you are hearing well. So, we will check your hearing in the sound booth.

(Child is screened at 25 dB HL at 1, 2, and 4 kHz. Child is instructed to raise hand when he/she hears a tone. If a hearing screening was already done as part of the clinical evaluation, then the screening is skipped.)

Next, we will simply record your voice while you say some silly sentences.

(While still in sound booth, head-mounted microphone is placed on child and adjusted to be at mouth level. Child counts to 10 while input gain on recorder is adjusted.)

Okay, let's practice the silly sentences. (Child is shown index card with sentence.) You say "Say ta again." Good. (Child is shown next sentence). Now say, "Say da again." Good.

Now, each time I show you one of the cards, simply say the sentence.

(Child is shown first card five times with an approximately 3 second pause between each production. Child is then shown the second card five times with an approximately 3 second pause between each production. The first and second cards are repeated so that the child produces each sentence a total of 10 times.)

Great! We are finished with the study. Would you like to pick a prize from the box?

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