

***Effect of Velopharyngeal Port Closure on Voice
Characteristics in Individuals with
Repaired Cleft Palate***

Project under AIISH Research Fund (ARF) 2012-13

(Ref: SH/CDN/ARF/4.50/2012-13)

With total funds of Rs. 3, 11,000.00

Department of Speech Language Pathology

All India Institute of Speech and Hearing

Manasagangothri, Mysore – 570006

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CHAPTER I

INTRODUCTION

Velopharyngeal dysfunction (VPD) refers to a condition where the velopharyngeal valve does not consistently and completely closes during the production of oral sounds. VPD results in a wide variety of resonance and speech disorders. Resonance disorders include hypernasality, hyponasality, cul-de-sac resonance and mixed resonance. Different types of velopharyngeal dysfunction have been described in literature where as specific terminologies are used by authors to suggest etiology (Trost-Cardamone, 1989).

Velopharyngeal insufficiency (VPI) refers to a structural defect that causes the velum to be too short to contact the posterior pharyngeal wall. VPD occurs due to several reasons including irregular adenoids, hypertrophic tonsils, oral cavity tumors, and cleft palate. Velopharyngeal incompetency is the term used to refer to neuro-motor or physiological disorders that result in poor movement of the velopharyngeal structures. It is often characterized by poor elevation and inadequate knee action of the velum during speech. Velopharyngeal incompetency may occur due to abnormal muscle insertion following cleft palate repair, generalized hypotonia of the entire velopharyngeal valve, dysarthria, and apraxia of speech. Velopharyngeal mislearning is another condition that refers to inadequate velopharyngeal closure due to faulty articulatory patterns.

Velopharyngeal dysfunction results in a wide variety of speech problems. Speech disorders such as nasal air emissions, nasal grimace, weak or omitted consonants, short utterance length, altered rate and segment durations and finally compensatory and obligatory articulation productions have been reported in this population. Resonance disorders are the most common in this population and include hypernasality, hyponasality, cul de sac resonance and mixed

resonance. Voice is another feature of speech that is affected in individuals with velopharyngeal dysfunction. Vocal problems such as hoarseness, strained voice, and breathiness have been reported in children with cleft palate and velopharyngeal dysfunction (Brooks and Shelton, 1963; McWilliams, Lavorato, and Bluestone, 1973; and D'Antonio, Muntz, Province, and Marsh, 1988).

Need for the study

Individuals with velopharyngeal dysfunction associated with cleft palate have been hypothesized to be at greater risk of vocal problems due to the presence of glottal articulation, laryngeal compensation and distorted anatomy in them. To verify this hypothesis, studies have been conducted investigating voice characteristics in individuals with repaired cleft palate. Many studies reported an anecdotal increase in the symptom of hoarseness in individuals with cleft palate. Higher prevalence of dysphonia at 12 to 43% have been reported in individuals with cleft palate compared to normal pediatric population (Grunwell, Henningson, Jansonius, Karling, Meijer, Ordning, Wyatt, Vermeij, and Sell, 2000; Timmons, Wyatt, and Murphy, 2001; and Hocevar-Boltezar, Jarc, and Kozelj, 2006).

Voice problems such as hoarseness, breathiness, pitch breaks, glottal attacks, restricted pitch and loudness range have been reported to be associated with velopharyngeal dysfunction associated with repaired cleft palate (D'Antonio, Muntz, Province, and Marsh, 1988). Direct laryngoscopy of individuals with cleft palate has revealed mixed results with varied incidence from 7% to 84% (Van Lierde, Claeys, De Bodt and Van Cauwenberge, 2004). However, most of these studies evaluated voice characteristics in these individuals under uniparametric domains such as acoustic assessment, perceptual assessment, aerodynamic assessment and physiological assessment using video imaging. There is a dearth of literature focusing on a multi-parametric

assessment of voice in these individuals. Thus, it warrants a more in-depth investigation to understand the physiological aspects, as well as to establish the true prevalence of hoarseness in individuals with cleft palate. Further it is also essential to understand the level of compensation by the laryngeal subsystem and its variations with respect to the extent/degree of velopharyngeal dysfunction. In light of this, the present study was thus taken up with the aim of studying the effect of velopharyngeal port closure on voice characteristics in individuals with repaired cleft palate using multiparametric measures.

Aim of the study

The present study was aimed at investigating the effect of velopharyngeal closure on voice characteristics in children with repaired cleft palate.

Objectives of the study

1. To investigate the effect of velopharyngeal dysfunction associated with repaired cleft palate on perceptual voice characteristics.
2. To investigate the effect of velopharyngeal dysfunction associated with repaired cleft palate on Dysphonia Severity Index and its constituent parameters.
3. To investigate the effect of velopharyngeal dysfunction associated with repaired cleft palate on Cepstral analysis of voice.
4. To investigate the effect of velopharyngeal dysfunction associated with repaired cleft palate on laryngeal aerodynamic parameters estimated subglottic pressure, Mean airflow rate, and laryngeal airway resistance.
5. To investigate the effect of extent/degree of VPD on DSI and its constituent parameters.

CHAPTER II

REVIEW OF LITERATURE

The velopharyngeal mechanism or velopharyngeal system refers to the action of velum along with the lateral and posterior pharyngeal walls to direct the sound transmission and air pressure to oral and/or nasal cavity at the level of the velum. Velopharyngeal port is formed by the space surrounded by the velum, the lateral pharyngeal walls, and the posterior pharyngeal wall. Normal velopharyngeal closure is achieved by the coordinated action of the velum, the lateral and the posterior pharyngeal wall. These structures function as a valve that separates the nasal and the oral cavity during production of oral speech sounds, singing, whistling, blowing, sucking, swallowing, gagging and vomiting.

During the production of oral speech sounds, the velum raises in the superior and posterior direction to come in contact with the posterior pharyngeal wall and lateral pharyngeal walls. Two important phenomenon takes place during the velar activity. Firstly, there is a “velar stretch”, which is the elongation of the velum in length during the function of speech. Then there is the “velar bend”, during which the velum exhibits a knee action where in it bends to provide maximum contact with the posterior pharyngeal wall. This is caused by the contraction of the levatorvelipalatine muscle.

During the production of the nasal sounds the velum is pulled down so that the acoustic energy can enter the nasal cavity. The lowering of velum is due to the contraction of the palatoglossus muscle and to a lesser extent, gravity and tissue elasticity (Kuehn and Azzam, 1978; Moon and Kuehn, 1996, 1997). The lateral pharyngeal walls contribute to the velopharyngeal closure by moving medially to close against the velum. There exists great

variation in the extent and symmetry of movement of the posterior pharyngeal wall among the normal speakers (Shpritz, Rakoff, Skolnick and Lavorato, 1977). The posterior pharyngeal wall may move anteriorly to form the valve, although its contribution is less (Iglesias, Kuehn and Morris, 1980). Some normal and abnormal speakers exhibit a well-defined area on the posterior pharyngeal wall that bulges on speech known as Passavant's ridge.

Factors affecting velopharyngeal port closure

Several factors influence the closure of velopharyngeal port. Variations in closure exist among normal speakers itself where patterns like coronal, circular, sagittal, circular with Passavant's ridge exist. These patterns occur due to differential contribution of the structures in forming the velopharyngeal port. Variations also occur based on whether the activity is pneumatic or not pneumatic. It is seen that velopharyngeal closure is complete for non-pneumatic activities like swallowing, but insufficient for speech or other pneumatic activities like sucking, blowing, singing (Shpritz, McCall, Skolnick and Lencione, 1975). Variations are also seen with respect to the timing of voicing onset and the velopharyngeal closure.

It was seen that rate and fatigue also affect the velopharyngeal mechanism. As the speech rate increases, the height of the closure decreases and it becomes less firm, thus making the speech more hypernasal (Moll and Shriner, 1967). For the production of oral sounds the voicing should occur before the movement of velum so that the velopharyngeal port is completely closed when the oral sound is produced. If the closure is incomplete then hypernasality may occur (Ha, Sim, Zhi, and Kuehn, 2004). Muscular fatigue also affects the efficiency of the closure. Young children's speech is described as hypernasal at the end of the day when they are tired due to velar fatigue.

Velopharyngeal closure is also altered as a result of age and growth. Maturation changes in the craniofacial skeleton causes changes in the size of the cavities of the vocal tract cavities. There is an increase in the length of the pharynx linearly with increase in the age and height (Rommel, Bellon, Hermans, Smet, De Meyer, Feenstra, Veereman- Wauters, 2003). There is also significant change in the angle of the posterior pharyngeal wall. At around 5 years of age, the angle is oblique between the posterior pharyngeal walls of the nasopharynx and the oropharynx (Kent, 1976; Kent and Vorperian, 1995) whereas the angle is 90 degrees at puberty and through adulthood. The velum also moves down slightly with the growth of the maxilla. The velum also increases in length and thickness and tends to stretch more to make up for the differences in the structural differences. Another factor that influences the velopharyngeal function in children is the size and presence of the adenoids. In some children it is the adenoids that help in closure to some extent, that the closure is veloadenoidal (Kent and Vorperian, 1995). When an alteration occurs, in the structure or function of the velopharyngeal port, it results in its dysfunction.

Velopharyngeal dysfunction

Velopharyngeal dysfunction (VPD) refers to a condition where the velopharyngeal valve does not consistently and completely closes during the production of oral sounds (Netsell, 1988; Marsh, 1991; Morris, 1992; and Witt, O'Daniel, Marsh, Grames, Muntz and Pilgram, 1997). Different types of velopharyngeal dysfunction have been described in literature where as specific terminologies are used by authors to suggest etiology (Trost- Cardamone, 1989).

Velopharyngeal insufficiency (VPI) is used to describe an anatomical or structural defect that prevents adequate velopharyngeal closure. VPI is the most common type of velopharyngeal

dysfunction because it includes a short velum, which is common in children with cleft palate after the palate repair. Velopharyngeal insufficiency refers to a structural defect that causes the velum to be too short to contact the posterior pharyngeal wall. There are several reasons for velopharyngeal insufficiency to occur including history of cleft palate. During the cleft palate repair, surgeons attempt to retain as much velar length as possible, however, 20% of them demonstrate VPI following cleft palate repair. Even the presence of a scar tissue in a repaired cleft palate may alter transpalatal acoustic transmission (Gildersleeve-Neumann and Dalston, 2001). Sometimes the velum might be of adequate length yet closure is not complete due to a deep pharynx secondary to cranial base abnormalities (Haapanen, Heliovaara and Ranta, 1991).

Some other reasons for VPI include irregular adenoids, hypertrophic tonsils and oral cavity tumors. Irregular adenoids result in an uneven surface and as the velum closes against the adenoids for speech, it does not achieve a tight seal. Hypertrophic tonsils affect velopharyngeal closure as they restrict the medial movement of the lateral pharyngeal walls. Oral cavity tumors and thus resection of the areas of the oral cavity also affect the function of velopharyngeal valve.

Velopharyngeal incompetency is the term used to refer to neuro-motor or physiological disorders that result in poor movement of the velopharyngeal structures. It is often characterized by poor elevation and inadequate knee action of the velum during speech. Velopharyngeal incompetency may occur due to abnormal muscle insertion following a cleft palate repair, generalized hypertonicity of the entire velopharyngeal valve, dysarthria in which there is abnormality of the strength, range, accuracy and tonicity of the speech musculature due to central or peripheral nervous system impairment. Apraxia may also cause velopharyngeal incompetency in which there exists a difficulty in coordinating and sequencing velopharyngeal movement during speech. The timing of closure may be affected as the initiation of phonation occur too late

(Warren, Dalston, Trier and Holder, 1985). Literature reports of velopharyngeal incompetency to occur in case of cranial nerve defect as well as velar fatigue.

Velopharyngeal mislearning is another condition that refers to inadequate velopharyngeal closure due to faulty articulatory patterns. A common misarticulation is the substitution of a pharyngeal fricative or posterior nasal fricative for sibilant sounds. Nasal air emissions associated with this condition is referred to as phoneme specific nasal emission (PSNAE) as they occur only on certain speech sounds. This is the result of faulty articulation rather than an anatomical defect or physiological defect. Also, since the individual has learnt and habituated faulty speech patterns prior to surgical correction, they remain the same post-surgery unless altered by speech therapy. Lack of auditory feedback as in case of hearing loss may also affect the production due to the inability to monitor resonance. In individuals with hearing impairment the velopharyngeal valve may close and open inappropriately causing hypernasality (Ysunza and Vazquez, 1993).

Influence of velopharyngeal dysfunction on speech production

Velopharyngeal dysfunction results in a wide variety of speech problems. Resonance disorders are the most common and include hypernasality, hyponasality, cul de sac resonance and mixed resonance. Hypernasality is usually caused by velopharyngeal insufficiency, but can also be caused by velopharyngeal incompetence. Hypernasality is most evident with large velopharyngeal openings and not perceived with smaller openings. Hyponasality is very common in individuals with cleft lip and palate and it may be even more prevalent than hypernasality in adults and adolescents in this population. This is because the VPI is usually corrected in the preschool or early school years so that hypernasality is no longer present as the individual gets

older. However as the surgical procedures aim to narrow or reduce the size of the velopharyngeal space, hyponasality is often a complication of such surgery (Witt, Myckatyn, and Marsh, 1998). Other causes of hyponasality in individuals with cleft palate include deviated septum, choanal stenosis or atresia or a maxillary retrusion which restricts the pharyngeal and nasal cavity space (Riski, 1995). Cul-de-sac resonance is common in children with history of cleft palate which can be due to a combination of VPI and blockage of nasal cavity as with a deviated septum.

Velopharyngeal dysfunction can also cause wide variety of other speech related dysfunctions such as nasal air emission, nasal grimace, weak or omitted consonants, short utterance length, altered rate and segment durations and finally compensatory and obligatory articulation productions. Nasal air emissions occur when there is a build-up of intra oral air pressure for the production of consonants in the presence of a velopharyngeal valve leak. Some of the airflow is released through the nose causing a disruption in the aerodynamic process of speech. It is mostly noted on pressure sensitive phonemes (fricatives, plosives and affricatives). The loudness of the nasal air emission relates to the size of the velopharyngeal valve. When the opening is large, there is little resistance to the flow and thus minimal friction is produced making the emission not very audible. When the opening is small, vice versa happens, the resistance causes the airflow to become turbulent and thus emission is audible. This type of nasal emission is referred to as nasal turbulence. However, the sound that is heard is primarily due to the bubbling of secretions and some use the term nasal rustle (Kummer, Curtis, Wiggs, Lee and Strife, 1992). Nasal grimace occur as a muscle reaction around the nares to achieve velopharyngeal closure. When air flows through an oro-nasal fistula or velopharyngeal valve, it reduces the amount of air pressure that is available in the oral cavity that is required for the production of consonants. This causes the consonants to be weak in intensity and pressure, and

causes them to be completely omitted (Baken, 1987 and McWilliam, Morris, and Shelton, 1990). It is seen that individuals with large velopharyngeal openings tend to increase the intraoral pressure by increasing the airflow rate during consonant production. Hence use respiratory volumes twice that of normal individuals (Warren, Wood and Bradley, 1969). When there is significant air emission, it reduces the amount of air pressure that is available for connected speech, thus requiring more frequent breaths.

Speech characteristics like nasal air emission, weak consonants are the result of direct velopharyngeal opening. Hence they are referred to as passive or obligatory articulation while articulation errors that are the individual's response to the velopharyngeal dysfunction are referred to as active or compensatory errors. These compensatory errors often occur due to the individual efforts to compensate for the inadequate intraoral pressure. During compensatory articulation the manner is maintained but the place is altered and moved most posteriorly to the pharynx or larynx. This allows the individual to make use of the air available in the pharynx or at the larynx before it is reduced due to the velopharyngeal opening. The obligatory errors are purely due to abnormal structures and hence require surgical intervention, while compensatory errors are the ones in which a modification can be brought by speech therapy and is under patient's control. Some of the commonly seen compensatory and obligatory errors are mid-dorsum palatal stop, generalized backing, velar fricative, and nasalization of oral consonants, nasalization of vowels, nasal snort, nasal sniff, pharyngeal plosive, pharyngeal fricative, pharyngeal affricate, posterior nasal fricative, glottal stops.

Influence of velopharyngeal dysfunction on the laryngeal system

Voice is another feature of speech that is affected in individuals with velopharyngeal dysfunction. Vocal problems such as hoarseness, strained voice, and breathiness have been reported in children with cleft palate and velopharyngeal dysfunction (Brooks and Shelton, 1963; McWilliams, Lavorato, and Bluestone, 1973; and D'Antonio, Muntz, Province, and Marsh, 1988). Many researchers have found an anecdotal increase in the symptom of hoarseness in individuals with cleft palate compared with the normal population. Previous researchers have found the rate of dysphonia in the cleft palate population is 12 to 43% which is higher than the rates in normal pediatric population (Grunwell, Henningson, Jansonius, Karling, Meijer, Ording, Wyat, Vermeij, Sell, 2000; Timmons, Wyatt, Murphy, 2001 and Hocevar-Boltezar, Jarc, Kozelj, 2006). Direct laryngoscopy of individuals with cleft palate has revealed mixed results with varied incidence from 7% to 84% (Van Lierde, Claeys, De Bodt & Van Cauwenberge, 2004).

Robison and Otteson (2011) aimed to document the incidence of voice disorders in children with cleft palate. They used questionnaires that consisted of questions directly related to hoarseness, VPI, and also used direct laryngoscopy and fiberoptic flexible laryngoscopy. The results of their study found the incidence of hoarseness in cleft palate population to be 5.5% which is similar to the incidence in normal pediatric population. The authors reasoned their finding as either there is no difference between normal pediatric population and cleft palate children or that the incidence of voice problems in children with cleft palate is underreported.

Researchers have provided many different hypotheses to explain the increase in hoarseness among the cleft palate population. However, the most common explanation was the laryngeal system trying to compensate for abnormal velopharyngeal valving (Lewis, Andreassen,

Leeper, Macrae and Thomas, 1993 and Van Lierde et al., 2004). A common finding in individuals with mildly impaired velopharyngeal valving is a hyper-functional voice disorder. This is due to increased respiratory and muscular effort, including hyper-adduction of the vocal folds, with attempts to close the velopharyngeal port. Initially, this behavior may cause thickening and edema of the vocal folds, ultimately leading to the formation of vocal nodules (Boone and McFarlane, 1988). Individuals with cleft palate are hypothesized to expend greater adductory force on their laryngeal structures in order to reduce nasality and reach a certain vocal intensity. In order to achieve a better constriction for the inadequately functioning velopharyngeal port, individuals with cleft palate exhibit a likely deliberate use of the vocal folds. This often causes more voice problems in individuals with clefts compared to non-clefts. When there is VPI, the lack of adequate oral acoustic energy in combination with the absorption of sound energy by the pharyngeal tissue results in a damping effect, causing the voice to be low in intensity (Bernthal and Beukelman, 1977). Finally low intensity and breathiness may occur as a compensatory strategy to mask the hypernasality and nasal air emission. However a recent study contradicts the long held theory of laryngeal compensation as the source of hoarseness (Hamming, Finkelstein and Sidman, 2009).

Individuals with cleft palate also often have associated velopharyngeal insufficiency which results in poor articulation and increased incidence of hypernasality. The poor articulation is often compensated by the use of pharyngeal and glottal sounds. Glottal stops in particular have been indicated in literature to cause hoarseness (D'Antonio, 1988 and Van Lierde et al., 2004). The glottal stops are produced by quickly and forcibly adducting the vocal folds causing damage to them. Vocal nodules may also develop secondary to the use of glottal stops as a compensatory articulation strategy.

The other hypothesis that has been explained is the type of palatoplasty used to correct the cleft and a possible role of the subject's gender (Musgrave, McWilliams, Matthews, 1975 and Van Lierde et al., 2004). Several organic as well as functional causes of voice problems have been suggested in this population including laryngeal anomalies, particularly in those individuals with congenital malformation syndromes. Thus, there seems to be no one definite cause of hoarseness, suggesting that it is mostly a multifactorial cause.

Individuals with cleft palate are possibly at greater risk of vocal problems due to the presence of glottal articulation, VPI, laryngeal compensation and distorted anatomy. Thus, it warrants a more in-depth investigation to understand the physiological aspects, as well as to establish the true prevalence of hoarseness in individuals with cleft palate.

Voice characteristics in individuals with Velopharyngeal dysfunction

Literature reports of dysphonia as a common speech dysfunction in individuals with VPD. The perceptual features of dysphonia including hoarseness, breathiness, glottal fry, hard glottal attack, inappropriate pitch level, restricted pitch range, diplophonia, and inappropriate loudness have been reported in this population (Kummer and Marsh, 1998). These abnormalities may vary from a mild to severe degree (Stemple, Glaze and Gerdeman, 1995).

Hamlet (1973) hypothesized that glottal tightness might contribute to vocal abuse which would lead to hoarseness, harshness, and vocal nodules as a consequence to hypernasality. McWilliams et al. (1973) reported of inappropriate phonatory habits, chronic hoarseness, periods of aphonia, vocal hyperfunction, soft voice syndrome with monotone voice, limited voice pitch range, strangled voice with excessive abdominal and laryngeal tension. Bernthal and Beukelman,

(1977) reported that, maintenance or increase in speech intensity in the presence of nasal coupling requires increased laryngeal effort, resulting in vocal abuse and nodule formation.

Leder and Lerman (1985) and D'Antonio et al (1988) reported inappropriate adduction of the vocal folds, acoustic evidence of abnormal laryngeal activity, roughness incomplete glottal closure, severe hyperconstriction, harsh, excessive high or low habitual pitch, strained, breathy, reduced or excessive loudness in individuals with velopharyngeal dysfunction.

In a recent study by Van Lierde et al., (2004), 28 children with mean age of 9.9 years and with a diagnosis of cleft of palate were assessed on a multiple parameters including perceptual, acoustic and aerodynamic. The auditory perceptual assessment was done using the GRBAS scale given by the Japan Society of Logopedics and Phoniatics (1981). Statistical analysis revealed significant difference between male children with cleft palate and control group on perceptual grade of hoarseness and roughness. The GRBAS results showed rating as G₁ R₁ B₀ A₀ S₀, indicating that there was slight hoarseness and roughness in voice of these children. In female children with cleft palate, GRBAS score was G₀ R₀ B₀ A₀ S₀, indicating normal voice on the perceptual scale. This study also revealed that 32% (9/20) of the cleft palate in this population had laryngeal pathology. Both bilateral vocal nodules (11%) and muscle tension dysphonia (Type I) (11%) were the most common ones. The results of the study revealed that the presence of dysphonia was more in male children with cleft palate.

Van Lierde et al. (2004) made an inventory of functional voice disorders in individuals with cleft palate patients as reported in the literature (Table 1).

Table 1

Inventory of functional voice disorders in cleft palate as reported in the literature.

<i>Authors</i>	<i>Functional voice disorders</i>
Seth and Guthrie (1935)	Habitual use of the vocal folds as an articulator
Cobb and Lierle (1936)	Lack of pitch variation and vocal intensity
Ritchie (1937)	Flat, monotone, intonation pattern
McDonald & Baker (1951)	Altering laryngeal valving, breathiness, hoarseness
Westlake (1953)	Improper phonatory habits, vocal pitch differences
Westlake & Eisenson (1953)	Spread of tension to the pharyngeal and Laryngeal Muscle during articulation, Harshness, higher fundamental frequencies in males.
Berry & Eisenson (1956)	Spread of tension to the pharyngeal and laryngeal muscles during glottal articulation, harshness.
Dickson (1962)	Higher fundamental frequencies in males reported
Brooks et al. (1963)	Breathiness, Hoarseness
Bzoch (1964,1979)	Soft voice syndrome, aspirated Phonation
Flint (1964)	Lower fundamental frequencies in females
Rampp and Counihan (1970)	Reported of improper phonatory habits
McWilliams et al. (1969)	Chronic hoarseness, periods of aphonia, vocal hyperfunction, soft voice syndrome with monotone voice, limited voice pitch range, strangled voice with excessive abdominal and laryngeal tension .
McWilliams et al. (1973)	
Tarlow and Saxman (1970)	Normal fundamental frequency
Bernthal and Beukelman (1977)	Changes in vocal intensity
Leder and Lerman (1985)	Inappropriate adduction of the vocal folds, acoustic evidence of abnormal laryngeal activity, roughness incomplete glottal closure, severe hyperconstriction, harsh, excessive high or low habitual pitch, strained, breathy, reduced or excessive loudness
D'Antonio et al. (1988)	

Acoustic analysis of voice in individuals with VPD associated with repaired cleft palate

The documented voice problems in cleft palate population are mainly subjective. The objective methods are few and mainly included acoustic and spectrographic studies. Leder and Lerman (1985) investigated the spectrograms of speakers with cleft palate and results revealed inappropriate vocal fold adduction during stop consonant production. Zajac and Linville (1989); Lewis, Andreassen, Leeper, Macrae, and Thomas, (1993), and Van Lierde et al., (2004) reported higher frequency perturbation measures in such children.

Zajac and Linville (1989) in their study aimed at studying the relation between the voice perturbations and perceived nasality in children with VPI. The participants included 10 children diagnosed with VPI and 5 normal children. Kay Elemetrics electroglottograph was used in the perturbation analysis. Subjects were instructed to phonate vowels/a/, /i/ and/u/ at their comfortable loudness. Mean jitter % was reported to be 1.16 ± 0.36 . Mann Whitney U test suggested that the jitter% was significantly different between the two groups. The voice perturbations of children with hoarseness correlated moderately with perceived hoarseness. The authors discussed this finding of their study as an evidence for the link between the laryngeal and velopharyngeal events.

Van Lierde et al. (2004) aimed at the evaluation of voice in children with cleft palate on a multiparametric domain. It included perceptual, acoustic and aerodynamic evaluation. The acoustic evaluation aimed at the evaluation of perturbations measures like jitter, shimmer. Multidimensional voice profile from Computerized Speech Lab module was used in the analysis of jitter. A steady portion of the vowel sustained was considered for the analysis. Results of the study suggest that there existed a statistical difference between the male children with cleft palate children and the normative data, while the female children in the cleft palate group did not show significant difference when compared to the normative data. The authors opined that increased jitter% is reflected perceptually as increase in hoarseness.

A laryngeal pathology is often associated with perceptual features like roughness and breathiness. The lesions of the vocal folds often cause inappropriate adduction and abduction of the vocal folds. When the glottis closure is inappropriate it results in breathiness. The breathiness causes the airflow through the glottis to be turbulent. This turbulent airflow results in an output which consists of high frequency energy and it is this high frequency energy that is perceived as breathiness. The resultant output is less periodic and thus observed as the increased cycle to

cycle variation in the fundamental frequency and amplitude. The roughness component results from lack of periodicity in the vocal fold vibrations. The aperiodicity is caused by changes in the flexibility of the mucosal layer of the vocal folds when there is a pathological lesion of the vocal folds. These changes are reflected in the perturbation measures. But a major disadvantage of these perturbation measures is that they rely on the fundamental frequency in their calculation and any small changes in calculation of fundamental frequency leads to erroneous results in the perturbation measures. This is especially relevant in case of aperiodic voice where the calculation of fundamental frequency is difficult.

Cepstral analysis overcomes the limitation of pitch extraction errors associated with time domain measurements. To obtain a cepstrum, first Fast Fourier Transformation (FFT) of a spectrum is done. The spectrum thus obtained is subjected to another FFT and thus a cepstrum is obtained. Thus a signal which is time domain (waveform) is converted to frequency domain (spectrum). Then the frequency domain signal (spectrum) is converted to a que frequency domain (cepstrum). A cepstrum gives a display of the harmonic structure. To obtain a cepstral peak prominence (CPP), a regression line is drawn through the cepstrum. CPP is the difference in magnitude of the highest peak of cepstrum to the value of the cepstrum that is obtained through a regression line. It gives an indication of how much the peak of cepstrum is above the average value obtained from the regression line. CPP is a reflection of the degree of harmonic organization. When individual cepstrum are averaged over a number of frames then smoothed cepstral peak prominence (sCPP) is obtained. Both CPP and sCPP have shown to be good predictors of dysphonia (Hillenbrand, Cleveland and Erickson, 1994; Hillenbrand, 1996; Olson, Goding and Michael, 1998). In case of breathiness the overall cepstrum is relatively flat thus the

peak i.e. CPP is of smaller amplitude. In case of normal voice with well-defined harmonic structure, the CPP is well defined with higher peak amplitude.

Garnier, Gallelo, Collet and Berger Vachon (1996) aimed at studying the spectral characteristics that helps to best differentiate voice of individuals with velopharyngeal impairment from that of normals. They considered 21 individuals with velopharyngeal impairment as the clinical group for the study and 42 individuals matched for gender and age as the control group. The speech material considered for the study included vowels /a/, /i/ and /u/ in isolation, vowel /a/ sustained and vowels in the context of two oral and two nasal sentences. The results of the study indicated that among the different spectral measures, cepstral coefficients and linear FFTs were the most efficient tools in discriminating the two groups compared to the other methods like bark FFTs and formants. The authors attributed the findings to the changes in the spectrum which is brought out by the additive noise. The authors gave future directions in terms of using LTAS and phonation intensity range to be used to study the changes in fundamental frequency brought about by laryngeal pathology.

Thus, uniparametric focus has been used to investigate the voice characteristics in individuals with cleft palate by means of perceptual, acoustic and physiological measures. However, the scope of uniparametric approaches is limited in providing the clinician a holistic nature of voice characteristics in these individuals. This issue can be resolved by using multiparametric approaches. Dysphonia severity index (DSI) is a multiparametric approach used for assessing voice quality objectively (Wuyts, De Bodt, Molenberghs, Remacle, Heylen, Millet, Van Lierde, Raes, Van de Heyning, 2000). Perceptual assessment, scored on GRADE from GRBAS was used to classify severity of dysphonia. The parameters used in DSI are Highest fundamental frequency (F_0 high in Hertz), Lowest intensity ('I-low' in decibel sound pressure

level), Maximum phonation time (MPT in seconds), Jitter% (percentage short term variability in fundamental frequency). The DSI is constructed as $DSI = 0.13 \times MPT + 0.0053 \times F_0\text{-High} - 0.26 \times I\text{-low} - 1.18 \times \text{Jitter} (\%) + 12.4$. It is constructed in such a way that a perceptually normal voice (Grade 0) corresponds with a DSI of + 5; a severely dysphonic voice (Grade 3) corresponds with a DSI of - 5. Also scores beyond this range are possible (higher than + 5 or lower than - 5). DSI is designed to establish a quantitative and objective correlate of the perceived voice quality. The more negative the individual's index the worse is his vocal quality.

The choice of variables in the DSI is entirely determined by the stepwise logistic regression procedure. The highest frequency was chosen as it is seen that in more than 50% of the dysphonic individual, the vocal folds are afflicted with excess mass as in case of vocal nodules, edema. This extra mass usually unevenly distributed along the cords hamper the higher vibratory rate. This is reflected by decreased F_0 high. Also the presence of nodules, edema etc. increases the glottal resistance, thus requiring a greater driving force to initiate and maintain vocal fold vibration. Consequently the lowest intensity will be increased in several participants with dysphonia. Similar effects of F_0 high and I_0 low are found in VRP studies of children with vocal nodules (Heylen et al., 1998). Perturbation measures such as jitter are intended to assess the degree of irregularity of the vocal fold vibration within certain limits. It is likely that a perceived dysphonia will result in an increased perturbation measure. MPT was chosen as it is a phonatory ability measure (Hirano, 1981) that reflects the efficiency of several mechanisms necessary for voice production such as subglottic pressure, airflow resistance etc.

Wuyts et al., (2000) correlated the DSI with GRBAS scale and found a correlation as high as 0.996 as DSI is based on the GRBAS scale rather than on the discrimination between normal and pathological voice. The effect of sex is implicitly included in the DSI, so that a

separate DSI for males and females need not be used. The opposite behavior of the F_0 high and MPT for both the cases cancels out, so that DSI for both males and females are identical.

The content and criterion validity of DSI has been justified. The content validity of DSI (i.e. it measures what it is intended to measure, in this case degree of dysphonia) is justified as the multiple parameters used to calculate DSI are clear indicators of dysphonic voice and they are markedly deviant compared to normal (Wuyts, De Bodt, Molenberghs, Bruckers, and BSGVD, 1996). Secondly DSI has been constructed based on a statistical stepwise procedure that constructs a rule to classify voice. The criterion validity (i.e. accuracy) of DSI is also justified as it is well correlated with the gold standard (i.e. auditory perceptual judgments) on the GRBAS scale. DSI also correlates with VHI suggesting that it not only reflects the vocal quality but also the individual's handicap to some extent. The parameters used in DSI are accessible in most voice clinic. The fact that DSI is based on aerodynamic, acoustic and voice range measurements makes it a robust objective measurement.

Van Lierde et al., (2004) used DSI to assess voice quality characteristics in individuals with repaired cleft palate. The voice quality in 28 children (18 males and 10 females) with a cleft palate and mild hypernasality were studied. As gender affects voice differently, the gender differences were given separately. The results of the study indicated that male children with cleft palate had a mean DSI of +0.62. The DSI was obtained from its constituent parameters of a mean maximum phonation duration of 14 seconds, mean highest fundamental frequency of 617 Hz, mean lowest intensity of 61 dB, and mean jitter of 1.2%. The presence of slight roughness on the perceptual scale could be correlated to the higher jitter % values (1.2%). Female children with cleft palate had mean DSI value of +2.4 which was obtained from its constituent parameters maximum phonation duration of 13.5 seconds, mean highest fundamental frequency of 725 Hz, a

mean lowest intensity of 58 dB, and a mean jitter of 1.17%, reflecting normal voice on a perceptual scale. The perceptually normal voice characteristics corresponded with a DSI-value of +2.4 and a normal jitter of 0.61%. The results of the study support the hypothesis that vocal quality disorders characterized by a more negative DSI-value and perceptual voice symptoms occur in male cleft palate children.

Aerodynamic analysis of voice in individuals with VPD associated with repaired cleft palate

Aerodynamic processes are responsible for all the acoustic aspects of speech production. During the inspiratory phase of respiration, air enters the lungs causing an increase in the volume of the thoracic cavity and lowering the pressure inside the lungs. During the expiratory phase, the thoracic muscles relax causing an increase in the pressure in the lungs, which when released causes the movement of the vocal folds, required for the production of all voiced speech sounds. The articulators of the vocal tract further modify this airflow for the speech production. During the production of the stop consonants, the lips briefly obstruct the airflow resulting in build-up and release of pressure. The acoustic bursts resulting from the release of the lips provide important acoustic cues.

Aerodynamic procedures provide information on (a) the timing aspects of the VP function on certain phonetic contexts and (b) the patency of the nasal airways during breathing. Aerodynamic measures used in evaluating the velopharyngeal mechanism includes measures like subglottic pressure, mean airflow rate, intra-oral pressure levels, rate of nasal air emissions etc. These intraoral pressure levels vary depending on the type of consonant and phonetic context. Voiceless sounds are known to have greater intraoral pressure than voiced consonants due to open glottis.

During breathing and swallowing, the respiratory and phonatory subsystems function autonomously. The two subsystems however act in unison during voice and speech production. The respiratory and laryngeal subsystems must coordinate to maintain a constant subglottic pressure, a constant airflow and a relatively constant upper airway construction for production of steady state utterances. A combined measure that captures relations among respiratory and laryngeal function would reflect the coordinated nature of the voice productions. Laryngeal airway resistance is one such measure that represents both the laryngeal and respiratory functions. Laryngeal airway resistance is subglottal pressure (cmH₂O) divided by average airflow (L/s). Thus laryngeal airway resistance is one such measure which gives a more genuine representation of the co-ordination between respiratory and phonatory subsystems. Although it does not give a direct indication of specific muscular or mucosal activity, it reflects relations across the sum of passive and active respiratory and phonatory forces and aerodynamic factors in voice production.

Adequate production of speech requires an effective velopharyngeal mechanism. During the production of oral consonants, the velopharyngeal mechanism must separate the oral and nasal cavities. Also during the production of nasal consonants, the VP mechanism must allow some degree of oral-nasal coupling. Sussman (1992) reported perceptual features like weak consonants, nasal emission in individuals with cleft palate. These perceptual features have clear aerodynamic foundations. There has been a dearth of literature related to laryngeal aerodynamics in cleft palate population. One reason for this has been attributed to the invasiveness of the techniques used and the assumptions of the various measurement techniques. Direct measurement of subglottal pressure is invasive and requires the use of the passage of catheters through the glottis. Indirect measurement involves substitution of intra oral air pressure in a

particular phonetic environment such as the syllable /pi/ (Smitheron and Hixon, 1981). This method assumes airtight closure of the velopharyngeal which does not occur in individuals with repaired cleft palate. To overcome this limitation the authors suggested that the nostrils of persons with velopharyngeal inadequacy be occluded/ plugged (D'Antonio et al., 1988).

The complex nature of air flow profile depends not only on pressure which acts as the driving force, the size of the constriction, and temporal patterns of the movement of the articulators, but also on the mechanical properties of oro-pharyngeal tissue, the size and shape of oro-pharyngeal cavities, and biomechanics of release or formation of articulatory constrictions.

Disturbance of the laryngeal system in general is showed by excessive air flow rates which occur due to the incomplete adduction of the vocal folds or by highly inconstant air flow rates during the sustained phonation due to the imprecise control of the larynx. This is clinically shown as an abnormally restricted range of vocal frequencies and intensities, leading to limited abilities in speech production in the affected individual. Glottal impedance and hence glottal integrity is estimated by the mean air flow rates for sustained phonation of vowels.

D' Antonio et al., (1988) studied the prevalence of voice disorders in 85 participants with velopharyngeal dysfunction. A multi-method protocol was used in the evaluation of velopharyngeal dysfunction. The authors report of no clear relationship between laryngeal findings and the nasoendoscopic and aerodynamic findings. However, the subglottic pressure was significant and the trend showed a higher than normal value in individual with laryngeal abnormalities when compared to those without laryngeal abnormalities. They also reported of difference in the way the individuals with velopharyngeal function compensated for the abnormalities in the laryngeal function. 43% of the velopharyngeal dysfunction individuals with

vocal thickenings exhibited increased respiratory effort to compensate for their vocal pathologies. At the same time 31% of them had reduced respiratory effort, suggesting that this subgroup of users with velopharyngeal inadequacy may have tried to cover up for their defects by using reduced respiratory effort

Morr, Warren, Dalston, and Smith (1989) investigated the role of differential pressure measurements (difference in the oral and nasal pressure during the production of voiceless plosive consonant productions) as a screening index. The pressure flow technique (Warren 1964, 1979) was used to estimate the velopharyngeal size and to measure the orifice differential pressure in 515 individuals with cleft palate, velar inadequacy or both. The differential pressure measurements were fair good predictors of velopharyngeal function. The authors suggested that differential pressure measurement is only a screening tool and in case inadequacy is observed a more comprehensive and detailed evaluation is necessary. Warren (1979) also reported that a differential pressure greater than 3cm of H₂O predicts adequate closure, while a pressure less than 3 cm of H₂O indicates inadequate closure.

Lewis et al. (1993) studied the laryngeal aerodynamic parameters in children with cleft palate and control group. The normal group had a velopharyngeal port size of 1.1mm² while children with cleft palate had a velopharyngeal port size of 1.17mm². Cleft palate children had higher transglottal airflows, subglottal pressures, laryngeal airway resistances when compared to control group. However these differences were not significant.

Leeper, Macrae, and McKnight (1994) compared laryngeal aerodynamic measures of children with cleft palate to the control group. Within the cleft palate children group itself, there existed two subgroups based on the velopharyngeal orifice size. They authors reported lower

laryngeal airway resistances, subglottal pressures, and higher trans-glottal airflows in individuals with velopharyngeal orifice size was greater than 5mm^2 than when compared to individuals with velopharyngeal orifice size less than 5-mm^2 and the control group.

Zajac (1995) aimed to evaluate the laryngeal aerodynamics in children with cleft palate and control group. Children with cleft were divided into two groups based on the nasal airflow. When the nasal airflow was greater than 10cc/sec , they were categorized into the incomplete closure group. When the nasal airflow was less than 10cc/sec they were grouped no complete closure group. Results of the study indicate there was no significant difference for laryngeal airway resistance between the cleft and non- cleft group (both the complete and incomplete). The authors opined that the velopharyngeal orifice size in the incomplete closure group is minimal probably not requiring compensation at the laryngeal level.

Thomas, Guyette, Anita, Sanchez, Bonnie, and Smith (2000) investigated the effect of velopharyngeal dysfunction on laryngeal aerodynamic parameters. The participants of the study included 36 children with cleft palate. They were further divided into two subgroups. The first subgroup consisted on the incomplete closure group who had a velopharyngeal size greater than 5mm^2 . The second subgroup consisted of complete closure group who had a velopharyngeal size less than 1mm^2 . The two subgroups were compared on laryngeal aerodynamic parameters of laryngeal airway resistance, transglottal airflow and transglottal pressure. One subgroup showed high trans-glottal pressures and resistances while the other showed low trans-glottal pressures and resistances. The authors explained this difference in vocal strategy as learnt and perseverated and also may be due to the difference in the size of the velopharyngeal orifice within the adequate range.

In summary, the studies have been conducted to verify the voice characteristics of individuals with cleft palate and how different subsystems of speech production compensate for the velopharyngeal dysfunction in them. However these voice characteristics have often been studied on single domain. In light of understanding the voice characteristics from a multi-parametric perspective, the present study was thus taken up with the aim of studying the effect of velopharyngeal port on voice characteristics in individuals with cleft palate.

CHAPTER III

METHOD

Participants

Thirty participants in the age range of four to fourteen years diagnosed with repaired cleft palate and velopharyngeal dysfunction were considered for the study. Thirty participants included 17 males and 13 females. The diagnosis of velopharyngeal dysfunction was made by a team of plastic surgeon, speech language pathologist and otolaryngologist using visual inspection, perceptual evaluation, nasoendoscopy, nasometry, and Glatzel mirror test. All the participants attended speech therapy for less than 10 sessions; where each session was of 45 minutes duration. Speech therapy sessions aimed to correct resonance disorders, eliminate compensatory articulation and achieve correct phonetic placement. None of the participants had complaint of nasal regurgitation. All the participants except two were undergoing schooling with either English or Kannada as their medium of instruction. 40 normal children were matched for age and gender formed the control group. They included 16 females and 24 males. They were free of any known cranio-facial anomalies and were judged by a SLP as having normal articulation, resonance and phonation.

The participants fulfilling the following criteria were included in the study

1. The participants diagnosed as repaired cleft of the palate.
2. The participants with unilateral or bilateral cleft palate and had undergone primary palatal surgery.
3. Participants with no history of pharyngeal surgery.
4. Participants with no cognitive deficiency.

5. Participants with no neuro-motor dysfunction and hearing threshold above 20 dB in the worst ear.

Procedure

A detailed case history of the participants were obtained including the type of cleft, age of palatoplasty, type of palatoplasty, associated speech problems such as hypernasality, misarticulations, nasal emissions. Information was also collected regarding non speech problems like difficulty in swallowing and nasal regurgitation. The participants and/or the caregivers were explained about their participation and a written consent was obtained for all the assessment procedures. To confirm the presence of velopharyngeal dysfunction, participants were subjected to clinical examination by the team of Plastic surgeon and Speech language pathologist. The participants were further subjected to instrumental evaluation of nasalance using nasometry and to evaluation of nasal air emissions using Glatzel mirror test.

Nasometry: Nasometer II (6450) by Kay Elemetrics was used to analyze the mean nasalance scores. Nasometer consists of a head set, which has a separator that was rested on the participant's upper lip and secured with the help of a head band with velcro strips. The separator has microphones on either side which picks up the nasal and oral energy separately. The signal from each microphone is processed individually and digitized. Participants were instructed to phonate vowels /a/ for as long as possible. They were instructed to phonate the vowel at a pitch and loudness level they were comfortable. They were also made to repeat oral sentences, nasal sentences and oro-nasal sentences. The samples recorded were analyzed by making a selection with the cursor marking at beginning and ending of the sample. This was followed by applying the Nasometer statistical function to obtain mean nasalance values.

Glatzel Mirror: A 20 x 13cm Glatzel mirror was used to check for nasal air emissions. The Glatzel mirror is made up of a stainless steel plate that has semi-circular markings indicating the severity of the nasal air emissions. The examiner ensured that the Glatzel mirror was free of any dirt and also free without any fogging prior to the testing. The participant was made to prolong the vowel /a/, /i/ and sibilant /s/, while the Glatzel mirror was placed below the nares and the philtrum of the upper lip. The examiner checked for the fogging of the mirror. Based on the area of fogging the scoring was given. Nasal air emission was given a 3 point rating of 1, 2 and 3 where a score of ‘1’ was given when fogging occurred on the most proximal end of the mirror, while a score of ‘3’ was given when the fogging occurred on the most distal end indicating severe nasal air emissions.

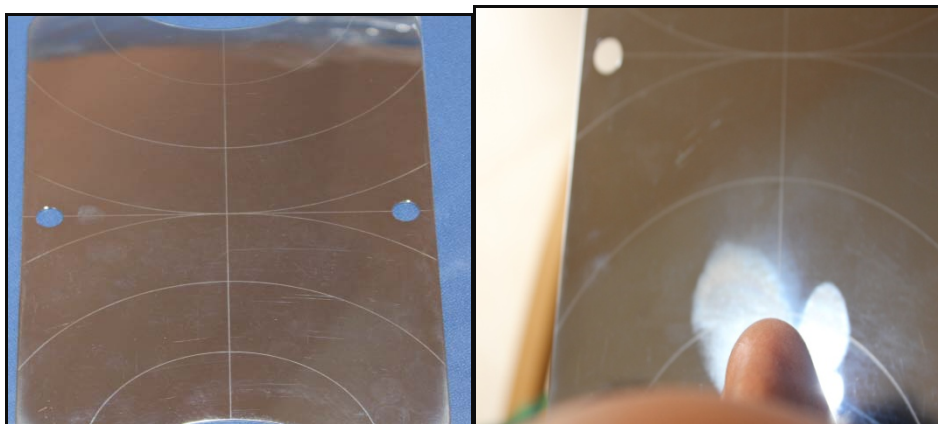


Figure 1. *Glatzel mirror; fogging associated with nasal air emission on Glatzel mirror.*

Following the evaluation, the individuals who were diagnosed to have velopharyngeal dysfunction associated with repaired cleft palate by Plastic surgeon and Speech language pathologist through clinical and instrumental evaluations were included in the study. The included participants were subjected to multidimensional assessment of voice three different domains including perceptual, acoustic, and laryngeal aerodynamic analysis.

Perceptual Analysis of Voice

A clinician rated measurement was carried out using the GRBAS scale. The GRBAS scale (1981) was developed by the Japan Society of Logopedics and Phoniatics. It is a four point rating scale used to rate the vocal quality. A 4 point rating scale of 0-normal, 1- slight, 2-moderate, 3-severe was used to rate 5 parameters which include G- the overall grade of hoarseness, R- rough, B- Breathly, A-Asthenic, S- Strain.

The participants were made to sit in a sound treated room and various speech tasks were elicited from them. The speech tasks included sustaining vowel /a/ and counting numbers from 1 to 10 and 60-70. Conversational speech sample was also elicited either using a picture description or dialogue exchange between the participant and the examiner. The elicited responses were audio recorded at a sampling rate of 44 KHz on the CSL software and stored as a wave file. The audio samples which were recorded were later played to the judges at a loudness they comfortable listening to. The judges were instructed to rate the participant's speech sample for each of the five parameters on the GRBAS scale. The judges were asked to rate both the tasks of vowel phonation and spontaneous speech. A rating sheet with subject details, GRBAS parameters, and scoring was given to the judges for perceptual rating (Appendix I). The judges were three Speech-Language Pathologists with more than 3 years of expertise in the area of assessment and diagnosis of voice disorders. To eliminate any bias about the severity of any of the parameters, the judges were blinded of the participant's diagnosis.

Acoustic analysis of Voice

Acoustic analysis of voice was done by using dysphonia severity index (DSI) and by using cepstral analysis. DSI is a weighted measure used to objectively quantify the voice quality.

The parameters used in DSI are Highest fundamental frequency (F_0 high in Hertz), Lowest intensity ('I-low' in decibel sound pressure level), Maximum phonation time (MPT in seconds), Jitter% (percentage short term variability in fundamental frequency). The DSI is constructed as: $DSI = 0.13 \times MPT + 0.0053 \times F_0\text{-High} - 0.26 \times I\text{-low} - 1.18 \times \text{Jitter} (\%) + 12.4$. The instrumentation used for computing DSI was Computerized Speech Lab (CSL) 4500 by Kay PENTAX was used for acoustic analysis of voice. Two modules within the Computerized Speech Lab (CSL) namely Advanced Multidimensional Voice Profile (MDVP) and the Voice Range Profile were used to obtain the raw parameters required to calculate the DSI.

Maximum Phonation Duration (MPD): MPD was measured using the CSL module (*Kay Pentax Corp, model 4500*). To obtain maximum phonation duration (MPD) the participants were seated comfortably on a chair and were asked to sustain phonation of vowel /a/ at their habitual pitch and loudness. They were instructed to inhale deeply and produce vowel /a/ on exhalation and sustain it continuously as long as he/she could. The procedure was modeled by the experimenter and the participant was visually encouraged and coached. Three test trials were obtained and the best trial with the longest duration was considered for further analysis. MPD was calculated by placing the cursor at the beginning and end of the waveform. The CSL software displays duration of the selected segment precisely to milliseconds, thus giving the value of maximum phonation duration.

Highest frequency (HF_0) and lowest Intensity (LI_0): Voice Range Profile module from the CSL (*Kay Pentax, Model 4500*) was used in the measurement of highest frequency and the lowest intensity. A unidirectional microphone was used for recording. The microphone to mouth distance was maintained at 30cm. It was ensured that the VRP mode was turned 'on' in the CSL hardware.

To obtain the highest frequency, two maneuvers were used. The first maneuver involved changing the pitch of vowel /a/ in a single glide from the lowest pitch to the highest pitch possible. The second maneuver involved changing the pitch of vowel /a/ in succession of three steps from lowest pitch to modal register and then the highest pitch. Both the maneuvers are demonstrated by the experimenter to the participant. Caution was taken to ensure that the participant does not enter the falsetto register. Once the plot of the pitch change was obtained on the VRP graph, the value of the highest frequency was obtained by placing the cursor on the most consistent plot (represented as the darkest shade) towards the left on the x axis.

Voice Range Profile module from the *CSL (Kay Pentax, Model 4500)* was used in the measurement of lowest intensity. An adapter was attached to the microphone to nullify the pre-amplification effects of the microphone. It was ensured that the VRP mode was turned ‘on’ in the CSL hardware. The minimum energy level in the VRP settings was adjusted to 35dB to ensure that the lowest intensity is visible on the VRP graph plot. The participant was instructed to phonate vowel /a/ at the softest loudness possible. They were asked to begin with phonating /ha/ and then slowly progress towards phonating vowel /a/ at the softest loudness possible. The value of lowest Intensity (LI_0) was obtained by placing the cursor on the most consistent plot (represented as the darkest shade) towards the bottom on y axis.

Jitter%: The acoustic parameter of Jitter % was obtained by the Advanced *Multi-Dimensional Voice Program (MDVP)* from *CSL (Kay Pentax Corp, model 4500)*. The participant was made to phonate vowel /a/ at their comfortable pitch and loudness. A 4 second middle segment consists of steady sample from the waveform was selected for analysis of jitter%. After analysis, using the MDVP statistical function, the value of jitter% was obtained.

The raw scores of the four parameters MPT, Hf_0 , LI_0 and jitter% were used to obtain the value of DSI by using the regression equation: $DSI = 0.13 \times \text{Maximum Phonation duration (MPD)} + 0.0053 \times F_0\text{High} - 0.26 \times \text{I-Low} - 1.18 \times \text{Jitter (\%)} + 12.4$. The regression equation was entered into an excel sheet and values of raw parameters were entered to obtain the DSI value.

Cepstral analysis: Cepstral analysis involved measurement of two parameters namely cepstral peak prominence (CPP) and smoothed cepstral peak prominence (sCPP). The participants were instructed to phonate the vowel /a/ at their comfortable pitch and loudness. A mic to microphone distance was maintained at 15cm. The sample was recorded at a sampling rate of 44 kHz using CSL. A 4 second steady portion of the sample was considered for the analysis and stored in the .wav format. The sample was analyzed by the software ‘speech tool’ developed by Hillenbrand. The software has options for computing CPP and sCPP. The recorded file stored in the .wav file is loaded on the software by opening the file from the file menu. Then and on applying the ‘CPPV’ (cpp for vowels) function, it displays the mean values of CPP and sCPP. It also provides information regarding the mean F_0 , size of averaging window which is 10 bins and the averaging window size which is 150 frames.

Laryngeal aerodynamic analysis

Laryngeal Aerodynamic analysis involved measurement of parameters estimated Sub-Glottic Pressure (ESGP) and Mean Air-Flow Rate (MAFR). The instrument used was the Aeroview 1.4.4 (Glottal Enterprises, USA). An optimized algorithm on this computer based system also provides information on derived parameters such as Laryngeal Airway Resistance (LAR) and Laryngeal Airway Conductance (LAC). Other measures of sound quality such as Sound Pressure Level (SPL), fundamental frequency of the measured vowel segment can also be obtained.

The aeroview instrumentation consists of a circumferentially vented (CV) Pneumotachograph mask, a wide band model air pressure transducer PT 25, an airflow transducer PT 2E. Both these transducers were coupled to the Pneumotachograph mask. The electronic unit MS-110 provides power to the transducers, pre amplifier and the analog to digital convertor. A BFC-2 adapter enables connecting the two transducers by means of a MS-110 input jack to the MS-110 front panel. Any USB jack on the computer can be connected to the MS-110 and it can be powered either using a USB connection or separately through a direct power connection.

Calibration of the instrument: Both the pressure and airflow transducers were calibrated before each recording or after every time the computer was booted, as per the instructions provided by Glottal Enterprises in the aeroview manual. FC-1 calibrator was used in calibration of airflow at 0.140 liters per sec, while PC-1 calibrator aided in the calibration of air pressure at 10 cm H₂O.

Recording: Comfortable seating of the participants was ensured and the testing procedure was explained to them. The circumferentially vented (CV) pneumotachograph mask was firmly held against the face so that there is no leakage around the nose and mouth. The intraoral tube was placed such that it was held between the upper and lower lip and without the tongue occluding the tube. Appropriate placement of the transducer and proper seal while using the mask was ensured by the examiner. The participants were asked to repeat CV syllable /pa/ 6-7 times into the circumvented mask. They were instructed to produce the syllable string at a pitch and loudness comfortable to them. It was ensured that the repetitions were adequate to obtain six to seven peaks of intraoral pressure with good wave morphology. The examiner also modeled the production for better understanding of the participant. In order to minimize errors while recording and to ensure adequate syllable rate is maintained two test trials were run before the

final recording. Final recordings were made after the practice trials. The recommended syllable rate was between 2.0 to 3.5 per second. Peaks with flat and consistent wave morphology were considered for further analysis. Figure 3 shows the typical morphology of pressure peaks.

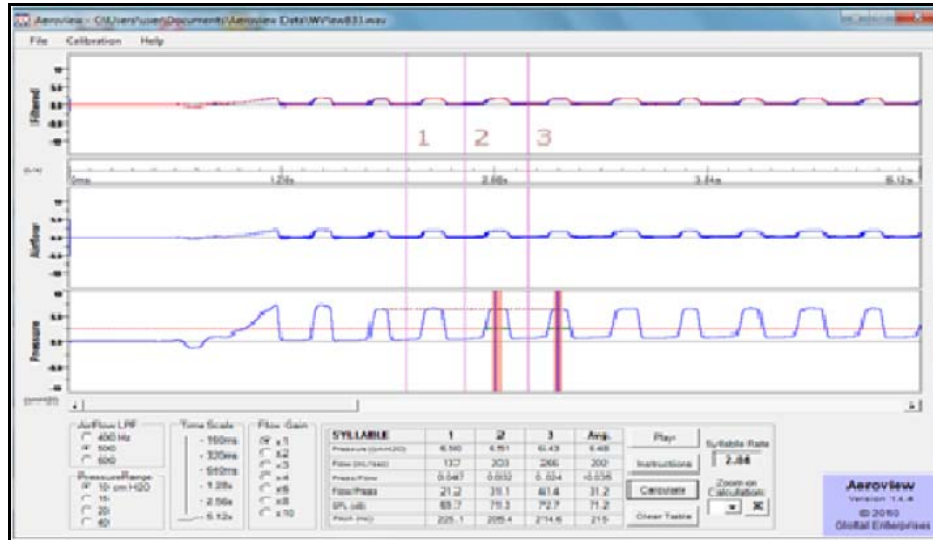


Figure 2. Typical peak morphology to be considered for analysis.

Analysis: Two adjacent pressure peaks with appropriate wave morphology were selected. The flat portions of adjacent pressure peaks were analyzed by using cursor selection. The application software analyzes the selected portions of the waveform and values of ESGP (cmH₂O), MAFR (ml/sec), LAR (cmH₂O/ml/sec), values are provided. Three peak to peak measurements were selected and further when the ‘calculate’ function in the software was applied, it gives an average value of the parameters. The instrument displays the unit of MAFR is in ml/sec, which was converted to Liters/sec so as to compare with the prior studies. The unit of LAR was also changed to (cmH₂O/L/sec).

Statistical Analysis

The raw data that included DSI and its constituent parameters, CPP, sCPP and laryngeal aerodynamic measures were tabulated and entered in the statistical software. The two groups considered in the study included individuals with cleft palate which formed the clinical group and normal children matched for age formed the control group. The SPSS software (version 21) was used for the statistical analysis of the data. 2-way MANOVA was performed to find the main effects of group, gender and group - gender interaction on DSI. Group and gender were the independent variables while DSI and its constituent parameters were the dependent variables. To compare the laryngeal aerodynamic measures 1-way MANOVA was performed with group as the independent variable and ESGP, MAFR and LAR as the dependent variables. As the sample size was small for comparison of laryngeal aerodynamic measures across gender, Mann Whitney U test was applied where gender was the independent variable and LA measures ESGP, MAFR and LAR were the dependent variables. To check for the effect of gender and group on CPP 2-way MANOVA was used where gender and group were the independent variables and CPP and sCPP were the dependent variables. To find if there was a gender effect within each group on CPP a non parametric Mann- Whitney U test was performed.

Dendogram was used to identify the clusters in the participants of clinical group based on their nasalance values. Dendogram is a tree diagram frequently used to represent clusters and is obtained by hierarchical clustering. Hierarchical clustering groups the data over a variety of scales by creating a cluster diagram or dendogram. This tree is not a single set of clusters but rather a multilevel hierarchy where clusters at one level are joined to clusters at other level. This allows deciding the level or scale of clustering that is most appropriate for the application. The clinical group was divided into two subgroups based on the clusters formed by using the Dendogram.

Following this, one way MANOVA was performed to verify if there was a significant difference between the two clusters in clinical group across their voice quality as measured on DSI. The two subgroups formed the independent variables while DSI and its constituent parameters formed the dependent variables.

CHAPTER IV

RESULTS

The present study compared voice characteristics of children with repaired cleft palate and velopharyngeal dysfunction, which formed the clinical group and typically developing, age and gender matched children, which formed the control group. The participants in the clinical group were subjected to clinical examination by the team of Plastic surgeon and Speech language pathologist and to instrumental evaluation of nasalance using nasometry and to evaluation of nasal air emissions using Glatzel mirror test to confirm the presence of velopharyngeal dysfunction. The male participants in the clinical had an average nasalance value of 38.79 (± 13.1), 38.61 (± 20.60), 52.81 (± 10.34), and 50.04 (± 7.61) for vowels, oral sentences, nasal sentences, and oral+nasal sentences respectively. The female participants in the clinical had an average nasalance value of 46.08 (± 13.1), 39.53 (± 12.95), 57.74 (± 10.58), and 56.33 (± 8.56) for vowels, oral sentences, nasal sentences, and oral+nasal sentences respectively. The mean nasal airflow emission of children in clinical group was observed using glattzel mirror and revealed a mean score of 2 in twenty two, 1 in six, and 0 in two participants. The average nasalance value in the clinical group was higher compared to normative nasalance scores described in the literature (Navya and Pushpavathi, 2013) indicating the presence of velopharyngeal dysfunction.

Following the clinical examination, and instrumental evaluation using nasalance, and nasal air emission measures, the participants with confirmed diagnosis of VPD were included in to the control group. Detailed voice evaluation was performed on both the clinical and control groups using multiparametric analysis under perceptual, acoustic and aerodynamic domains. The data thus obtained was tabulated and statistically analyzed using appropriate statistical tools

including tests of central tendency, dispersion, tests of significance and Dendogram. The results obtained are presented in the following sections.

- I. Comparison of control and clinical groups based on perceptual voice measures.
- II. Comparison of control and clinical groups based on Dysphonia Severity Index (DSI).
- III. Comparison of control and clinical groups based on cepstral analysis.
- IV. Comparison of control and clinical groups based on laryngeal aerodynamic measures.
- V. Comparison of DSI scores between the clinical sub-groups.

I. Comparison of control and clinical groups based on perceptual voice measures

The scores of GRBAS scale was averaged among the three judges. Perceptual analysis revealed that females in the clinical group had a mean GRBAS score of $G_0 R_0 B_0 A_0 S_0$ indicating that the voice quality was perceptually normal while males in the clinical group had a mean GRBAS score of $G_1 R_1 B_1 A_1 S_0$ voice indicating that the vocal quality was slightly breathy and rough.

II. Comparison of control and clinical groups based on Dysphonia Severity Index

Mean and standard deviation of the parameters were used to calculate the value of DSI, such as maximum phonation time (MPT), Jitter %, highest frequency (HF_0) and lowest intensity (LI_0). They were computed and presented in table 2. Two way MANOVA was performed to verify the effect of group and gender on the DSI and its constituent parameters. The results of two way MANOVA indicated significant effect of group and gender on DSI values. However, no interaction of group and gender was observed for DSI as well as its constituent parameters. Interaction effect of group and gender for DSI can be seen in Table 5.

Table 2

Mean and standard deviations of the acoustic measures in clinical group and control group.

Sl. No	Parameter	Clinical group				Control group			
		Males		Females		Males		Females	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	MPT	8.31	5.25	5.97	1.87	11.53	3.62	9.60	3.42
2	Jitter%	1.55	1.08	1.03	0.77	1.40	0.83	0.57	0.53
3	Hf ₀	339	52	398	54	401	65	410	95
4	LI ₀	43.0	1.69	43.30	1.75	43.92	2.73	43.41	2.73
5	DSI	2.24	1.69	2.81	1.10	2.93	1.23	3.83	1.10

II. A. Effect of group on DSI and its constituent parameters

On comparison of the voice parameters across the groups, it can be noted that the MPT values are higher in the control group (10.75 seconds) than the clinical group, which has a mean of 7.30 seconds. It showed significant difference on 2-way MANOVA [$F(5, 29) = 13.77, p < 0.05$]. The frequency perturbation measure, jitter % was higher in the clinical group (1.32), while the control group had a mean jitter % of 1.07; however, the difference was not statistically significant [$F(5, 29) = 2.20, p > 0.05$]. The highest frequency was also higher in the control group (404 Hz), when compared to clinical group which had a mean value of 365Hz, and showed a significant difference on 2-way MANOVA [$F(5, 29) = 4.66, p < 0.05$]. The lowest intensity did not vary across the groups (control and clinical) with a mean value of 43 LI₀ [$F(5, 29) = 0.37, p > 0.05$]. The DSI values were higher in the control group with a mean value of 3.29, while the clinical group had a mean DSI value of 2.49 which showed a significant difference on 2-way MANOVA [$F(5, 29) = 6.78, p < 0.05$]. Figure 4 shows raw parameters of Dysphonia Severity Index in clinical and control group. Figure 5 shows Dysphonia Severity index (DSI) in clinical and control group.

Table 3

Test of significance across group for DSI.

Sl.no	Parameter	N	df	F value	p value
1.	MPT	30	1	13.77	0.00*
2.	HFo	30	1	4.66	0.03*
3.	Llo	30	1	0.79	0.37
4.	Jitter%	30	1	2.20	0.14
5.	DSI	30	1	6.78	0.01*

*level of significance at $p \leq 0.05$; df- degrees of freedom

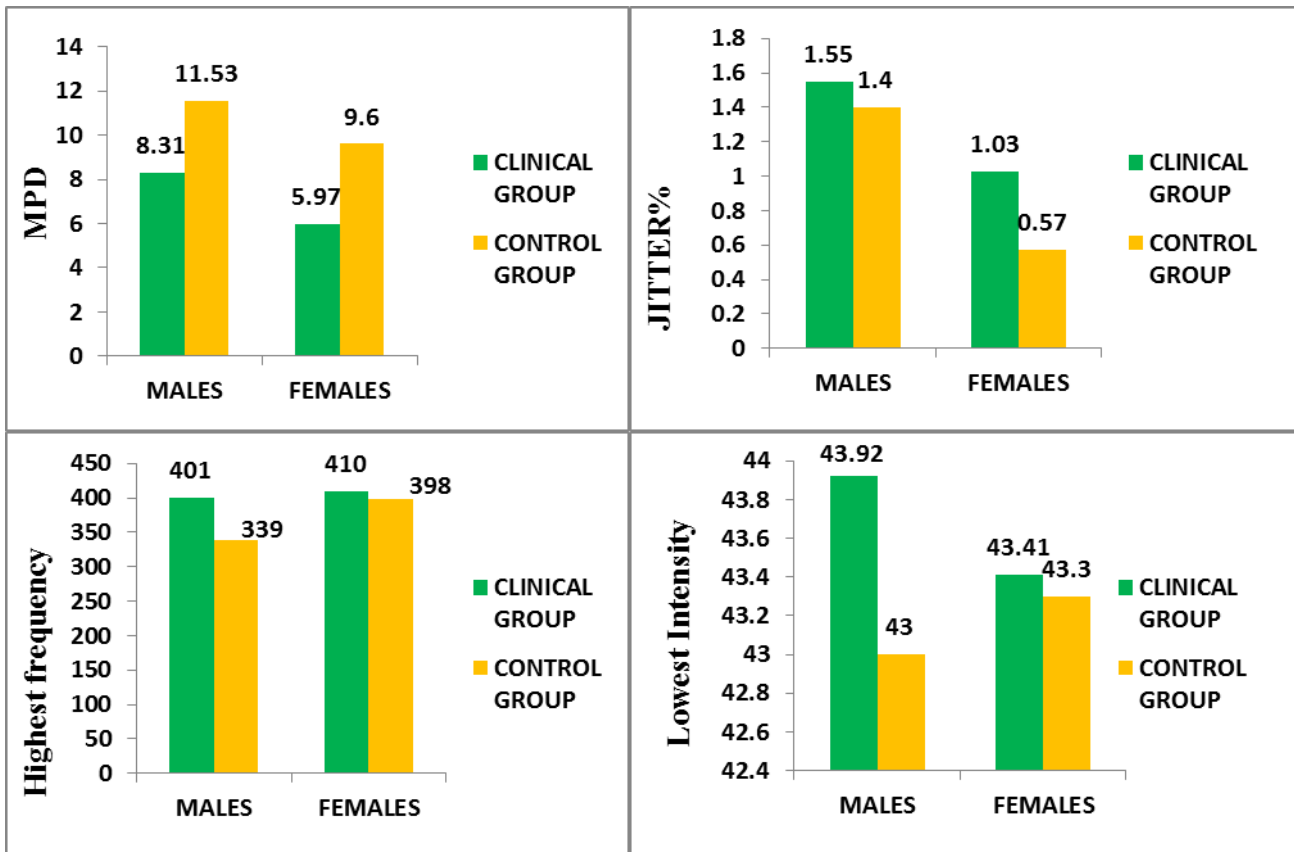


Figure 3. Effect of group on constituent parameters of Dysphonia Severity Index.

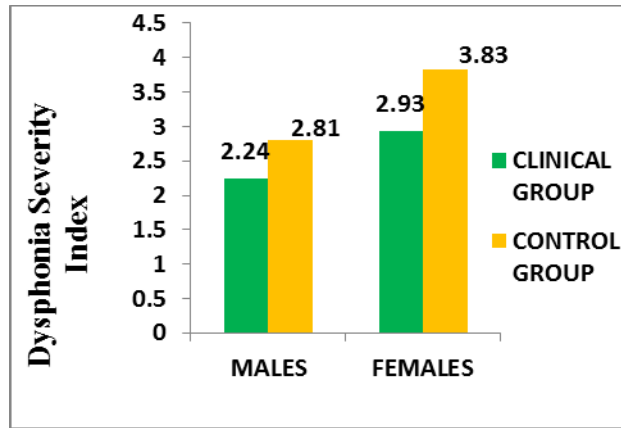


Figure 4. Effect of group on the Dysphonia Severity Index.

II. B. Effect of gender on Dysphonia Severity Index and its constituent parameters

Statistical analysis revealed that the maximum phonation time was higher in males compared to females in both the groups. The mean values of MPT in clinical group and control group among males was 8.31 ± 5.25 and 11.53 ± 3.62 seconds respectively, while females had a mean of 5.97 ± 1.87 and 9.60 ± 3.42 seconds, which was statistically significant [$F(5, 41) = 5.35$, $p < 0.05$]. Jitter % was also higher in males in both the groups with mean value of 1.55 ± 1.08 and 1.40 ± 0.83 in clinical group and control group respectively, which showed a significant difference on 2-way MANOVA [$F(5, 41) = 11.28$, $p < 0.05$]. The ‘F’ and ‘p’ values for DSI and its constituent parameters are presented in table 4. The interaction effect of group and gender on DSI and its constituent parameters is given in table 4.

Table 4

Test of significance across gender for DSI.

Sl.no	Parameter	N	df	F value	p value
1.	MPT	30	1	5.35	0.02*
2.	HF ₀	30	1	4.04	0.04*
3.	LI ₀	30	1	0.03	0.86
4.	fitter%	30	1	11.28	0.00*
5.	DSI	30	1	5.06	0.02*

*level of significance at $p \leq 0.05$; df- degrees of freedom

Table 5

Interaction effect of group and gender for DSI.

Sl. No	Parameter	N	df	F value	p value
1.	MPT	30	1	0.04	0.82
2.	HF ₀	30	1	2.25	0.13
3.	LI ₀	30	1	0.50	0.48
4.	Jitter%	30	1	0.57	0.45
5.	DSI	30	1	0.27	0.60

*level of significance at $p \leq 0.05$; df- degrees of freedom

HF₀ was higher in the females in both clinical and control group with mean values of 398 Hz \pm 54 and 410 Hz \pm 95 respectively and showed a statistically significant difference [F (5, 41) = 4.04, $p < 0.05$]. The lowest intensity did not however vary much across gender with mean value of 43dB and showed no significant difference across 2-way MANOVA LI₀ [F (5, 41) = 5.06, $p < 0.05$]. The DSI value was higher in females in both the groups. Females had a mean value of 2.81 \pm 1.10 and 3.83 \pm 1.10 while males had a mean value of 2.24 \pm 1.69 and 2.93 \pm 1.23 in clinical and control group respectively. Figure 6 shows effect of gender on Dysphonia Severity Index (DSI). Figure 7 shows effect of gender on constituent parameters of DSI.

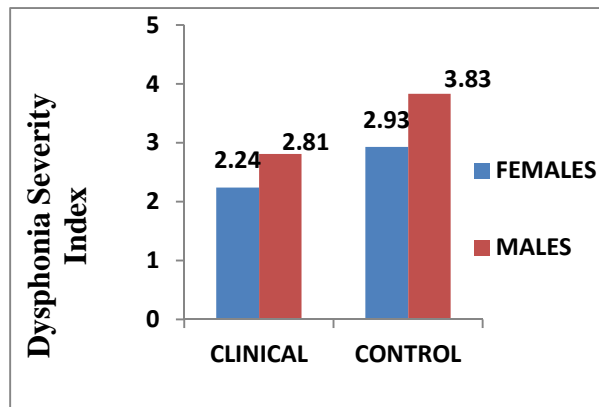


Figure 5. *Effect of gender on the Dysphonia Severity Index (DSI).*

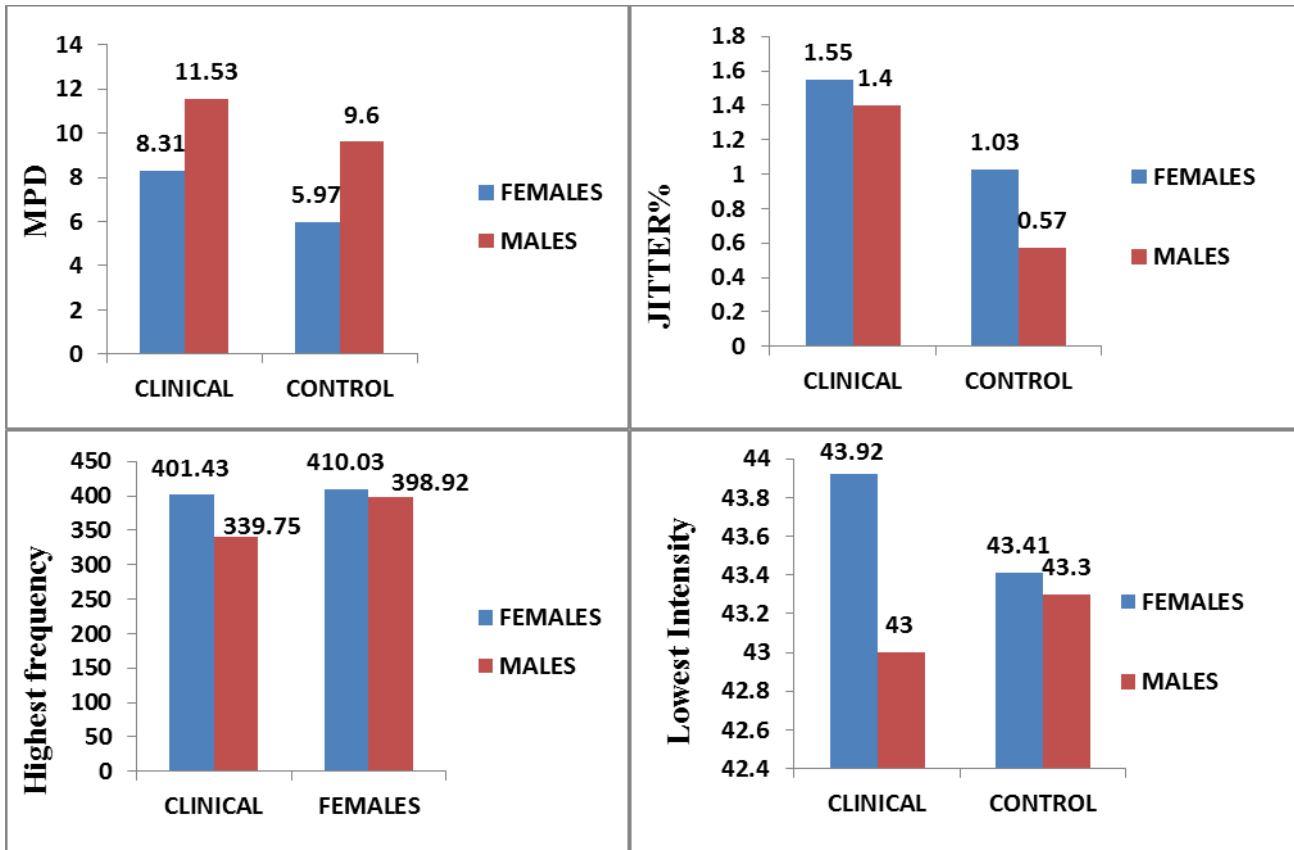


Figure 6. Effect of gender on the constituent parameters of Dysphonia Severity Index (DSI).

III. Comparison of clinical group and control group based on cepstral analysis

Mean and SD of Cepstral peak prominence (CPP) and smoothed cepstral peak prominence (sCPP) were computed across group and gender and tabulated as seen in table 6. The CPP values did not show significant difference between the clinical and the control group with a mean value of 18 and 17.58 respectively CPP [F (2, 2) = 0.04, $p > 0.05$]. The sCPP were higher in the control group compared to the clinical group; however the difference was not significant with mean values of 6.05 and 6.59 in the clinical and control group respectively CPP [F (2, 2) = 2.25, $p > 0.05$].

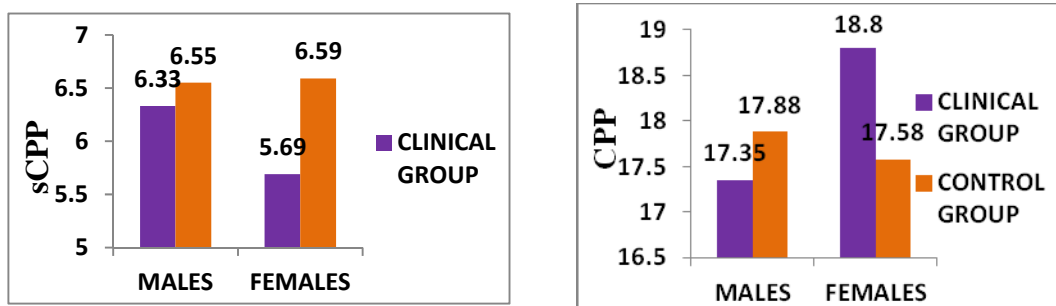


Figure 7. Effect of group on smoothed CPP and CPP.

Table 6

Mean and standard deviation of cepstral analysis in clinical group and control group.

Sl. No.	Parameter	Clinical group		Control group	
		Males mean (\pm SD)	Females mean (\pm SD)	Males mean (\pm SD)	Females mean (\pm SD)
1	CPP	17.35 (\pm 2.16)	18.8 2.81	17.88 2.81	17.58 1.09
2	sCPP	6.33 (\pm 3.03)	5.69 3.32	6.55 1.09	6.59 1.68

Table 7

Test of significance across group, gender and group-gender interaction for cepstral analysis.

Effect	Parameter	N	df	F value	p value
Group	CPP	16	1	0.04	0.65
	sCPP	16	1	2.25	0.52
Gender	CPP	16	1	0.35	0.55
	sCPP	16	1	1.73	0.19
Group*Gender	CPP	16	1	1.36	0.24
	sCPP	16	1	0.68	0.41

*level of significance at $p \leq 0.05$; df- degrees of freedom

In clinical group, there were 9 males and 7 females, therefore Mann Whitney U test (non-parametric) was performed to verify if there was a gender effect. Results of Mann Whitney U test revealed that there was no significant effect of gender across CPP ($Z = -1.21$, $p = 0.22$) and sCPP ($Z = -0.37$, $p = 0.71$) as shown in Table 8.

Table 8

Mann-Whitney U test with gender as the grouping variable for cepstral analysis.

Sl.no	Parameter	Z value	p value
1.	CPP	-1.21	0.22
2.	sCPP	-0.37	0.71

level of significance at $p \leq 0.05$

IV. Comparison of clinical and control groups based on laryngeal aerodynamic measures

Mean and SD of laryngeal aerodynamic measures such as subglottic pressure, mean airflow rate and laryngeal airway resistance were computed across group and gender and tabulated as shown in table 9.

Table 9

Mean and standard deviation of laryngeal aerodynamic measures in clinical group and control group.

Sl.no	Parameter	Clinical group				Control group			
		Males		Females		Males		Females	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	ESGP	10.89	3.86	11.77	3.59	6.17	1.07	6.08	1.42
2	MAFR	0.34	0.23	0.22	0.14	0.19	0.72	0.30	0.17
3	LAR	60.39	56.85	71.86	47.9	37.66	23.2	27.81	9.59

Statistical analysis revealed that estimated subglottic pressure was higher in clinical group with a mean value of 10.89cm/H₂O whereas the control group had a mean value of 6.12 cm/H₂O which was statistically significant [$F(3, 8) = 25.89, p < 0.05$]. Mean airflow was higher in clinical group with mean value of 0.53 L/s while control group had a mean value of 0.49 L/s; however, this difference was not statistically significant [$F(3, 8) = 0.81, p > 0.05$]. Laryngeal airway resistance was higher in clinical group with mean values of 60.39 and 71.86 in males and females respectively, while the control group showed lower mean values of 37.66 and 27.81 in males and females respectively. Results of one way MANOVA showed that this difference in

laryngeal airway resistance between the two groups was not significant [$F(3, 8) = 1.52, p > 0.05$].

Table 10 shows 'F' and 'p' values for laryngeal aerodynamic measures.

Table 10

Test of significance across group for laryngeal aerodynamic measures.

Sl.no	Parameter	N	df	F value	p value
1.	ESGP	9	1	25.89	0.00
2.	MAFR	9	1	0.81	0.37
3.	LAR	9	1	1.52	0.22

Level of significance at $p \leq 0.05$

V. Comparison of DSI scores between the clinical sub-groups

Dendrogram was used to divide the clinical group into two clusters based on the nasalance scores. The clusters thus obtained had 18 and 11 participants in each of the subgroup. Figure 9 depicts dendrogram showing the clusters among the clinical group.

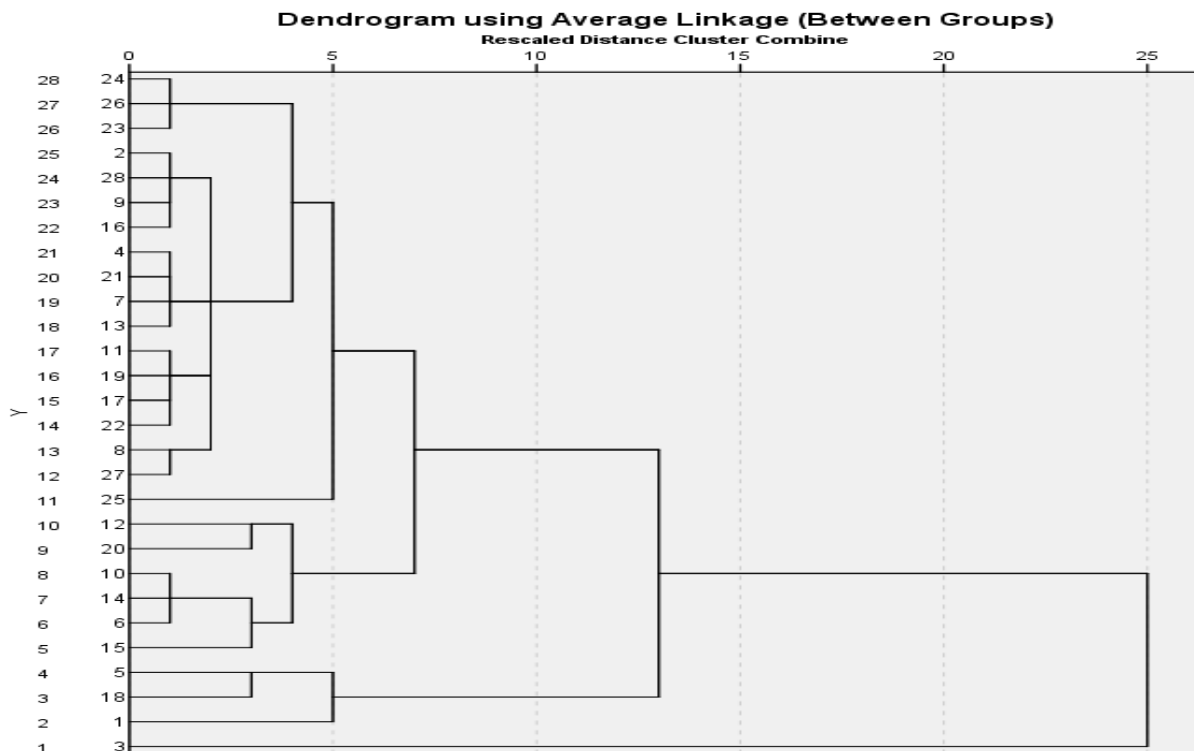


Figure 8. Dendrogram showing the clusters in clinical group.

One way MANOVA was used to compare the DSI scores and the constituent parameters between the two clusters in the clinical group were compared. Results of one way MANOVA revealed that there was no significant effect of extent of velopharyngeal dysfunction on their voice quality as measured on DSI. Table 11 shows mean values of DSI and its constituent parameters across the two clusters in the clinical group. Table 12 shows 'F' and *p* values across the clinical subgroups for DSI.

Table 11

Mean values of DSI and its constituent parameters across the two clusters in the clinical group.

Sl.no	Parameter	Cluster I	Cluster II
1.	MPT	6.65	9.0
2.	HF ₀	364	368
3.	LI ₀	43.05	43.60
4.	Jitter%	1.42	0.99
5.	DSI	2.28	1.46

Table 12

Test of significance across the clinical subgroups for DSI.

Sl.no	Parameter	N	F value	df	<i>p</i> value
1.	MPT	30	1.81	1	0.19
2.	HF ₀	30	0.03	1	0.85
3.	LI ₀	30	0.61	1	0.44
4.	Jitter%	30	1.34	1	0.25
5.	DSI	30	1.57	1	0.22

Level of significance at $p \leq 0.05$

CHAPTER V

DISCUSSION

The study aimed at assessing the effect of the velopharyngeal port closure on voice characteristics in individuals with cleft palate. Children with cleft palate formed the clinical group while typically developing children with no voice problems matched for age and gender formed the control group. The two groups were assessed on multiple parameters of voice including perceptual evaluation using GRBAS, acoustic evaluation using Dysphonia Severity Index (DSI) and cepstral analysis (CPP and sCPP) and laryngeal aerodynamic analysis. Perceptual evaluation was done using GRBAS scale. Acoustic evaluation involved measurement of DSI from its constituent parameters of MPD, Hf_0 , jitter% and Li_0 . Cepstral analysis involved measurement of CPP and sCPP. Laryngeal aerodynamic evaluation involved measurement of estimated subglottic pressure, mean airflow rate and laryngeal airway resistance. The measurements thus obtained from both the groups were organized, tabulated and statistically analyzed using appropriate statistical tools. The findings of the study are discussed in the following section.

I. Comparison of control and clinical groups based on perceptual voice measures.

Perceptual assessment done using GRBAS scale revealed a GRBAS score as follows in males in clinical group $G_1 R_1 B_1 A_1 S_1$. This suggested the voice in male children with cleft palate to be deviant perceptually. Females in the clinical group revealed a GRBAS scale as $G_0 R_0 B_0 A_0 S_0$ which indicates a voice that was perceptually normal.

Similar findings were also reported in the study by Van Lierde et al., (2004) who in their study reported a GRBAS score of $G_1 R_1 B_0 A_0 S_0$ in males with cleft palate indicating a slight

grade of roughness and hoarseness and female participants in their study had a GRBAS score of G₀ R₀ B₀ A₀ S₀ indicating perceptually normal voice. The findings of the present study are also in consensus with earlier studies by Brooks et al., (1963), McWilliams et al., (1973), Leder and Lerman (1985), and D'Antonio et al., (1988) who described the voice quality of cleft palate as rough and hoarse. Timmons, Wyatt and Murphy (2001) and Hocevar-Boltezar, Jarcand, and Kozelj (2006) also reported the incidence of perceptual feature of hoarseness in cleft palate to vary from 12% to 43% which is much higher than the incidence in typically developing individuals.

However there have been studies which reported contradictory findings to that of the present study. Hamming, Finkelstein and Sidman (2009) reported that there existed no relation between hoarseness and cleft palate and VPI. Robison and Otteson (2011) reported the incidence of hoarseness in cleft palate population to be 5.5% which is similar to the incidence in normal pediatric population. The authors reasoned their finding as either there is no difference between typically developing children and children with cleft palate in terms of their voice characteristics or that the incidence of voice problems in children with cleft palate is underreported.

II. Comparison of control and clinical groups based on DSI and its constituent parameters

a. Effect of group on DSI and its constituent parameters

Dysphonia severity index is a weighted measure used to measure the quality of voice (Wuyts et al., 2000). It constitutes of parameters like Hf₀, Li₀, jitter% and MPT. It is seen that lesser the value of DSI, lesser the deviancy in voice. The present study revealed lower DSI value in clinical group when compared to the control group. Lower DSI values are primarily due to its affected constituent parameters of Hf₀, Li₀, jitter% and MPT. This indicates that vocal capacity

and vocal stability are compromised in individuals with VPD. Lower DSI value in clinical group may be attributed to the direct vocal fold pathology or an indirect response to a velopharyngeal disorder. Van Lierde et al., (2004) explained lower DSI value as the cleft palate child's attempt to compensate for poor velopharyngeal mechanism resulting in poor voice quality and vocal hyperfunction. The lowered DSI value, suggestive of deviant voice in children with cleft palate may be due habitual use of vocal folds as the articulator, development of muscle tension of the laryngeal and pharyngeal muscles during glottal articulation (Berry and Eisenson, 1956) or the presence of vocal nodules and incomplete glottis closure in children with cleft palate (Leder and Lerman, 1985 and D'Antonio, Muntz, Province and Marsh, 1988).

Maximum phonation duration refers to the ability of the individual to sustain a phonation for the longest time possible in a single breath. It reflects the capacities of both the laryngeal and respiratory system. It reflects the capacity of the laryngeal system in maintaining adequate glottis closure. It also reflects the capacity of the respiratory system in providing adequate breath support for voice production (Prater and Swift, 1984).

The present study revealed that the MPT was shorter in the clinical group when compared to the control group. The maximum phonation time are shorter in clinical group compared to the control group as a laryngeal system which is subnormal or disordered does not use the available air supply effectively, thus producing a shorter than expected MPD (Tait, Michel and Carpenter, 1980).

Perturbation measures reflect cycle to cycle variation either in frequency or amplitude. Frequency perturbation measures cycle to cycle variation in the frequency of voice. Perturbation

measures indicate aperiodicity of voice. Higher values of perturbation measures may be caused by laryngeal pathology which alters the periodicity of vocal fold vibrations.

The current study revealed frequency perturbation measures of jitter% being higher in clinical group when compared to control group. Frequency perturbation measures being higher in children with cleft palate are supported by studies by Zajac and Linville (1989), Lewis, Andreassen, Leeper, Macrae and Thomas (1993), and Van Lierde et al., (2003).

Zajac and Linville (1989) in their study aimed at studying the relation between the voice perturbations and perceived nasality in children with VPI. The results of their study indicated that the voice perturbations of children with hoarseness correlated moderately with perceived nasality. The authors discuss this finding of their study as an evidence for the link between the laryngeal and velopharyngeal events. They explain the findings on the basis of aerodynamic regulation. It is known that some amount of voice perturbations is present even in normal voice and they are indicative of sudden changes in the aerodynamic and neuromuscular events. However increased jitter, suggest an altered aerodynamic or neuromuscular event due to the oro- nasal coupling caused by VPI. An open velopharyngeal port during the production of the vowel may cause changes in the flow rate and the transglottal pressures. An individual with VPI attempts to compensate for these changes by increasing the glottal resistance and thus would decrease the flow rate and thus would maintain the subglottic pressure required to sustain phonation. Thus these efforts at regulating the aerodynamic and neuromuscular processes, when the coupling of the oro nasal cavity is inappropriate may result in increased voice perturbations.

Van Lierde et al., (2003) also supported the finding of increased jitter% in voice of individuals with VPD by correlating it with increase in hoarseness. The authors also reported the

presence of vocal nodules and muscle tension dysphonia type I in 9 of 28 of their participants. The presence of such laryngeal pathology causing alteration in the periodicity of the vocal fold vibrations may also be the reason for increased value of the perturbation measures.

DSI considers highest fundamental frequency as one of the important constituent parameters. In more than 50% of dysphonic individual, the vocal folds are afflicted with excess mass as in case of vocal nodules, edema. This extra mass usually unevenly distributed along the cords hamper the higher vibratory rate. This is reflected by decreased F_0 high (Wuyts et al., 2000).

The highest fundamental frequency was lower in the clinical group when compared to the control group. The highest fundamental frequency being lower in clinical group may be explained as a compensatory strategy to reduce hypernasality (Vanriper, 1954 and Boone and McFarlane, 1988) while Westlake and Rutherford (1966) suggest that lowered frequency range is associated with nasality. Studies by Rampp and Counihan (1970) and Van Lierde, De Bodt, Baetens, Schrauwen, Van Cauwenberge (2003) also reported use of lower F_0 in children with cleft palate.

Rampp and Counihan (1970) reported lower fundamental frequency in individuals with cleft palate compared to the control group. They explained that there is actually an increase in the fundamental frequency in the cleft palate group which is masked by some factors and thus causes lowering of fundamental frequency. The authors suggest that when there is a velopharyngeal dysfunction, due to oro- nasal coupling, there is large amount of energy absorption. Due to the large amount of energy that is lost, an individual with cleft palate has to generate greater acoustic energy at the laryngeal level. Curtis (1968) posited that an individual with VPI must use greater subglottic pressure to compensate for the energy lost due to oro-nasal coupling. Since this abnormally high pressures must be maintained at all intensities, the fundamental frequency

increases at all levels. Rampp and Counihan (1970) opined that this increase in fundamental frequency is masked by factors like hyperemia and hyperplasia of vocal folds in individuals with cleft palate. These changes in vocal folds occur either as a result of the upper respiratory tract infections which these children are prone to or due the development of vocal nodules as a result of vocal abuse. Increase in mass of the vocal folds due to either of the reasons, results in lowering of the fundamental frequency.

Rampp and Counihan (1970) also suggested another explanation for the decrease in the fundamental frequency in individuals with cleft palate. They suggest that when the vocal tract cross sectional area is increased, it results in decrease in impedance in transmission of glottal sound energy through the vocal tract. This increase in vocal tract cross sectional area is achieved by altering tongue heights and mouth openings. The decrease in impedance causes greater intensity levels to be achieved at lesser increments in subglottic pressure and fundamental frequency.

b. Effect of gender on DSI and its constituent parameters

MPT was higher in males than females in both clinical and control groups. MPT is related to air volume available and is proportional to vital capacity which in turn depends on the gender, age and is in general reported to be higher in males (Yanagihara and Koike, 1967). Prior studies have also reported MPT to be higher in males than females (Hirano, 1981 and Wuyts et al., 2000). Shorter maximum phonation duration in females in both clinical and control group may be attributed to the shorter vocal tract in female children compared to males. Shorter vocal tract permit shorter expiratory airflow and thus shorter maximum phonation time (Shigemori, 1977 and Venugopal, Rajasudhakar and Savithri, 2005).

Jitter % was higher in males when compared to females in both clinical and control group. Frequency perturbation jitter % being higher in males in both the groups may be attributed to the difference in fundamental frequency. Study by Robert and Baken (1984) reports of decrease in jitter% as the fundamental frequency increases.

DSI values were lower in males when compared to females in both the clinical and control group. DSI values being lower in males in both the groups may be due to the reason that boys are in general involved in vocally abusive behaviors like screaming when compared to girls (Moore, 1986 and Senturia and Wilson, 1968). This was contradictory to the finding by Wuyts et al., (2000) who reported the absence of gender effect caused by opposite counteracting of Hf_0 (higher in females) and MPT (higher in males). However, Jayakumar and Savithri (2012) reported significant effect of gender for DSI values and attributed this to the smaller difference in MPT values between the male and female participants in their study which failed to cause the counter balance between MPT and Hf_0 .

III. Comparison of control and clinical groups based on cepstral analysis.

Cepstrum gives a display of the harmonic structure. To obtain a cepstral peak prominence (CPP), a regression line is drawn through the cepstrum. CPP is the difference in magnitude of the highest peak of cepstrum to the value of the cepstrum that is obtained through a regression line. It gives an indication of how much the peak of cepstrum is above the average value obtained from the regression line. CPP is a reflection of the degree of harmonic organization. When individual cepstrum are averaged over a number of frames then smoothed cepstral peak prominence (sCPP) is obtained. Both CPP and sCPP have shown to be good predictors of dysphonia (Hillenbrand, 1996; Hillenbrand, Cleveland and Erickson, 1994; and Olson, Goding and Michael, 1998)

CPP and CPPs did not show any significant difference between the clinical group and control group. No significant difference between the two groups suggested that the harmonic structure of voice was not significantly altered in children with cleft palate. The CPP and sCPP not showing significant difference between the two groups could be due to two reasons. The amount of breathiness in the clinical group might not be significant enough to alter the periodicity and thus the harmonic structure to bring about a change in the cepstrum. Secondly as evident from the study by Yolanda, Heman, Deirde and George (2001), CPP and sCPP might not be a reliable predictor of the roughness and thus couldn't highlight the roughness in voice in the clinical group.

Yolanda, Heman, Deirde and George(2001) attempted to correlate measures of dysphonia with CPP and CPPs. They suggested that cepstral analysis was used since it did not depend on accurate fundamental frequency calculation. The authors report the ability of CPP and sCPP to predict the overall dysphonia and thus demonstrate the robustness of this measure when compared to other acoustic measures. They highlight the low correlation of roughness and the CPP and sCPP. They suggest that CPP and sCPP poorly correlating with roughness probably suggested that there might be some other factor other than periodicity that is a better predictor of roughness.

However a contradictory finding was reported by Garnier, Gallelo, Collet and Berger Vachon (1996). They aimed to find a spectral measure that can best differentiate voice of individuals with velopharyngeal impairment from that of normals. It was reported that cepstral analysis was a more efficient way to discriminate the two voices when compared to other methods like formant analysis and bark FFTs which is a contradictory finding to the present study. The authors reason that the formants were determined by ILS software and thus its results

may be altered by its algorithm. Vowel/a/ was reported to be the most appropriate speech material used for discrimination. They report that this difference lies in the sensitivity of different vowels to the modification of the nasal component of voice in accordance with the geometry of the vocal tract. For some configuration the pathology might not be sufficient enough to cause a shift in the parameter and thus discrimination of the voices becomes even more difficult. A nasalized vowel for instance, even in case of pathology does not bring about much change in this parameter. The authors also reason that the modifications in spectral characteristics of vowel are required for detecting the presence of nasality. The presence of an additional pole zero in the valleys of the spectrum helps in discriminating the two voices. The authors further extrapolate the findings that the presence of pathology introduces additional noise and thus affects the spectral characteristics.

IV. Comparison of control and clinical groups based on laryngeal aerodynamic measures.

During breathing and swallowing, the respiratory and phonatory subsystems function autonomously. The two subsystems however act in unison during voice and speech production. The respiratory and laryngeal subsystems must coordinate to maintain a constant subglottic pressure, a constant airflow and a relatively constant upper airway construction for production of steady state utterances. The laryngeal aerodynamic measures capture relations among respiratory and laryngeal function and would reflect the coordinated nature of the voice productions. Although they do not give a direct indication of specific muscular or mucosal activity, it reflects relations across the sum of passive and active respiratory and phonatory forces and aerodynamic factors in voice production.

Laryngeal airway resistance in the present study was higher in the clinical group when compared to the control group; however the difference was not statistically significant. This may be attributed to compensation at the laryngeal level, where speakers with velopharyngeal dysfunction use greater adductory force on their laryngeal structures. The higher laryngeal airway resistance may be due to the fact that children with cleft in order to compensate for the air leakage at the velopharyngeal port using increased muscular effort. This has been supported by electromyographic studies of Kuehn and Moon (1995) which showed greater physiological effort for the levator activity for velopharyngeal closure during speech.

The results of the present study indicated no significant difference in laryngeal airway resistance between the control and clinical groups. Zajac (1995) studied laryngeal airway resistance in individuals with complete and incomplete velopharyngeal closure associated with cleft palate. The author reported no significant difference in laryngeal airway resistance between the groups and opined that the velopharyngeal orifice size in the incomplete closure group is minimal and probably not requiring compensation at the laryngeal level. The participants in the clinical group of the present study might probably have minimal velopharyngeal orifice size, thus not bringing about a significant increase in the laryngeal airway resistance.

Lewis et al., (1993) investigated laryngeal airway resistance in children with cleft palate. The authors reported greater estimated transglottal pressures, glottal airflows and glottal resistance in children with cleft palate, with or without cleft lip compared to the control group. The authors further reported that there existed two subgroups among children with cleft palate based on the type of vocal strategy used by them. One subgroup showed high trans-glottal pressures and resistances while the other showed low trans-glottal pressures and resistances. The

authors explained this difference in vocal strategy as learnt and perseverated and also may be due to the difference in the size of the velopharyngeal orifice within the adequate range.

Thus, no significant difference between the clinical and control group across laryngeal airway resistance in present study may be due to above mentioned reasons. Either the velopharyngeal orifice size might be minimal (Zajac, 1995) or it might be attributed to the type of compensatory strategy adopted by them (Lewis et al., 1993).

In the present study subglottic pressure was significantly higher in the clinical group when compared to the control group. Curtis (1968) reported that in individuals with cleft palate due to the oral nasal coupling there is large amount of energy being absorbed. To compensate for this energy loss and to maintain the same level of intensity, greater levels of subglottic pressure must be built. This increased subglottic pressure is required to maintain the required intraoral pressure. Lewis et al., (1993); and Zajac, 1995 also reported high subglottic pressures in cleft palate children.

Findings of increased subglottic pressure in the present study are also in consensus with D' Antonio et al., (1988). They studied the prevalence of voice disorders in 85 individuals with velopharyngeal dysfunction. The authors report of the laryngeal aerodynamic parameter of subglottic pressure was significant and the trend showed a higher than normal value in individuals with laryngeal abnormalities when compared to those without laryngeal abnormalities. They also report of difference in the way the individuals with velopharyngeal dysfunction compensated for the abnormalities in the laryngeal function. 43% of the velopharyngeal dysfunction individuals with vocal thickenings exhibited increased respiratory effort to compensate for their vocal pathologies. At the same time 31% of them had reduced respiratory

effort, suggesting that this subgroup of users with velopharyngeal inadequacy may have tried to cover up for their defects by using reduced respiratory effort.

Airflow measures were higher in clinical group in the present study however; the difference between the two groups was not significant. This may be due to the fact that the velopharyngeal insufficiency in these participants might be small enough that they need not have to compensate for the airflow leak (Lewis et al., 1993; and Zajac, 1995).

Warren et al., (1969) reported that individuals with cleft palate use air volumes double that of normal. This is due to increased rate of airflow and increased time interval taken for the production of an utterance in individuals with cleft palate. However there are limitations of compensating the velopharyngeal incompetence by increasing the respiratory effort. The airflow can only increase 600-800 cc per sec (Warren and Hinton, 1983).

Guyette, Sanchez and Smith (2000) investigated the effect of velopharyngeal dysfunction on laryngeal aerodynamic parameters. Two subgroups were formed on the basis of velopharyngeal orifice size. The subgroup with velopharyngeal size lesser than 1mm^2 formed the complete closure group, while those with velopharyngeal size greater than 5mm^2 , formed the incomplete closure group. The two subgroups were compared on laryngeal aerodynamic parameters of laryngeal airway resistance, transglottal airflow and transglottal pressure. One subgroup showed high trans-glottal pressures and laryngeal airway resistance resistances while the other showed lower transglottal airflow. The mean transglottal airflow was higher in the incomplete closure group when compared to the complete closure group.

Another reason suggested is the difference in the size of the velopharyngeal opening. Leeper et al., (1994) compared children with cleft palate to the control group and found a relation

between the size of velopharyngeal opening and the aerodynamic measures, though it wasn't statistically different. They found that when the velopharyngeal orifice size was greater than 5mm^2 they exhibited lower laryngeal airway resistances, lower subglottal pressures, and higher trans-glottal airflows when compared to cleft children with the velopharyngeal orifice size less than 5-mm^2 and the control group. The subjects in the clinical group in the present study probably had a velopharyngeal orifice area of greater than 5mm^2 which however couldn't be confirmed.

Sapienza (1993) reported increase in glottal airflow when loudness increased from soft to comfortable to loud. The non-cleft children in general exhibited higher level of resistance yet they were within the normal range. It was further seen that though no significant difference was noted between the cleft and non- cleft group there existed a trend. Children with cleft palate and incomplete closure exhibited large increase in laryngeal airway resistance when their nostrils were unoccluded. The mean difference for the group was $10\text{ cmH}_2\text{O/L/s}$. This trend was in support of the regulation hypothesis, which suggests that these children use increased laryngeal airway resistance in order to limit the nasal escape of air or to help regulate subglottic pressure or both.

V. Comparison of DSI scores between the clinical sub-groups

Although the results of the present study indicated reduced DSI values indicating abnormal voice quality in individuals with VPD associated with repaired cleft palate, it is not clear whether the extent/degree of VPD has a differential effect on their dysphonia severity. Therefore, an attempt was made to subdivide the participants under the clinical group based on their nasalance scores. A statistical tool called Dendogram was used to divide the clinical group into two clusters based on their nasalance scores. The clusters thus obtained had 18 and 11

participants under each subgroup and obtained different DSI scores. However, one way MANOVA did not reveal significant difference in DSI values between the clinical subgroups indicating no effect of extent/degree of velopharyngeal dysfunction on their dysphonia severity as measured on DSI.

The lack of difference between the clinical subgroups might be due to the absence of marked difference between the two groups in terms of their velopharyngeal orifice. Lewis et al., (1993) also reported no significant difference in the transglottal airflow, subglottal pressure, and laryngeal airway resistance in children with cleft palate with velopharyngeal port size of 1.17mm^2 and the control group with velopharyngeal port size of 1.1mm^2 . However, Thomas et al., (2000) reported higher trans-glottal pressures and resistances in individuals with higher velopharyngeal size ($>5\text{mm}^2$) compared to those with normal velopharyngeal port closure ($<1\text{mm}^2$). From these studies it may be concluded that the influence of VPD on laryngeal system depends on the size of velopharyngeal orifice on closure. Therefore, it might be possible that the participants under clinical subgroups in the present study do not have adequate variation in their velopharyngeal closure to influence their laryngeal system.

Another reason for lack of difference might be the validity of sub grouping the participants itself. Although the participants with VPD were sub grouped based on their nasalance values, this is a preliminary effort and the validity and efficacy of such procedure is unknown and requires further studies.

Regulation control hypothesis

The compensations at the laryngeal level due to a velopharyngeal dysfunction in the participants of the present study may be explained on the basis of regulation control hypotheses

(Warren, 1986). Most of the maladaptive and compensatory behaviors in children with cleft palate have been explained on the basis of a regulating system. Warren (1986) describes larynx as one of the potential valves for regulating speech aerodynamics when velopharyngeal inadequacy is present. The increased prevalence of voice disorders in individuals with cleft palate has been hypothesized by the regulation control model. Warren uses this model to describe that in order to maintain constant pressure for speech production, an individual may increase laryngeal airway resistance when there is reduced velopharyngeal resistance. This is further supported by the evidence that individual with velopharyngeal inadequacy further use greater respiratory volumes. It was further seen that some children use generalized laryngeal tension as the compensatory valving mechanism. When there is error in the system the aerodynamic stability is maintained at the expense of speech production. Greater respiratory effort result in an increased airflow rate and since resistance is flow dependent, the resistance along the vocal tract including the glottis increases, and as a consequence greater intra oral pressure can be achieved. Glottis serves as an expiratory brake during respiration and adducts more during expiration than during inspiration.

A glottal stop used by cleft palate individuals is a more forceful manifestation of this reflex. The study by Warren (1985) suggested that a speech regulating system aims to maintain constant pressures and the compensatory behaviors used by the individuals with cleft palate are control responses to maintain it. Warren (1985) hypothesizes that speech aerodynamics in individuals with cleft palate fit in the theory of regulating/control system. Compensatory behaviors associated with velopharyngeal inadequacy represent behaviors that satisfy the requirements of a speech regulating system (Warren, 1989).

The hypothesis further explains that in order to compensate for the decrease of resistance within the vocal tract, attempt is made to maintain adequate pressures for consonant production. Strategies employed by population of speakers with structural defect ensure the stability of pressure. The vocal tract may be considered as a tube with oral, nasal, pharyngeal and glottal components with the lungs being the source of energy. The speech motor control program coordinates the activities of these structures so that functionally and anatomically distinct parts of the speech system act together towards achieving a common goal. In this respect, the respiratory and articulatory systems are theorized to work in a coordinated manner for the purpose of regulating speech (Warren, 1986). To maintain the constancy of subglottic pressure, the elastic forces are enhanced and the abrupt changes in the respiratory load that occurs with opening and closing of the upper airway are compensated for. Accurate control over the movement of the upper airway structures and airflow tend to keep subglottic pressure controlled (Warren, 1982).

Warren (1985) proposed a possible sequence of control mechanisms to velopharyngeal incompetence. Greater respiratory muscle activity results in greater airflow rate. Velar resistance would increase as the resistance is flow dependent and as a consequence, greater intraoral pressure would be achieved. Warren further opined that subjects with velopharyngeal dysfunction might provide an opportunity to assess how the speech system is maintained and regulated when an error is introduced. The results of the present study indicated compensatory influence of velopharyngeal dysfunction on the laryngeal system. This is evident from the higher values of ESGP, LAR, Perturbations and reduced values of DSI, MPT, HFo used by individuals with velopharyngeal dysfunction associated with repaired cleft palate. Thus, the results of the present study support the hypothesized regulation control by subsystems of speech production.

CHAPTER VI

SUMMARY AND CONCLUSION

The voice characteristics in individuals with cleft palate have been explored at different levels like acoustic, aerodynamic, physiological and perceptually. However most of these studies are based on uni-parametric approaches and the results have been inconclusive. The present study hypothesizes the effect of velopharyngeal dysfunction in individuals with repaired cleft palate on the laryngeal system. To verify this, voice of individuals with repaired cleft palate and age matched controls were assessed on a multidimensional paradigm.

The study included a clinical group which consisted of 30 children with repaired cleft palate in the age range of 4-14years. 40 typically developing children matched for age and gender with no history of any craniofacial abnormalities and no voice problems formed the control group. All the participants in the clinical group were diagnosed with VPD through clinical examination by SLP and plastic surgeon. Further the diagnosis of VPD was confirmed using objective method of nasality and nasal airflow using nasometry and glattzel mirror respectively. The voice characteristics in clinical and control groups was assessed using perceptual, acoustic and aerodynamic parameters.

The perceptual assessment included rating the voice samples of sustained vowel/a/ by three experienced judges on GRBAS scale. The acoustic assessment involved measurement of dysphonia severity index (DSI) and cepstral analysis. DSI was measured from its constituent parameters of highest fundamental frequency (Hf_0), Lowest Intensity (Li_0), Jitter% and Maximum Phonation time (MPT). Highest frequency and lowest intensity was measured by using the voice range profile (VRP) module of Computerized speech lab (CSL)(*Kay Pentax Corp, model 4500*),

while jitter% was measured using the Multidimensional Voice Profile (MDVP) module. Maximum phonation time was calculated using CSL (*Kay Pentax Corp, model 4500*). Cepstral analysis involved measurement of cepstral peak prominence (CPP) and smoothed cepstral peak prominence (sCPP) using Hillenbrand's software speech tool. The sample used was a 4 second steady portion of vowel /a/. Aerodynamic assessment involved measurement of parameters estimated subglottic pressure, mean airflow rate and laryngeal airway resistance. The instrumentation used for aerodynamic assessment was Aeroview1.4.4.

The obtained measures was organized and tabulated and subjected to appropriate statistical tools. The results of the statistical analysis revealed that males in the clinical group had a more deviant voice quality with GRBAS scores indicating slight breathiness, hoarseness and strained voice quality. However the females in the clinical group showed normal scores on the GRBAS scale. DSI and its constituent parameters of Hf_0 , Li_0 , Jitter% and MPT were compared between the two groups. DSI scores were lower in the clinical group when compared to the control group. Clinical group showed significantly higher values for jitter % compared to the control group. MPT and Hf_0 showed significantly lower values in the clinical group compared to the control group. Li_0 showed no significant difference between the two groups. Cepstral analysis including both CPP and sCPP showed no significant difference between the two groups. Results of aerodynamic analysis revealed that all the aerodynamic were higher in the clinical group compared the control group however significant difference was observed only in case of subglottic pressure. A statistical tool, dendrogram was used to form two clusters from the clinical group based on the nasometry values. The two clusters were compared across DSI and its constituent parameters to analyze the extent of velopharyngeal dysfunction on the voice

characteristics in individuals with cleft palate. The results revealed that there was no significant difference between the two clusters across DSI and its constituent parameters.

The results of the study are discussed in light of the regulation control hypothesis (Warren, 1986) which explains how compensation occurs at one of the subsystems, when error occurs in the other. According this model, in order to maintain constant pressure for speech production, an individual may increase laryngeal airway resistance when there is reduced velopharyngeal resistance. This is further supported by the evidence that individual with velopharyngeal inadequacy further use greater respiratory volumes. It was also observed that some children use generalized laryngeal tension as the compensatory valving mechanism. When there is error in the system the aerodynamic stability is maintained at the expense of speech production. Greater respiratory effort result in an increased airflow rate and since resistance is flow dependent, the resistance along the vocal tract including the glottis increases. As a consequence greater intra oral pressure can be achieved. The speech motor control program coordinates the activities of these structures so that functionally and anatomically distinct parts of the speech system act together towards achieving a common goal. In this respect, the respiratory and articulatory systems are theorized to work in a coordinated manner for the purpose of regulating speech.

The limitations of the present study are that the presence or absence of velopharyngeal dysfunction could be determined only by using nasometry and glattzel mirror. However the quantification of velopharyngeal dysfunction using videofluoroscopy couldn't be done. Therefore further studies could consider the quantification of VPD using videofluoroscopy as this might facilitate better understanding of influence of VPD on the laryngeal system. Also it would provide useful information on how different size of velopharyngeal port closure affects

the voice differently. Secondly, the presence of vocal pathology in these individuals couldn't be confirmed by means of video imaging techniques like stroboscopy and endoscopy. The use of such imaging methods in future studies might provide insight in understanding the mucosal wave alterations as well as the supraglottic compensations in these individuals.

The findings of this study provide useful insight in understanding the influence of velopharyngeal dysfunction on the laryngeal system and the voice characteristics in repaired cleft palate individuals with VPD. It is thus helpful in determining the therapy goals for individual with repaired cleft palate.

Several variables can influence measurement of laryngeal aerodynamics in individuals with velopharyngeal dysfunction. Some of these are measurement of estimated subglottic pressure using airway interruption method assumes the subglottic pressure to be equal to the intraoral pressure when the vocal tract acts as a single chamber. However, the presence of oro nasal fistulas in 10 of the participants affected the measurement of laryngeal aerodynamic measures efficiently. The laryngeal aerodynamics in the cleft palate population can be further studied under conditions of unoccluded and occluded nares. The present study did not consider constant intensity levels while measuring the subglottic pressure. Future studies may control this factor when laryngeal aerodynamic parameters are measured in order to obtain more reliable laryngeal aerodynamic measures.

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