A FEW OBJECTIVE MEASUREMENTS OF QUALITY OF VOICE

IN

CLEFT PALATE INDIVIDUALS

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Reg.No. M8904

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DEDICATION

IF YOUTH IS BRAVE

AND ALSO BOLD

THEN,

'OLD THEY SAY'

IS ALWAYS GOLD

" TO MY GRAND FATHERS WHOSE VOICES FROM THE HEAVEN EMBARKED MY HEART AND ENCOURAGED ME AND WHOSE IMAGERY PICTURES FLASH IN MY MIND EVEN NOW. AND TO MY GRAND MOTHERS WHO LEFT EVERLASTING GREEN MEMORIES IN MY HEART WITH THE LOVE THEY GAVE ME "

CERTIFICATE

This is to certify that the Dissertation entitled " A few objective measurements of quality of voice in Cleft Palate individuals " is the bonafide work in part fulfilment for the Degree of M.Sc, (speech and Hearing) of the student with Register No.M8904.

Mysore
May 1991

Director
All India Institute of
Speech and Hearing
Mysore-6.

CERTIFICATE

This is to certify that this Dissertation entitled " A Few Objective Measurements of Quality of Voice in Cleft Palate individuals" has been prepared under my super-vision and guidance.

DECLARATION

I hereby declare that this Dissertation entitled: "A Few Objective Measurements of Quality of voice in Cleft Palate individuals" is the result of my own study under the guidance of Mr.c.S.Venkatesh, Lecturer in Speech Sciences, All India Institute of Speech Hearing, Mysore-6, and has not been submitted earlier at any University for any other Diploma or Degree.

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INTRODUCTION :

"Voice is the laryngeal modulation of pulmonary air stream which is further modified by the oonfiguration of vocal tract. (Micheal and Wendahl, 1971)".

Voice plays an important role in speech communication. The production of voice depends on the synchrony between respiratory, phonatory and the resonatory systems. Anatomical or physiological deviations in any of these systems would lead to a voice disorder, cleft Palate is one of the anatomical deviations of speech mechanism. This alters the resonatory system of speech production mechanism effecting both resonatory and phonatory aspects of voice.

The presence of voice disorders in individuals with Cleft Pala have been documented by many investigators. (McDonald and Koeppbak 1951; Westlake, 1953; Hess, 1959; Brooks and Shelton, 1963; Bzoch, 1964; McWilliams, Bluestone and Musgrave, 1969; Marks, Barker and Tardy, 1971 and D8Antonio, Muntz, Province and Marsh, 1988). These Investigators have reported phonatory problems, such as hoarseness (both with and without vocal cord pathology), breathiness, reduced loudness, deviant speech restricted pitch range and tensed straine vocal quality.

McDonald and Koeppbaker (1951) stated that faulty phonation which is a problem among Cleft Palate individuals results in voice quality of breathiness and hoarseness.

Bzoch (1964) reported that hoarseness and breathiness are associated with pharyngeal flaps and speculated that these aspirated phonation might occur as a response to hypernasal distortion and might represent an effort to mask hypernasality.

McWilliams et al (1969) reported of bilateral vocal cord nodules in Cleft Palate individuals along with velopharyngeal incompetance. They suggested that these children may have had laryngeal compensation other than glottal stops that contributes to the development of their vocal pathology. They concluded that a logical relationship exists between VPI and vocal cord nodules.

Curtis (1968) suggested that individuals with cleft palate may need to exert greater respiratory effort to achieve normal inteasity level because of acoustic damping in the nasal tract. Analog model studies (House and Stevans, 1956) and in vivo studies (Brenthal and Beukeiman, 1977) have demonstrated that the overall sound energy of vowels is reduced as a consequence of oronasal coupling. Warren, Dalston, Morr and Smith (1988) have further suggested individual may increase respiratory effort as a way to develop adequate intraoral air pressures. However, the increased respiratory effort may contribute to vocal abuse.

Hamlet (1973) reported that the vibratory characteristics of vocal cords were altered when nasality was present. Specifically Hamlet (1973) found that the opening phase of the glottal cycle was reduced for nasalised vowels compared with non nasalized vowels even when vowels were matched for intensity. Hamlet (1973) Interpreted these results to indicate that nasalization increased the force of vocal cord adduction independent of the level of vocal effort.

Leder and Lerman (1985) reported that adults with cleft palate and clinically significant hypernasality demonstrated in appropriate vocal cord adduction and voicing during the production of voiceless stop plosives. They suggested that phonation was facilitated by transglottal pressure change that resulted from inadequate velopharyngeal function. They further suggested that inappropriate voicing may serve to reduce nasal air emission.

D'Antonio et al (1988) reported that 41% out of 85 samples of a group of individuals with VPI exhibited laryngeal abnormality and/or abnormal voice qualities. The prevelance of voice disorder was not significantly different between normals and cleft palate cases. But with symtoms of inappropriate velopharyngeal valving there was a signficant relationship between the presence of vocal findings and increased subglottal pressure. Although a cause and effect relationship was not established.

The significant degree of nasality associated with cleft palate may result from morphological involvements in the speech structures particularly the velum is well recognized (McDonald and Baker, 1951; Johnson, 1952). Hoarseness among cleft palate speakers has been explained as a result of;

(a) Chronically inflammed and swollen tissues of vocal cords (McDonald and Baker, 1951)
(b) Habitual glottal articulation (Seth and Gothrie, 1935)
(c) ventricular voice (Berry, 1956)
(d) Lowering of pitch and increased intensity (Hess, 1959)

Breathiness of cleft palate speech may be a vocal consequenc of faulty phonation (McDonald and Baker, 1951).

However, there are very limited number of studies which tri to evaluate the vocal fold functions objectively in cleft palate individuals.

The present study is designed to investigate the laryngeal functions and amount of noise component in the voice of cleft palate/individuals and to compare them with normals using pitch perturbations, intensity perturbations and long term average spectrums measurements. Relative average perturbations (RAP) for frequency and intensity, Directional perturbations for frequency and intensity and Alpha ratio, Seta ratio and Gamma ratio using LTAS has been taken as a tool for measurements.

Specifically, the study aims at testing following hypothesis

1. There will be significant difference between cleft Palate and lip individuals and normals for vowels /a/, /i/ &/u/ in terms of ;

   (a) Relative average perturbation for frequency
   (b) Relative averge perturbation for intensity
   (c) Directional perturbation for frequency
   (d) Directional perturbation for intensity

2. There will be significant difference between cleft palate and lip individuals and normals for alpha, beta and gamma ratios of long term average spectrum measurements.

In this study 13 Cleft Palate and lip subjects (6 males and 7 females) in the age range of 8 years to 25 years were served as subjects. For each subject two measurements of pitch perturbation and intensity perturbation were carried out namely ;

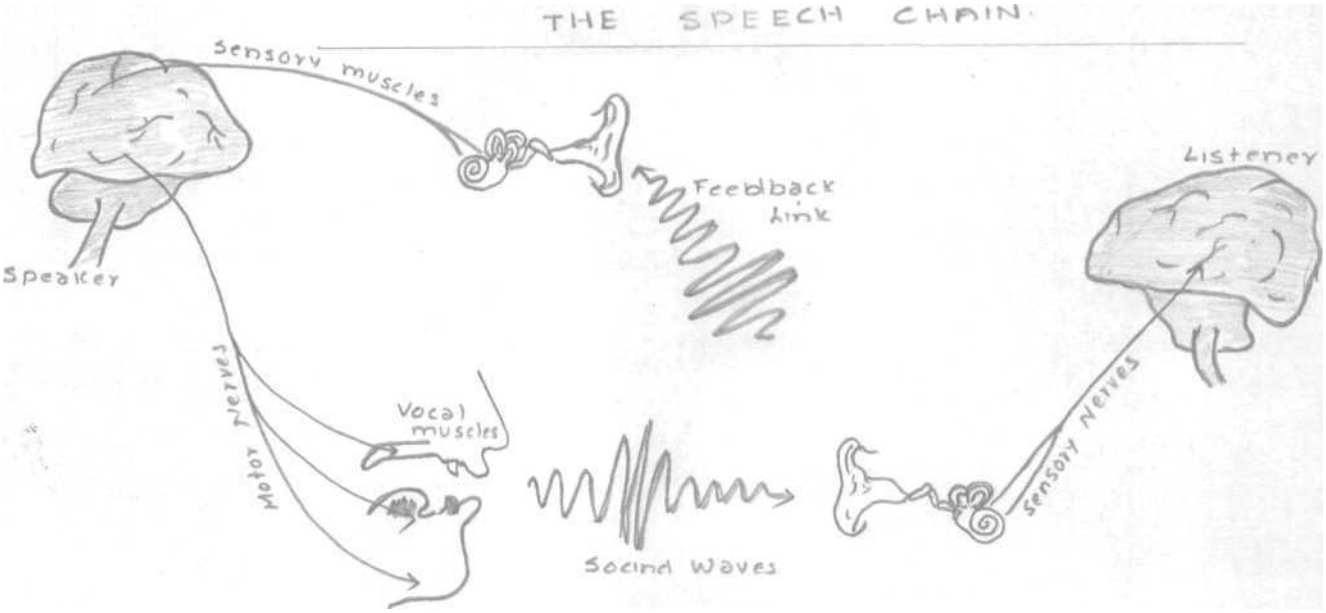
1) Relative average perturbation
2) Directional perturbation for three vowels /a/, /i/ & /u/ and Alpha, Beta and Gamma ratio for LTAS were studied.

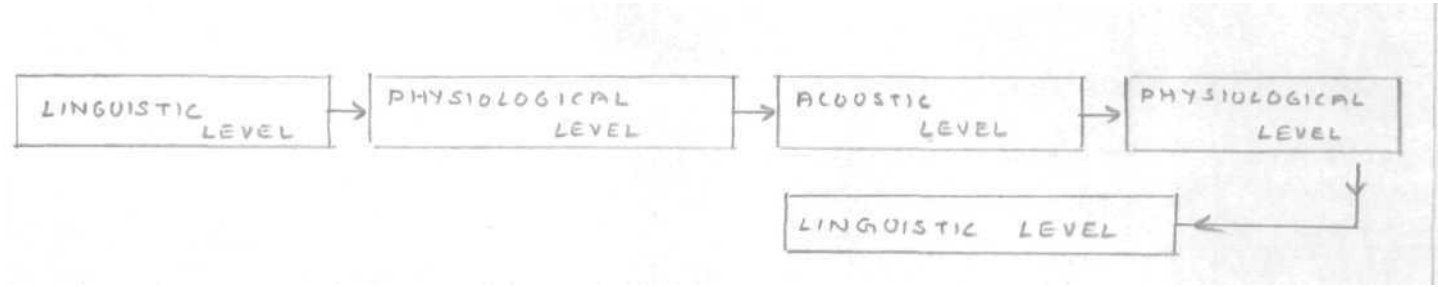
REVIEW OF LITERATURE

Speech is unique to human being and it is a complex human behaviour. It can be defined as a genetically determined individual psycho physiological activity consisting of the production of phonated, articulated sound through the inter-action and coordination of cortical, laryngeal and oral structure (Newman, 1963). Speech exists only in concrete actualities. Although it can be developed in some species through training it seems to developed spontaneously only in human beings.

The movement of vocal organs generate a speech sound wave - that travels through the air between speaker and listener. Pressure changes at the ear activate the listeners hearing mechanism and produce nerve impulses that travel along the acoustic nerve to the listener's brain. In the listeners brain considerable amount of nerve activity is already taking place and this activity is modified by the nerve impulses arriving from the ear. This modification of brain activity in ways we do not fully understand, brings about recognition of speakers message. Since speech communication consists of chain of events linking the speakers brain with the listeners brain. This chain of events can be termed the speech chain which is represented in the figure 1.

Production of speech requires simultaneous and coordinated use of respiratory, phonatory, articulatory and resonatory system controlled by the nervous system.





Development of speech depends on many factors ;

A. Adequate function of central Nervous System
B. Adequate Feedback Mechanism
C. Adequate Intelligence
D. Adequate sensory Motor Development
E. Structure and function of Oral cavity

Defects at any of these levels lead to speech disorder. Defect in oral cavity leads to abnormal speech. Cleft Palate and lip is one type of defect in the oral cavity. They are malformation that occur in utero and are present at birth. Deformaties similar to these congenital defect can also be acquired due to severe injury to the oral structure and from ablative surgery, usually during the treatment of malignant tumors. Some problems

associated with these acquired deformities resemble those that result from congenital abnormalities.

The term Cleft accurately describes the major deformities under consideration. The upper lip may be open on one or both sides and the palate or roof of the mouth may be divided so that oral and nasal cavities are coupled. More extensive cleft may affect the face, the nose, the eyes and related structures. These opening represent developmental failure or disruption in the midface and oral cavity.

A complete cleft of the lip and palate extend from the external lip posteriorly through the alveolar arch and the hard and soft palate. The soft palate and uvula are split in half. The nasal septum is usually attached to the larger of the two palatal segments in unilateral cleft, but is not attached to bilateral cleft. In both cases, the inferior aspect of the septum frequently can be seen during examination of oral structure

Isolated cleft palate without cleft lip may include all of the hard palate posterior to the incisive foramen and soft palate. It may involve only a small portion of the posterior part of the soft palate or it may be between these two extremes. The simplest defect is by bifid uvula which many people refer to as double uvul In reality it is two halves which never fused. , In & of itself, it is not usually symptomatic. In these cases the nasal septum is not attached to either segment, but is at the midline and can be seen during physical examination, if the cleft is wide enough and include the hard palate.

Causes not established, the theories of cleft palate at present emphasize the interruption of the nutritional or oxygen supply to

the embryo. The interruption may be attributed to extreme mal-nutrition to the mother, vitamin A,B deficiency the Rh incompata-bility or to an atypical or insufficient circulation in the embry Defective development at the periphery of adjoining process at this time interferes with fusion and once the schedule time is elapsed, further growth would widen the gap (Stark, 1954). Although Kaufman (1946) stresses the importance of heredity, he also believes that a defective vascular supply, particularly at the terminal distribution may be a cause of arrested development. He suggests that vascular supply to the right half of the head is greater than to the left and therefore it probably accounts fo] the more frequent occurance of the cleft of the lip and alveolar process on the left side.

The experiment conducted by the Harvard School of Public Health in 1951 on the subjection of mice to intrautrine anoxia suggests that anoxia may be the cause of many structural anamolies Mechanical obstruction in utero are stressed frequently in medical literature as probable cause, utrine tumors, cord interference-pressure of the lower jaw and the interference of the tongue and of hands and feet, when the fetus is in a state of flexure may operate to retard or obstruct the normal union of the palate.

Among all the possible causes of oral cleft, hereditory facto have received greater importance. In a study of the families of 100 cleft palate children Simon (1950) reported that the defect had appeared either in one of the parents, in the brothers and sisters of the parents or in the sibilings of the propositors. Kemper (1944) also supports the genic theory and he states that in 20% of the cases the deformity is found in the immediate family or in one of its collateral branches. Although Ritchie (1955)

found a familial incidence in only 9.7 percent of his cases, he sites the survey of Devis (1951) who reported one thousand cases of various deformities of the face and jaw with hereditory tendencies in 54 percent. Coursin (1950) discusses a constellati of genic factors which may account for 50 percent of cleft palate cases. Another theory is that one may inherit a constitutional predisposition to vitamin deficiency which in turn can produce cleftness. The study of the relation of hypothyroidisms to cleft palate presents some evidence that the inheritable factor is a particular type of endocrine constitution which alone or assisted by unfavourable environmental factors causes cleft.

The manner of inheritence has been studied exhaustively by Foghandersen (1942). He concludes that in cases of harelip combined with cleft palate the manner of inheritance is presumabl that of conditioned dominance (conditioned by the genetic milieu with sex limitation to males. . . . . the gene occuring generally as a recessive gene. . . . .isolated cleft palate is only heriditary in rather small number of cases and the manner of inheritance is here in all likelihood that of simple dominance with failing manifestations and sex limitations for females. One conclude that genetics of cleft palate is inperfectly understood.

Speech and Language disorders among cleft palate individuals can be divided into;

(a) Voice Disorder

(b) Prosodic Disorder

(c) Articulatory Disorder

(d) Language disorder

VOICE DISORDER IN CLEFT PALATE :

Voice Disorder in cleft palate may be described under two headings ;

1. Resonatory Disorder
2. Phonetory Disorder

Resonatory Disorder:

Resonance, an acoustical phenomena is a complex attribute of speech, that is not completely understood. It may be defined as the vibratory response of a body or air filled cavity to a frequency imposed upon it (Wood, 1971). Thus resonance is physical rather than perceptual phenomena. Speech that is outstanding in any of in resonance characteristics depend upon the integrity of the supra-glottal structure. Any thing that upset the ideal relationship between oral and nasal cavities will be reflected in speech patten For example too much nasal resonance coupled with reduced oral resonance will increase the perception of nasality. The extent to which these characteristics are present will be influenced not only by the cleft but also on the degree of velopharyngeal valving deficit, nasal resistence, pharyngeal resistence and speech behaviour introduced by the speakers in attempt to achieve phonetic distinc-tion and/or mask VPI. Hypernasality, which is a resonatory disorder is a consequence of alteration in any of these factors.

The term Hypernasality implies that oral and nasal cavities are coupled, when they should not be, with their result that the sound wave is diverted into the nasal airways and speech sounds as if it is coming through nose. Hypernasality phenomena associated with vowels which are differentially affected by the

shift from a primary laryngeal, pharyngeal and oral system with competing oral and nasal cavities both playing role in resonating aspects of speech and serving to alter the acoustic signal so that its spectrographic characteristics are changed and the auditory percept of the signal is recognised as different*

Same related factors to hypernasality are respiratory effort and the degree of tension in the subglottal and supra-glottal structure including oral and nasal pharynx. Super imposed upon these elements is the positioning of the tongue which riding high posteriorly may essentially block oral access of the sound stream and effectively reduces or eliminate the possibility of oral resonance thus altering the relative oral nasal balance (McDonald and Baker, 1951). The extent to which this occurs in cleft palate speakers remain unclear. However Dickon (1969) reported that most hypernasal speakers use an anterior tongue carriage.

Constriction of oral part by mandibular positioning, movement, tension and lip function will further serve to restrict or minimise the oral component of speech, since cleft palate speakers have a tendency to restrict the size of cavity (Folk and Kopp, 1968) their hypernasality springing from velopharyngeal competance may be increased. In addition, the size of the velopharyngeal port during speech will in relation to respiratory effort and other tract feature dictate the air and energy lost into the nasal cavity.

Nasal cavities are complex in normals and are rendered more so in individual with cleft palate. These patients have deviated septum collapsed nares, large communicating oral nasal fistulae and chronic respiratory problem or some combination of these which may impedes or alter both air flow and characteristics of sound waves, which

will obviously pass through the part of the system which offers the least resistence. Tissue variation may be implicated. If velopharyngeal tissue is hypoplastic, there may be hypernasality even in the presence of velopharyngeal port. Such factor as the thickness of the soft palate and verticle depth of the contact between the velum and the pharyngeal walls may be factors in the hypernasality. Schuwartz (1971 & 1979) speculated that listening to nasalized vowels involves listening not only to the feature produced by the nasal coupling condition but also to normal variation of quality which are unrelated to nasal coupling. The degree to which hypernasality will be perceived by a listener depend upon the characteristics of the vocal tract and not only upon the size of the functions opeing in the velopharyngeal valve (Curtiz, 1968).

Hypernasality is a vocal resonance disorder. But the means by which a person having cleft palate attempts to compensate for hyperasality have been implicated as possible cause of laryngeal pathology (McWilliams and Bluestone, 1969). Two lines of reasoning can be advanced in support of this possibility. The first derives from theoretical and experimental analog studies by Fant (1960) House and Stevens (1956) have shown that changes in the resonating characteristics of vocal tract resulting from oronasal coupling can along cause a drop of 5 - 10 dB in overall vowel amplitude. A person with hypernasal resonance is operating at a considerable acoustical disadvantage and in order to be heard must compensate by greater vocal effort. There is a possibility that additional vocal effort required actually causes vocal abuse.

The seond line of reasoning considers that the effort to achieve adequate velopharyngeal closure with an inadequate

mechanism may cause a person to compensate a velopharyngeal distress. (McWilliams and Bluestone, 1969)- Zemlin (1968) reviewed the early literature on nasality and noted that there was considerable speculation about the possibility that hyper-nasality might have its origin in the larynx. He cited Curry (1910) as beleiving that one of the causes of nasality could be increased laryngeal tension. Other writers suggested that nasality may be atleast in part of the result of some abberations in the vibratory pattern of vocal fold. Fletcher (1947) noted the opening phase of the vocal cord to be different where hyper-nasality is present and pattern of movement to be asymmetrical with greater movement in the right than in the left folds. However as Zemlin pointed out, it is possible that articulation will act to modify the manner in which vocal fold vibrate.

Influence of Perceived Nasality to vocal Pitch :

It is felt that changing vocal pitch of people with hyper-nasality might tend to reduce the perception of hypernasality, However Kelly (1934) did not confirms a relationship between nasality and pitch. Hess (1959) has also failed to find a Strong relationship between habitual pitch and degree of perceived nasality. Sherman and Goodwin (1954) studied individuals with functional hypemasality and hence there was probably no consonant disintegration. The subjects read a passage at his or her habitual pitch level and at lower or higher level. There was slight improvement for males at lower pitches on forward play of the tapes, but not on backward play. Authors speculated that there might be a reversal for cleft palate speakers since one of their patient had shown improvement at a higher pitch.

Dickson (1962) reported that adult male cleft palate speakers who were most nasal, generally had higher mean fundamental frequency than subjects who were least nasal. However, Flint (1964) failed to find a higher fundamental frequency for adult male and female cleft speaker who were compared to normal subjects matched for age and sex. In fact cleft female had significantly lower mean FO, especially for vowels /i/ and/u/ than had the normal subjects. There was no difference for males. Tarlow and Saxman (1970) found no difference in mean FO of cleft and non cleft children of both sexes.

Phonatory problem in Cleft Palate ;

Individuals with cleft palate and velopharyngeal imcompetancy have traditionally being described in the literature as having an hypernasal voice quality. Since there is oronasal coupling, leads to laryngeal tension, so these individual have voice problem,

McDonald and Baker (1951), Westlake (1953) and Hess (1959) recognised faulty phonation as an important attribute of the speec of people with palatal clefts. Brooks and Shelton (1963) reported that haarseness occured with a prevalance rate of 10%. in a sample of 76 individuals with palatal cleft. Takags et al (1965) reported that voice problem other than hypernasality were found in approximately 0.6% of patients. This figure is lower than that reported in general populations. Bzoch (1964) reported hoarseness and breathiness associated with pharyngeal flaps and speculated that these problems might occur as a response to hypernasal distortions and might represent an effort to mask

hypernasality. Bzoch's hypothesis was supported in part by Curtis (1968) who reported that a hypernasal speaker would have to expand more than the usual vocal effort to attain a given intensity. If such effort were not made, speech would not be loud enough to be easily heard. Reduction in loudness is often a characteristics of people with VPI (McWilliams & Philips, 1979).

Curtis hypothesized that pitch ranges might also be reduced by VPI with the air loss in vocal system, the greatest subglottic pressure possible with maximum effort would produce less output energy than would be the case if velopharyngeal closure were achieve. Since increase in both subglottic pressure and vocal effort are closely related to increase intensity and to pitch elevation. The speaker with VPI might have some what high pitched voice with limitation in the lower part of the pitch range.

Compensatory glottal stop articulation error patterns were cited as probable cause for the frequently found hoarseness of voice in cleft palate populations [(Bzoch (1970), Moore (1971) and Wilson (1972)]. Such compensatory laryngeal articulation functions creates additional strain and tension on the larynx which when continued over time may result in vocal abuse. The persistent abuse of vocal cord may lead to organic change such as vocal modules.

McWilliams et al (1969) investigated 43 children in age range 4-13 years with cleft palate and hoarseness and reported that 32 children had chronic hoarseness. Eighty four percent had positive vocal cord findings. The most common pathological condition was Bilateral vocal cord nodules which occured in 71.9% of the children. Other pathological conditions included

posterior glottal chink, bilateral vocal cord hypertroping, slight anterior edema and inappropriate approximation of vocal cord. Voice of one boy who wore a prosthetic appliance worsened while he underwent therapy. Laryngoscopic study revealed large Bilateral vocal cord nodules. The idea of relationship between fulty valving, hoarseness and speech therapy was reinforced when it was discovered that 16 of the 22 children with vocal cord nodules had speech therapy prior to diagnosis of laryngeal pathology. They concluded from this study that children with cleft palate associated with chronic hoarseness are likely to have some vocal cord pathology particularly bilateral vocal cord nodule. The suggestion was made that cleft palate children who are hoarse or have any other evidence of velopharyngeal valving deficiencies shoud probably not be subjected to the stress of . speech therapy because of the chance that they will compensate laryngeal for velopharyngeal valvlng distress, vocal cord nodules appear to be a danger signal and to provide evidence of a need for further evaluation of the valving mechanism and consideration of secondary management procedure.

McDonald and Baker (1951) mentioned hoarse voice as an abnormal quality constituting of one of several differences frequently found in cleft palate voice. 3zoch (1979) specified that weak and aspirate phonatory voice quality was found in 31.3% of 1000 cleft palate case studies. It appears that aspirate phonation may become habitual to cleft palate children attempting to lessen the perception of distortion of voice from speaking with prolonged velopharyngeal incompetency during early development. The occurance of aspirate voice in cleft palate population could best be prevented by an early treatment plan that confirms

velopharyngeal inadequacy or correct inadequacy when present after primary closure so that normal velopharyngeal function is able to support normal voice development.

Hamlet (1973) using ultra sound investigated the characterist of vocal fold vibration in 11 normal subjects both children and adults in phonation, nasal and non nasal vowels. She found that at equal level of sound intensity, the open quotient of nasalized vowels was comparable to the open quotients of vowels that are non nasalized, but that were produced during loud phonation. In the subject for whom the degree of mouth opening was controlled, the difference between the two modes of speaking was increase. Results indicated that glottal tightness resulted in reduced open quotient. The strong muscular adducting force might contribute to vocal abuse which in turn lead to hoarseness, harshness and vocal nodules secondary to hypernasality. The conclusion appears resonable in view of the clincal evidence. If patients who attempt to compensate hypernasality by tightening both the respiratory muscles and laryngeal muscles to control air flow before it reached crucial velopharyngeal valve.

The whole area of laryngeal involvement in the presence of velopharyngeal incompetency is further complicated by recognition of the 5 to 10 dB drop that can be created in vowel amplitude when oronasal coupling is present (House and Stevens 1956, Fant 1960). cited that some individual with hypernasal speech seem to compensate for their velopharyngeal valving incompetance by using increased vocal effort Curtis (1968). Their data suggested that as velopharyngeal orifice area increases, the effective damping of vocal tract is increased and amplitude of acoustical energy radiated from speech mechanism is reduced. This results in an

increase in vocal effort and potential in laryngeal abuse in individual with velopharyngeal valving deficit. In fact when children are instructed by the clinician to speak more loudly, thy may be unable to do so. Reasons for this inability probably vary. Some children do not speak more loudly because of inhibition and anxiety. Others may not be able to increase loudness because they cannot compensate laryngeal for the energy loss at the faulty velopharyngeal valve.

Soft Voice Syndrome

Because of loss of air through the velopharyngeal port, some cleft patients use reduced loudness as a compensatory strategy. Patients who adopt this voice pattern may have little voice escape, although their velopharyngeal valving mechanism are usually deficiencey In order to increase loudness their subglottic pressure might be higher than normal and there is air loss leading to an increase in nasal emission together with altration in the perception of hyper-nasality. Often accompanying are perhaps as part of the soft voice syndrome is the monotonous voice with little pitch variation. Patients with this problem are often unable to demonstrate pitch variation and do not do so even when they attempt to sing.

Articulation Problems in Cleft Palate :

Articulation disorder of varying degrees of severity occur commonly in patients with cleft palate. In two early descriptive studies Mcwilliams (1953) and Spristerbach (1956) described the articulation of heterogenous groups of cleft palate adults and children. Consonants misarticulated more than 60 percent of the time by the children studied by Spristerbach were /z//s/ /0/ /ts/ /t / /dz/ / / / / and /t /. The consonant most correctly articulated at least 80 percent of the time where /m/ /h/ /n/ / j / and /z /. In the Mcwilliams (1953) study the most frequently misarticulated sounds were /a/ (63%),/z/ (61%) / /(48%) and /ts/ (44%). Only 11% of p sounds and 9% of /b/ sounds were misarticulated.

The /s/ is the speech sound most frequently misarticulated by individuals with cleft palate (Mcwilliams, 1951, 1958) and it is also the sound most likely to be consistent in error. Byrne (1961) reported that their subjects correctly produced only 50% of the /s/ items studied. Fletcher (1978) reported that his subjects misarticulated 46%, 43% and 49% of /s/ sounds studied in the initial, final and medial positions of words respectively. His subjects most frequently misarticulated the blends str - (67%), st - (63%), sk -(63%) and sp - (61%).

Types of Error :

If the misarticulation associated with cleft palate primarily reflects anatomical defects, the articulation disorder should be phonetic in nature. The speaker would be expected to produce the closest possible approximasion of the target sound.

(McWilliam ,1953, 1958) found that of 1814 misarticulated sounds produced by adult cleft palate subjects 1436 were distortions, 335 were omission, and 43 were substitution. Similarly in a study by McDermott (1962) most cleft palate speakers between 8 and 18 years of age produced distortion of /s/. He classified 70% of the /s/ sounds studied as distortion, 23 percent as correctly articulated, 5 percent as omitted and 1% as substituted by other sounds. While nasal distortion accounted for 25% of the articulation errors, distortion accounted for 30%, omission for 29%, substitution for 13%, glottal substitution for 2% and nasal substitution for 1%.

The type of error has been found to vary with palce and manner of articulation and with whether single consoant or blends are being tested Spristerbach (1961) in a study of children between the ages 3 to 17 years found more omissions and distortion and fewer substitutions for blends than for single elements.

Manner of Articulation :

Cleft Palate in association with velopharyngeal incompetance is more likely to interfere with some manner classification than with others. The tendency for nasal consonants to be correctly articulated most frequently followed in order by glides, plosives and fricatives was cited earlier.

Vandemark et al (1979) observed a higher rate of misarticulat of fricatives and affricatives than of plosives. Pitzner and Morris (1966) found that cleft palate individuals with poor velo-pharyngeal closure were more likely to misarticulate /r/ and /l/ than of cleft palate persons with good velopharyngeal function. That conclusion is compatible with van Denmarks (1969) finding

that children with low oral monometer quotients produced liquids correctly less frequently than did individuals with higher quotients.

Place of Articulations :

Counihan (1956) rank ordered sounds from least to most frequency misarticulated as follows: Lip sounds, tongue tip simple sounds, tongue tip complex sounds. Moll (1968) pointed out that the tongue tip complex sounds Include fricatives and affricates. From his review of literature regarding cleft palate speakers, Moll (1968) concluded that within different manner of production categories sound involving lingual contacts tended to be more defective than those involving only the lips. He further concluded that place of articulation is less important in speakers with cleft palate than in manner of articulation.

Voicing :

Individual with clefts appear to misarticulate voiceless sounds more frequently than the voiced cognates (Spristersbach et al 1956), McWilliams (1953, 1958). Sherman et al (1959) found that glottal stops were used more frequently to replace voiceless than voiced consonants. However the percentage of coiced and voiceless sounds misarticulated by the younger subjects in the study by Philips and Harrison (1969) were similar. Spristersbach et al (1961) found that voiced stops and affricates were better articulated than their voiceless counterparts, but that reverse were true for fricatives.

Language Disorder :

Many of earlier studies on the development of communicative skills in children with cleft focused on articulation proficiency and vocabulary size. Later, limited attention was paid to other aspects of like language as a gradual evolution of concern for the total communication process occured.

Szoch (1966, 1959) indicated that more than 50% of the children he studied had delayed linguistic development. Spriestersbach (1958) reported shorter mean length of utterance in cleft palate children than normative group but their structure complexity did not differ. Later Morris (1962) expanded the stud of Spriesterbach (1958) and reported that vocabulary recognition was also reduced.

Nation (1970) used the peabody picture vocabulary test to assess both comprehension and usage in 25 children with clefts 25 of their non cleft sibilings and 25 normal controls. The normal in this study performed better than the sibilings of the cleft palate children and sibilings better than the cleft subject Nation concluded that cleftness affects vocabulory development.

Shanes et al (1966, 1971, 1979) studied 75 cleft and 75 unmatched non cleft children between the age of 1.5 and 5.5 years. They used a standardised interview system composed of 135 stimulus episode designed according to skinner's 1957 model of verbal behaviour (Echoic) Mand, tact, intra verbal and autoclatic). The study showed that children with clefts master the forms of verbal behaviour described by Skinner on a slower pace than do their peers, but that they quickly overcame these lags and are similar to others by age of 4 years 3 months. However, inspite

of this, the children with clefts maintained a higher grammatical error rate at all age levels under investigation, a finding consistent with others studies indicative of slight language impairment.

McWilliam (1968) administered illonis test of psycholinguist abilities (ITPA) to 136 children with cleft lip and palate and reported particular weakness in vocal expression,gestural output and visual memory.

Pannbacker (1975) reported that cleft subject were inferior, to normals on mean length of sentence, number of words in the longest response, mean length of the five longest responses, number of differeent word used and the intelligibility of speech.

Horn (1972) undertook a transformational analysis of the language output of 3 left palate children between 4-6 years of age. She described the output of the cleft children as immature. The rule that children used more clearly indicative of rules applied in the early stages of language acquisition. The differences were still present at age 5, but were less marked. Horn speculated following factors to explain the language depress found in these children - Hearing problems, poor mother child relationships, inhibition, parental over protection and inferior speech modeling.

Pitch and Intensity Perturbation ;

Cycle to cycle variation in frequency is called pitch perturbation and cycle to cycle variation in amplitude is called as inteasity perturbation. Presence of small perturbation on irregularity of glottal vibration in normal voice has long been known (Moore and Van Leden 1958)von Leden, Moore and Timeke 1960 A periodic laryngeal vibratary pattern have been related to the abnormal vocal production by various investigators (Car Hart, 1938, 1941, Bowler 1964). Relatively few attempt have been made to note the pertubbation in fundamental frequency and intensity, although such a measure may have valve in describing the stabilii of laryngeal control (Liberman, 1963)

Frequency pertubbation commonly called jitter, is the variability of the fundamental frequency or resiprocal of fundamental period. Jitter measurement are concerned with short term variation. That is Jitter is a measurement of how much a given period differs from the period that Immediately follows it. Thus the cycle to cycle variation in periods that occurs when an individual is attempting to sustain phonation at a constant frequency has been termed as jitter. Jitter is than a measurement of frequency variability not accounted for by voluntary change in FO. If the phonatory system were an ideal and perfectly stable mechanism there would be absolutely no difference in fundamental period except when a speaker purposefully changed pitch. To the extent that jitter is not zero. "Perturbation is an acoustic correlates of erratic vibratory pattern (Beckett, 1969) that result from diminished control over the phonatory system (Serenson, Horii and Leonard, 1980)". Simon (1927) concluded the phonatory system is not a perfect machine and

every speakers vibratory system is erratic to some extent. But abnormal larynx would produce a more erratic voice than healthy one. Thereis by now a considerable body of literature that asserts the usefulness of frequency perturbation measures in evaluation of laryngeal and vocal pathology (Kitajina, Tanabet, Isshiki, 1975; Davis, 1976; Horii, 1979; Liberman, 1961, 1963; Hecker and Kraul, 1971; Klingholz and Martin, 1983; Bull, McDonald and Johns, 1984).

While considering the neurophysiological significance of jitter, Heiberger and Horii (1981) state that physiological interpretation of jitter in sustained phonation would probably include physical,structural and myoneurological variation during phonation. High speed laryngoscopic motion picture revealed the laryngeal structure are not totally symmetric. Accumulation of mucous on the surface of folds, turbulent air flow at the glottis laryngeal servomechanism through articular myetitic and mucosal reflex system and laryngeal muscle tone contribute for laryngeal perturbation. Baer, 1980 explains vocal jitter as inherent to the method of muscle excitation based on the neuromuscular model of fundamental frequency and muscle physiology. He claims neuromuscular activities as the major contributor for the occuran< of perturbation.

Liberman (1963) found that pitch perturbation in normal voices never exceed 0.5 m.sec in magnitude in the stady state portion of long sustained vowels. Similar variation in funda-mental periodicity of acoustic wave form have been measured by Fairbanks (1940), Reiberg (1961) and Saito et al (1958). The results reported by Liberman (1963) were confirmed by Iwata and Von Leden (1970) and the 95% confidence limits of pitch perturbati in normal subjects ranged from - 0.19 m.sec. to 0.2.m.sec.

von Leden et al (1960) report that in frame by frame analysis of ultra high speed motion pictures the commonest observation in pathological conditions is a strong tendency for frequent and rapid changes in the regulatory of vibratary patter

Iwata (1972) tested the voice of 20 normal subjects and 27 patients with various laryngeal disease for pitch perturbatio and opined that the correlograms were useful in differentiating normal and abnormal voice and different types within abnormal voice.

'Zajac and Linville (1989) studied 10 cleft palate children with VPI in terms of frequency perturbation and intensity perturbation with percived nasality and hoarseness. Their findings suggested that voice perturbation of the children with VPI were correlated moderately with percived nasality and hoarseness. Additionally, the results suggested that voice perturbatic (atleast jitter) of those children were significantly greater than those of children without VPI. The positive relationship between jitter and percived nasality provides additional evidence for a link between laryngeal and velopharyngeal events.

Some amount of voice perturbation is normal and may reflect random aerodynamic and muscular events (Titze, Horii and Scherer, 1987). The increased jitter level of children with VPI suggest that laryngeal aerodynamic and/orneuramuscular processes may be altered as a result of oronasal coupling. When velopharyngeal port is open during vowel production, airflow has two alternative path to follow (analogous to a parallel electric circuit). In a parallel circuit, total current flow is greater than that in either branch. A similar situation in the vocal tract would result in chages in glottal volume velocity (flow rate) and

transglottal pressure change (Leder and Lerman, 1985). However, this situation assumes conditions of constant glottal resistence and respiratory effort without compensatory responses

Individuals with VPI may attempt to regulate actively vocal tract resistence as a compensation (warren, 1986). Increased glottal resistence during vowel production would decrease flow rate and facilitate regulation of subglottal pressure required to sustain phonation. Additionally compensatory change in chest wall dynamics may occur either to increase or to decrease sub-glottal pressure as needed. Therefore, attempts to regulate respiratory and laryngeal aerodynamics and neuromuscular processes when inappropriate oronasal soupling exists may contribute to increased vocal perturbation. In their study intensity level among subjects were not controlled. Research has indicated that children may produce greater intensity levels than adults when instructed to talk at a comfortable loudness level (Stathopoulos, 1986). Differences in intensity level among children may have a affected jitter and shimmer. Zajac and Linville (1988) reported increased jitter for adult speakers when phonating at greater than normal loudness level. However Glaze, 3less and Milenkovic (1988) reported that acoustically derived voice perturbation of children decreased with increased loudness. This finding may attributable to difference in laryngeal anatomy between children and adult.

Perturbation Factor :

Normal vibratory patterns of the vocal folds are disrupted in the presence of laryngeal pathology and in particular there is a greatly increased tendency for rapid and frequent lapses

of vibratory irregularity (Liberman, 1961, 1963). He reasoned that frequency perturbation reflect i) change in glottal periodicity, ii) altration of glottal wave form, iii) variation of vocal tract configuration that result in phase shifts of acoustic wave. Liberman (1963) proposed an index called perturbation factor defined as the integral of frequency distrlbuti< of Δt> o.5 m.sec. Though some validation of these conclusion has been done (Iwata and Von Leden, 1970) more will be needed before the perturbation factor can be widely used.

Directional Perturbation Factor ;

It was defined as the percentage of the total number of differences for which there is a change in algebric sign. Directional perturbation factor put forth by Hecker and Kruel, 1971 is concerned with the number of times that the frequency change shifts directions. Directional perturbation factor takes into account the algebric sign rather than the magnitude of the difference between adjacent glottal pulse interval.

The magnitude of frequency perturbation shows considerable correlation with mean fundamental frequency. A number of researcher (Liberman, 1963, Beckett,1969; Koike, 1973, Horii, 1979, 1980) have noted longer cycle to cycle differences are associated with longer fundamental periods. Analysis by Horii (1979) and by Hollien, Michel and Doherty (1973) tend to demonstrate that there is no way to compensate exactly for the effects of mean F0 and thereby achieve on uninfluenced jitter index. The best compromise is to generate a ratio of some form of mean perturbation to mean period.

Jitter Ratio :

The simplest form of F0 adjusted perturbation index is the mean perturbation divided by mean wave form duration (Hor±i,1979 by definition

$$\text{Jitter ratio} = \frac{\frac{1}{n-1}\left[\sum_{i=1}^{n-1} |P_i - P_{i+1}|\right]}{\frac{1}{n}\sum_{i=1}^{n} P_i} \times 1000$$

where Pi = Period of ith cycle in miliseconds

N - Number of period in the sample

Numerator is the sum of absolute values of the differences between successive periods divided by the number of difference measured.

Period Variability Index :

Was put forth by Deal and Emanuel (1975). It requires the computation of a co-efficient of variance (CV) defined as

$$CV = \frac{\frac{1}{n}\sum |(P_i - \bar{P})^2|}{\bar{P}^2}$$

Where Pi = Period of ith cycle

P = Mean period

The term (Pi - P) signifies the difference between the itch period and mean period. The co-efficient of variation is the mean of the square of deviation from the mean divided by the square of the mean. PVI is the CV times 1000.

Relative Average Perturbation:

Relative Average Perturbation (RAP) was put forth by Koike (1973) also called as frequency perturbation quotient (Takashi

and Koike, 1975) attempts to mitigate the difficulties by using a form of straight line averaging that greatly reduces the effect of relatively slow change in FO.

RAP function is defined as

$$RAP = \frac{\frac{1}{n-2}\sum_{i=2}^{n-1}\frac{P_{i-1}+P_i+P_{i+1}}{3} - P_i}{\frac{1}{n}\sum_{i=1}^{n} P_i}$$

The numarator called average absolute perturbation is recognizable as the average difference between actual period and three points estimate. The denominator is the mean period which is included to compensate for the change in mean absolute jitter that occurs with change in FO.

Deviation from Linear Treads (DLT):

Founded on the same rationale as RAP has recently been proposed by Ludlow, Coulter and Gentges (1983). Deviation from linear trends which is defined as the difference between a period and the average of the periods two cycles away from it in each direction are considered. The formal definition is ?

$$DLT_i = \frac{Pi-2 + Pi+2}{2} - Pi$$

The measure of perturbation the mean DLT

$$DLT = \frac{\sum_{i=a}^{b} |DLT_i|}{b-a}$$

Since DLT evaluates alternate cycle only Pi - 2, Pi, Pi +2, it might not detect perturbation caused by a short cycle regularl

alternating with a long one as occurs in pulse register (Cavall Baken, Shaiman, 1984; Hollien, Girard and Colman, 1977).

Amplitude Perturbation :

Measure of amplitude perturbation generally called shimmer are analogous to those of fundamental frequency perturbation. Like frequency perturbation scores measurement of shimmer are used to quantify short term instability of vocal signal. Shimmer is as important as Jitter in its contribution to the perception of hoarseness (Wendahl, 1965).

Cycle to cycle variation in amplitude is defined as shimmer in any voice is dependent atleast upon the modal frequency level the total frequency range and the SPL relative to each individual voice (Michel and wendahl, 1971)

Kitajina and Gould (1976) studied the vocal shimmer. During sustained phonation in normal subjects and subjects with laryngec polyp and found the valve of vocal shimmer to range from 0.04 Db to 0.21 Bb in normals and from 0.08 Db to 3.23 Db in the case of vocal polyp. Although some overlap between the two groups was observed they noted that the measured value may be useful index in screening for laryngeal disorder.

Directional Perturbation Factor :

Originated by Hecker and Kruel (1971) as a measure of period perturbation, the directional perturbation factor can also be applied to amplitude variation. The measure tallies the number of times that the amplitude change between two successive wave shifts directions. DPF for amplitude takes into account the algebric signs rather than magnitude of difference between adjacent

glottal pulse interval. It is defined as the percentage of tot number of differences for which there is a change in algebric sign.

Amplitude Variability Index (AVI) :

AVI of Deal and Emanuel (1978) resembles their period varied bility index.It represents the average degree of variation from mean peak amplitude of the sample.

AVT based on a co-efficient variation (CV) that applied to amplitude. This is defined as ?

$$CV = \frac{\frac{1}{n}\sum_{i=1}^{n}(x_i - \bar{x})^2}{\bar{x}^2}$$

Where n = No. of peaks measured

xi = Individual amplitude values

x = Mean peak amplitude

AVI is then calculated

$$AVI = \log_{10} (CV \times 1000)$$

Shimmer in dB :

Since the decible scale is based on a ratio of amplitude it can be used to quantify shimmer. The ratio should be that of two continuous cycles (Ai and Ai + 1) for which amplitude difference in Db is

$$\text{dB Shimmer} = \underline{20 \log_{10} Ai/Ai + 1}$$

The average Shimmer for a sample of N cycle is the ;

$$\text{Db Shimmer} = \frac{\sum_{i=1}^{n-1} 20 \log Ai/Ai+1}{n-1}$$

This approach to the quantification of Shimmer has the distinct advantage of freeing the measurement from the absolute amplitude.

Amplitude Perturbation Quotient :

Takahashi and Koike (1975) and Koike, Takahashi and Calcatten (1977) have suggested amplitude perturbation quotient (APQ). The function uses a 11 point average for smoothing and defined as

$$\text{APQ} = \frac{\frac{1}{n-10} \sum_{i=6}^{n-5} \frac{Ai-5 + Ai-4 + \cdots + Ai + \cdots + Ai+5}{11} - Ai}{\frac{1}{n} \sum_{i=1}^{n} Ai}$$

where Ai = Peak amplitude of each wave

n = Number of wave measured

Davis (1976, 1979, 1981) has evaluated the effect of window size (number of wave in running average) and has found that the APQ function is optimalized at 5.

Long Term Average Spectrum :

There are a number of methods by which speech can be analyses spectrally, one such analysis procedure takes atime average of the sound-pressure level per cycle across frequency. This measurement is commonly referred to as the Long-Term Average Spectrum of speed (LTAS) (Formby and Monsen, 1982). The measurement of the long term average speech spectrum is made by passing the speech energy through a series of contigenous band pass filters and interpreting the energy at the output of each filter. These average values are then plotted to arrive at the visual representation, a smoothed plot by the envelope of the power spectrum of the speech sample (Formby and Monsen, 1982).

LTAS has been used for studies of the human voice source. The speech signal represents the product of the seund source and the vocal tract transfer functions. The vocal tract transfer function differs for different sound segments, but in the averaging process, the short term variations due to phonetic structure will be averaged out and the resulting spectrum can be used to obtain information on the sound source (Lofquist and Manderson, 1987).

Lofquist and Manderson reported that if the analysis is restricted to voice sounds, the sound source is the vibrating glottis. The analysis can be made of readings of a standard text in order to further minimise variation due to phonetic structures.

The earliest measurements of Long Term Average spectrums was reported by Bell Telephone Laboratories about 60 years ago (Crandall and Mackenzie, 1922). Ever since then, speech spectra have been obtained by different investigators for different purposes

Recently LTAS has been obtained to study the disorders of speech production (Frokjaei - Jensen and Prytz, 1976, wendler et 1980; Weinberg et al, 1980; Hammer berg et al, 1984; Hartman and Von cramon, 1984; Dejonckere, 1986).

Lofquist and Manderson (1987) states that the study of long term average spectra is potentially useful in the clinics, their possibilities and limitations are yet to be fully understood in relation to other acoustic measures of voice functions.

The speech power is greatest between 100 to 600 Hz, where the energies of the fundamental frequency of the voice and the first formant overlaps. It drops off with increasing frequency above about 600 Hz such that at 10,000 Hz, the level is approximately 50 dB below the peak levels measured at lower frequencies (Denes and pinson, 1963).

In normal voice, the amplitude of source spectrum partially decreases with 12 dB/octave approximately, where there is complet closure of glottis (Frietzell, Hallen and Sundberg, 1974). This spectra for group speech are generally comparable both within ant across language (Fant, 1973). However the general shape of the spectra can be altered depending upon the experimental variables used in given studies.

Among the most salient variables are age (Niemoelleu et al 1974) sex of the speaker (Benson and Hirsh, 1953; Niemoller et al 1974), the analysis band widths (Stevens et al, 1947; Frokjaer - Jensen and Prytz, 1976) and vocal effort (Brondt et al, 1969).

Byrne (1977) studied the spectral differences between males and females speech samples. According to him, the main difference is that the region of 0.1 KHz and 0.125 KHz, where as female

level is much lower, although it could not be measured precisely in this study. This is explicable by the fact that this frequenc region corresponds to the fundamental frequency of male voices.

Tarnoczy (1956) reported that the LTAS measurements highly dependent upon the personal characteristics of talkee such as voc effort, pitch, timbre, articulation and speech of uttarances.

Different investigators have employed different methodologie for obtaining LTAS.

(1) weinberg et al (1980) obtained long term average spectrum using an FFT computing spectrum analyzer. The analyser was set in cumulative mode for 64 frames with a frequency range of 0 to 10,000 Hz and a time window of 40ms. the quantized speech signal is weighted by a hanning function, Fourier transformed and stored for cummulation of 64 frames. Each frame represented FFT results for a 40-ms speech segment. An amplitude spectrum with A - weighting plotted at the end of cummulation. Thus the amplitude spectrum represented an average long time spectrum derived from a total of 64 "sections" made from an oral reading by each subject.

(2) Kitzing (1986) obtained Long Time Average Spectrums by means of a B & K signal analyzer 2033, analysing 400 frequency lines in the choosen range, using linear average over 128 triggered spectra by flat weightings. The analysis were accomplished in two series with different base bond frequency rang viz 0-5 KHz and 0-2 KHz, respectively. In order to avoid nosie fr consonant articulation, unvoiced parts of the signal were elimi-nated by gate, controlled either by EGG signal or by a low pass filter device.

(3) Wendler et al (1984) analysed the tape recordings of on going speech by means of a real time analyzer, using 25 1/3 octave filters in the area of 64 Hz to 12.5 KHz in combination, with an average NTA 512, one analysis was carried out with the unmanipulate continous signals, another with the voice less consonants elimination As there were no significant differences with regard to these two variants, they generally based all further explanation on the data from the continous signals.

(4) Lofquist and Manderson (1987) digitalized the speech signal at a rate of 20 Hz and analysed in frames of 12-ms duration using FFT analysis. Pauses voiceless segments were excluded from the analysis.

Several different measurements have been may by different investigators on long term spectrum, although, there are no generally agreed upon principles for making these measurements (Lofquist and Manderson, 1987).

Fronkjser - Jensen and prtytz (1976) used the ratio of energy below and above 1 KHZ and named it as α parameter. According to them since the amplitude above 1 KHz is normalized relative the amplitude below 1 Khz, is independent of the microphone distance, amplitude level etc.

Hammarberg et al (1984) measured the level of the fundamental, the peak amplitude in frequency bond 400-60 Hz, the spectral level at 1.5 and 5 KHz, respectively, and the peak amplitude in the frequency band 5-10 KHz. They there used the difference between the peak level in the 400-600 Hz and other levels as a measures of spectra tilt.

There is no literature available on LTAS in cleft palate and lip individuals. The proposed study aims to study the following parameters ?

1. α - ratio (alpha) : Energy between 0*1 KHz to 1-8 KHz

$$\frac{0 - 1 \text{ KHz}}{1 - 8 \text{ KHz}}$$

2. β - ratio (beta) : Energy between 0-2 KHz to 2-8 KHz

$$\frac{0 - 2 \text{ KHz}}{2 - 8 \text{ KHz}}$$

3. γ - ratio (Gama) ; Energy between 0-1 KHz to 5-8 KHz

$$\frac{0 - 1 \text{ KHz}}{5 - 8 \text{ KHz}}$$

Routinely used procedure at the department of speech sciences All India Institute of Speech and Hearing, Mysore is as follows ;

The speech sample (voiced) is recorded using a philips deck. The speech samples there fed into the computer Pc-AT 386, based upon 80386 CPV and 80387 NDP with 12 bit ADC and DAC It is having anti-analysing filters at 7500 Hz. The speech was sampled at 16000 Hz. For carrying out spectral analysis the programme "LTAS" with auto correlation was used. This programme was develop by voice and speech system. Bangalore. The spectral analysis were carried out for the frequency range 0 - 8 KHz with a resolution of 10 and block duration of 20 (the block duration depends upon the F0 of the subjects).

The spectral analysis data thus obtained contained the following ; -

1. A graphical display of the spectral pattern in the frequency range of 0 - 8 KHz.

2. A data of the energies of all the different points which were analysed by the computer.

3. Alpha ratio

4. Energy above 1 KHz (1 KHz - 8 KHz)

5. Energy below 1 KHz (0 - 1 KHz)

Further information regarding the energy concentration in the frequency band 5 - 8 KHz was obtained through the computer - programme by summing up the energies of all the different points in that frequency band and dividing it by the number of all the points in that frequency band.

M E T H O D O L O G Y

The aim of the study was to investigate the difference in the laryngeal control mechanism of cleft palate and lip individuals. TO evaluate the efficiency of laryngeal control mechanism the following measurements were done ;

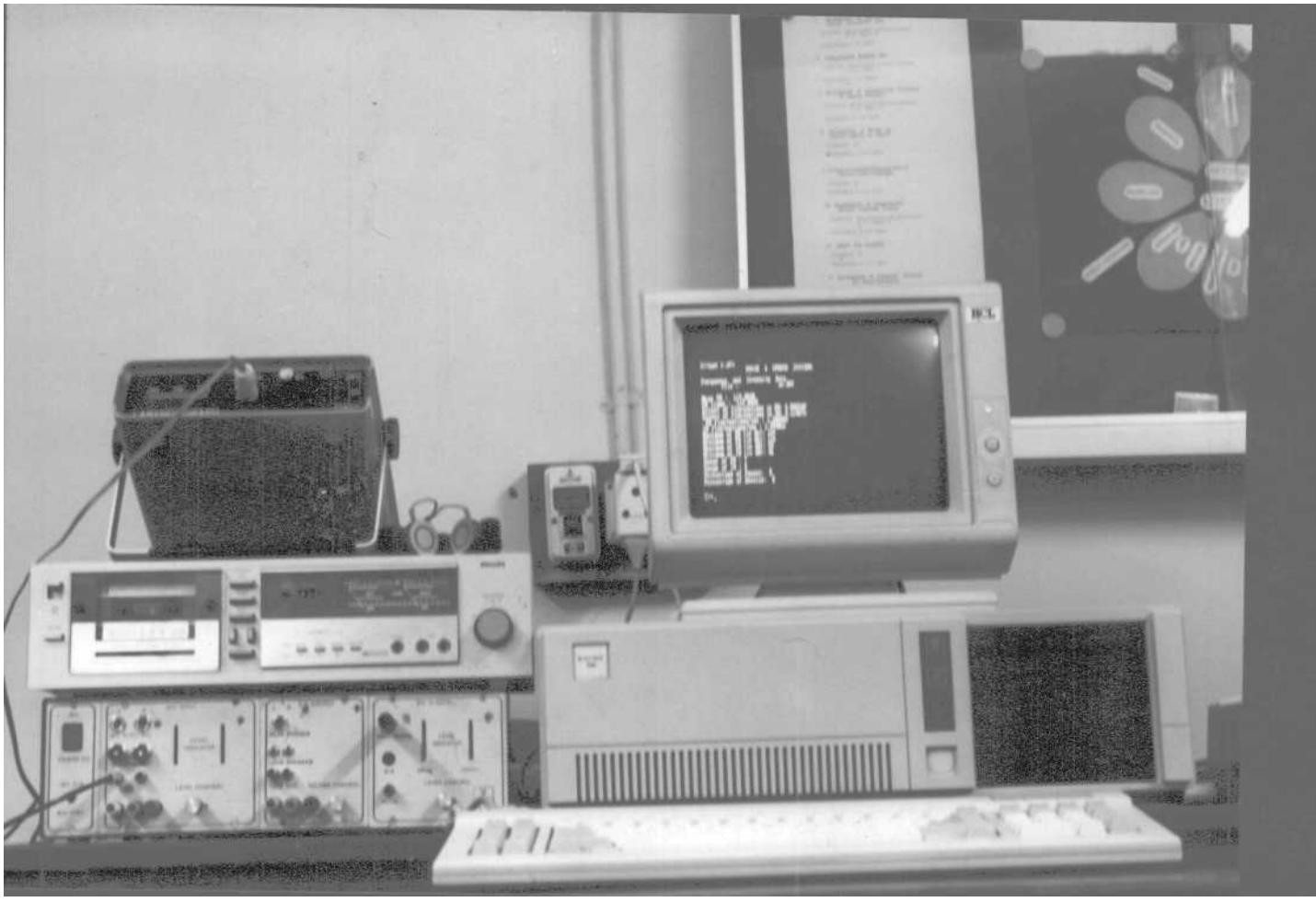
1. Pitch perturbation measurements
2. Amplitude perturbation measurements
3. L.T.A.S. measurements

Subjects :

Thirteen persons having cleft palate and lip with no history of chronic laryngitis or pharyngitis and with no known history of voice problem served as a experimental group. Thirteen normals having normal oral structure and with no known history of voice problem served as central group. These two groups were matched for age, sex and intelligence. Details are shown in Table 1.

	Male		Female	
Subjects	Age range	No.of Subjects	Age range	No. of Subjects
1. unrepaired Cleft Palate and Lip	19-25Yrs	4	13-18 Yrs	3
2. Repaired Cleft Palate and Lip	8 - 19YrS	2	8-24 Yrs	4
Normals	8 - 25Yrs	6	8-24 Yrs	7

Table 1 shows sex, age range, number of subjects in unrepaired and repaired cleft palate and lip and normals.



PHOTOGRAPH SHOWING THE EXPERIMENTAL SET UP

Speech Sample :

1. Phonation of vowels /a/ /i/ and/u/ for 5 seconds.
2. Heading of a Kannada voiced passage having 125 syllables.

Instruments :

1. Kay Laryngograph (Kay NO.80138)
2. Philips F6121 Stereo Audio recording deck with AKG D222 cordoid dynamic microphone.
3. PC - AT computer based on Intel 80386 microprocessor and Intel 80387 N.D.P.
4. V.S-S. - 12 bit ADC and DAC Data I/O cord with speech interface unit.
5. V.S.S. - software programme to extract FO, to calculate pitch and intensity perturbation and to calculate LTAS.

Procedure :

1. Measurement of Pitch and amplitude perturbations :

The subjects were seated comfortably and the electrodes of the laryngograph were placed on the thyroid alae. The subjects were instructed to phonate vowels /a/ /i/ and /u/ three times each for 5 seconds at their habitual pitch and comfortable loudness. Wave form of EGG were digitized into computer using 12 bit ADC (VSS - data I/O cord) at the rate of 8000Hz. The vowels were segmented and middle 1 sec. segment was taken for further analysis. Successive pitch periods were extracted using the EGG wave forms wit the help of a digital computer. The digitally stored EGG wave form were differentiated and interpolated to 32 KHz sampling rate. Using peak picking method, the exact pitch period were calculated. The obtained pitch periods were used to calculate the pitch and

intensity perturbation using following formulae ;

Pitch Perturbation : Defined as cycle to cycle variation in frequency.

1. Relative Average Perturbation for frequency

RAP for frequency is defined as

$$RAP = \frac{\frac{1}{n-2}\sum_{i=2}^{n-1}\left|\frac{P_{i-1}+P_i+P_{i+1}}{3}-P_i\right|}{\frac{1}{n}\sum_{i=1}^{n}P_i}$$

Where Pi = Period of ith cycles

n = Number of periods.

2. Directional Perturbation factor for frequency : It was defined as the percentage of total number of differences for which there is a change in algebric sign. DPF takes into account the algebric sign rather than the magnitude of the differences between adjacent glottal pulse intervals.

Amplitude Perturbation : Defined as cycle to cycle variation in amplitude.

3. Relative Average Perturbation for Intensity

RAP for intensity is defined as ?

$$RAP = \frac{\frac{1}{n-10}\sum_{i=6}^{n-5}\left|\frac{A_{i-5}+A_{i-4}+\cdots+A_i+\cdots+A_{i+5}}{11}-A_i\right|}{\frac{1}{n}\sum_{i=1}^{n}A_i}$$

where Ai = Pitch amplitude of each wave

n = Number of waves measured

4. Directional Perturbation factor for intensity: It was defined as the percentage of the total number of differences for which there is a change in algebric sign. DBF for intensity takes

into account the number of times that the amplitude change between two successive waves shifts directions.

2. LTAS Measurements :

For the purpose of obtaining L.T.A.S. subjects were seated in the comfortable position and asked to read "Kannada voiced Passage" at comfortable loudness. Their readings were recorded on a Hi-Fi magnetic cassette tape using philips audio tape recording deck (F6121) with AKG cordoide dynamic microphone (4222). Distance between the speaker and microphone was kept constant i.e. 6-9 inches

The tape recorded speech were digitized using 12 bit VSS data I/O cord at 16000 sampling frequency and these digitized data were stored in computer memory. These digitized data were subjected to 1024 point short term FFT analysis for every 10 miliseconds using a digital computer. The window size for FF analysis was 30 mili-seconds and the resolution was 10 miliseconds. The FFT analysis was done for 40 seconds of speech sample, obtaining 4000 spectrums. These spectrums were added and averaged to obtain a LTAS.Brom the obtained term average spectrum, the following measurements were done ;

1. Alpha ratio = 0-1 KHz / 1-8 KHz
2. Beta Ratio = 0-2 KHz / 2-8 KHz
3. Gama Ratio = 0-1 KHz / 5-8 KHz

Appropriate descriptive and non parametric statistical procedure were administered to compare the different measurements of pitch and intensity perturbation and LTAS obtained from individuals having cleft palate and lip from control group.

R E S U L T S

The aim of the study was to investigate the differences in the laryngeal control mechanism of cleft palate and lip individuals. TO evaluate the efficiency of laryngeal control mechanism, the following parameters were measured;

1. Pitch Perturbation :

a) Relative Average Perturbation for frequency

b) Directional Perturbation factor for frequency

2. Intensity Perturbation :

a) Relative Average Perturbation for Intensity

b) Directional Perturbation factor for Intensity

3. Long Term Average spectrum :

a) Alpha ratio

b) Beta ratio

c) Gama ratio

1. Relative Average Perturbation for Frequency :

Table 2 shows mean, standard deviation and range of RAP FO for vowels /a/ /i/ and /u/ in normals and in cleft palate and lip individual s and significance of mean difference between them.

Group	Vowels	Mean	SD	Range Min	Range Max
Normals	a	.0609	.0516	.00457	.2029
	i	.0479	.0594	.00423	.2345
	u	.0698	.0746	.00433	.2546
Cleft Palate and lips	a	.06393	.0837	.0097	.2488
	i	.05552	.08019	.0078	.2467
	u	.08934	.1154	.0096	.3871

Significance of Mean differences between Cleft Palate and and Normals

Vowels	Z	P
a	-.5940	0.55
i	-.2446	0.80
u	.3844	0.70

From the above table it may be observed that RAP for F0 values, for vowels /a/ /i/ and /u/ in the cleft palate and lip subjects were higher than normals. Cleft Palate and lip group also showed greater range and variability than normals.

The Mann Whitney "U" test was applied to test the significance of differenced Analysis revealed no significance difference between mean values of RAP for F0 for vowel /a/ /i/ and /u/ in cleft palate and lip and in normal subjects. The Z values and P values are shown in Table 2.

Thus the hypothesis l(a) stating that "there will be signficant difference between cleft palate and lip individuals and normals for vowels /a/ /i/ and /u/ in terms of Relative Average Perturbation for frequency was rejected.

2. <u>Relative Average Perturbation for Intensity</u> :

Table 3 shows mean, standard deviation and range of RAP for intensity for vowels /a/ /i/ and /u/ in normals and in cleft palate and lip individuals and significant of mean difference between them.

Group	Vowels	Mean	SD	Range Min	Range Max
Normals	a	.02918	.01105	.01443	.05263
	1	.02428	.00998	.009	.0369
	u	.0281	.01374	.00797	.05127
Cleft Palate & lip	a	.0461	.01653	.0239	.07983
	i	.0372	.01798	.01183	.0713
	a	.03483	.01346	.01757	.0578

Significance of Mean Differences between Cleft Palate and and normals

Vowels	Z	P
a	-2.6270	.008
i	-1.9917	.046
u	-1.2229	.221

From the above table it may be observed that RAP for intensity values, for vowels /a/ /i/ and/u/ in the cleft palate and lip subjects were higher than normals. Cleft palate and lip subjects also showed greater range and variability than normals.

The Mann Whitney "U" test was applied to test the significance of difference. Analysis revealed no significance of differences between the mean values of RAP for intensity for vowel /a/ /i/ and /u/ in cleft palate and lip and in normal subjects. The Z values and P values are shown in Table 3.

Thus the hypothesis l(b) stating that "there will be significar difference between cleft palate and lip individuals and normals for vowels /a/ /i/ and /u/ in terms 66 relative average perturbati for intensity was rejected.

3. <u>Directional Perturbation Factor for Frequency</u> :

Table 4 shows mean, standard deviation and range of directions perturbation for F0 for vowels /a/ /i/ and /u/ in normals and cleft palate and lip individuals and significant of mean difference betwe them.

Group	Vowels	Mean	SD	Range Min	Range Max
Normals	a	66.4647	4.9413	55.007	73,753
	i	64.4552	6.7666	50.327	71.094
	a	64.2924	4.2478	48.248	70.323
Cleft Palate & lip	a	68.4974	6.8571	59.708	86.283
	i	64.9704	5.0124	51.919	69.502
		65.7396	6.7439	46.748	72.983

Significance of mean difference between Cleft Palate and lip and normals

Vowels	Z	P
a	.5940	.5525
1	-.7338	.4631
u	.5940	.5525

From the above Table it may be observed that DPF for F0 values, for vowels /a/ /i/ and /u/ in the cleft palate and lip subjects were higher than normals. Cleft Palate and lip subjects also showed greater range and variability than normals.

The Mann Whitney "U" test was applied to test the significance of difference. Analysis revealed no significance of difference between the mean values of DPF for F0 for vowels /a/ /i/ and /u/ in cleft palate and lip and in normal subjects. The Z values and P values are shown in the Table 4.

Thus the hypothesis i(c) stating that " There will be significar difference between cleft palate and lip individuals and normals for vowels /a/ /i/ and /u/ in terms of directional perturbation for frequency was rejected.

4. Directional Perturbution factor for Intensity !

Table 5 shows mean, standard deviation and range of Directional perturbation factor for intensity for vowels /a/ /i/ and /u/ in normal and in cleft palate and lip individuals and significant of mean difference between them.

Group	Vowels	Mean	SD	Range Min	Range Max
Normals	a	64.8189	5.1733	59.8433	78.8732
	i	64.3622	3.2663	60.8460	70.7644
	u	64.0559	3.8941	60.6373	66.3469
Cleft Palate & lip	a	67.1109	3.0318	59.9222	71.6511
	i	65.0934	1.7276	62.8089	68.8818
	u	65.5913	2.6915	59.5736	70.1224

Significance of Mean difference between Cleft Palate and Lip and Normals

Vowels	Z	P
a	-1.4326	.1520
i	-0.8037	.4216
u	-1.6423	.1005

From the above table, it may be observed that DPF for intensity values, for vowels /a/ /i/ and/u/ in the cleft palate and lip subjects were higher than normals.

The Mann Whitney "U" test was applied to test the significance of difference. Analysis revealed no significance of difference between the mean value of DPF for intensity for vowels /a/ /i/ and /u/ in cleft palate and lip and in normal subjects. The Z values and P values are shown in the Table 3.

Thus the hypothesis i(d) stating that "There will be significant difference between cleft palate and lip individuals and normals for vowels /a/ /i/ and /u/ in terms of directional perturbation factor for intensity was rejected.

5. Alpha Ratio :

Table 6 shows mean, standard deviation and range of Alpha ratio for kannada voiced passage in normals and in cleft palate and lip subjects and significant of mean difference between them.

Group	Mean	SD	Range Min	Range Max	Significance of Mean difference between Cleft Palate and lip and normals
Normals	3.3635	1.4668	1.9387	6.6061	Z P
Cleft Palate & lip	4.0009	1.3333	2.6231	7.0523	.9435 .3495

From the above table it may be observed that Alpha values for LTAS for Kannada voiced passage in the cleft palate and lip subject: were higher than normals.

The Mann Whitney "u" test was applied to test the significance of difference. Analysis revealed no significance of difference between the mean values of Alpha ratio in cleft palate and lip and in normals. The Z values and P values are shown in the Table.

Thus the hypothesis 2 stating that "There will be significant difference between cleft palate and lip individuals and normals in terms of Alpha ratio for LTAS was rejected.

6. <u>Beta Ratio</u> :

Table 7 shows mean, standard deviation and range of Beta rati for LTAS for Kannada voiced passage in normals and in cleft palate lip subjects and significant of mean difference between them.

Group	Mean	SD	Range Min	Range Max	Significance of Mean difference between Cleft Palate and lip and normals
Normals	2.8124	1.0145	1.6749	4.5316	
Cleft Palate &	2.9724	0.8297	1.9900	4.7492	Z P
					.2446 .8068

From the above table it may be observed that Beta values for LTAS for Kannada voiced passage in the cleft palate and lip subjec were higher than normals.

The Mann Whitney "U" test was applied to test the significanc of difference. Analysis revealed no significance of difference between the mean values of Beta ratio in cleft palate and lip and in nommal subjects. The Z values and P values are shown in the Table 7.

Thus the hypothesis 2 stating that "There will be significant difference between cleft palate and lip individuals and normals in terms of Beta ratio for LTAS was rejected.

7. <u>Gama Ratio</u> :

Table 8 shows mean, standard deviation and range of Gama rati for LTAS for Kannada voiced passage in normals and in cleft palate and lip subjects and significant of mean difference between them.

Group	Mean	SD	Range Min	Range Max	Significance of Mean difference between Cleft Palate and Lip and Normals Z	Significance of Mean difference between Cleft Palate and Lip and Normals P
Normals	5.3842	2.6130	2.8213	10.4689		
Cleft Palate & lip	5.6720	1.9474	2.9179	9.8069	.3145	.7532

From the above table it may be observed that Gama values for LTAS for Kannada voiced passage in the cleft palate and lip subjec were higher than normals.

The Mann Whitney "U" test was applied to test the significance of difference. Analysis revealed no significance of difference between the mean values of Gama ratio in cleft palate and lip and in nonmal subjects. The Z values and P values are shown in Table 8

Thus the hypothesis 2 stating that "There will be significant difference between cleft palate and lip individuals and normals intterms of Gama ratio for LTAS was rejected.

DISCUSSION

It is well known that some amount of voice perturbation is normal and may reflect random aerodynamic and neuro-muscular events (Titze et al, 1987). The increased amount of voice perturbation in the voice of the individuals suggest that laryngeal aerodynamics and/or neuro-muscular process is altered. In Cleft Palate cases laryngeal dynamics is altered. That is Cleft Palate cases the velopharyngeal port is open during vowel production. As a result of oronasal coupling and the airflow has to alternative paths to follow (analogous to parallel electric circuits). In the parallel circuit the total current flown is greater than the current flown in either of the branches (i.e. additive). A similar situation in vocal tract would result in changes in glottal volume velocity (flow rate) and transglottal pressure changes as suggested by Leder and Lerman (1985). However, this situation assumes conditions of constant glottal resistance and respiratory effort without compensatory responses.

Individuals with VPI may attempt to regulate actively vocal tract resistances as a compensation (Warren, 1986). Increased glottal resistance during vowel productions would decrease flow rate and facilitate regulation of subglottal pressure required to sustain phonation. Additionally, compensatory changes in chest wall dynamics may occur either to increase or to decrease subglottal pressure as needed.

Therefore, attempts to regulate respiratory and laryngeal aerodynamics and neuromuscular processes when inappropriate oronasal coupling exist may contribute to increased voice perturbation.

Hence in the present study, it was hypothesized that

a) There would be significant difference between cleft palate and lip individuals and normals for vowels /a/ , /i/ , and /u/ in terms of voice perturbation.

b) There would be significant difference between cleft palate and lip individuals and normals for alpha, beta, and gama ratios of long term average spectrum measurements.

Zajac and Linville (1989) found increased jitter levels in children with VPI and this increases was statistically significant. Their findings correlate with the theoritical explanation given for the increased in voice perturbation levels of cleft palate and lip indivuduals.

Contrary to expectations, the result of the present study showed no significant difference in voice perturbation in individuals with cleft palate and lip when compared to that of normal individuals. This results are in contradiction with the results of Zajac and Linville (1989). The absence of significant difference in voice perturbation between cleft palate and lip individuals and normals may be due to ;

(a) The present study mainly consisted of individuals with unrepaired cleft palate and lip and a few individuals (6) with repaired cleft palate and lip.

In case of unrepaired cleft palate and lip, because of cleft in the palate both nasal and oral cavity act as a very large oral cavity and the side tube is apparently absent. The absence of the side tube reduces the resistance to air flow. Hence there is reduced glottal resistance and respiratory effort. This reduced glottal resistance and respiratory effort when compared to individual with VPI, offers minimum stress on laryngeal system. Hence reduced voice perturbation.

(b) According to McWilliam et al (1969), the compensatory laryngeal functions for velopharyngeal valving inadequacy will lead to abnormal laryngeal functioning or pathology. They also pointed out that therapy given to improve velopharyngeal inadequacy may lead to hoarseness of voice due to higher sress on the laryngeal system. In the present study majority of subjects had not undergone voice therapy for the reduction of hypernasality. Therefore, these subjects probably had not developed abnormal laryngeal tension. Where as the subjects of Zajac and Linville (1989) had undergone surgical intervention for cleft palate by the age of two years and also they might have under gone voice therapy. The therapeutic techniques to reduce nasality might have caused hoarseness, hence higher voice perturbation in those subjects.

(c) The subjects used in this study were belongs to rural area, where standards of normal speech are not very high. Therefore, children may not attempt to modify or improve their nasality* Hence, there is no stress on the laryngeal system which leads to normal voice having minimal voice perturbation.

(d) The difference in the observation of present study and study conducted by Zajac and Linville (1989) may be due to the difference in subject selection. In the present study majority of subjects were having unrepaired cleft palate and lip where as Zajac and Linville (1989) used subjects having VPI.

In the present study it is also found that alpha, beta and gama ratio of LTAS were not significantly different from normals. This is due to lower noise level in the voice of cleft palate and lip.

The absence of significant difference in voice perturbation and LTAS between normals and individuals with cleft palate and lip may indicate normal laryngeal function in individual with cleft palate and lip. However, the contradictory study by Zajac and Linville (1989) warrens further detail study of laryngeal behavior in cleft palate and lip individuals with VPI and individuals with cleft palate and lip as a separate group. It also suggest that to study thev voice perturbation before and after the therapy for the reduction of hypernasality.

SUMMARY AND CONCLUSIONS

Voice plays an important role in speech communication. The production of voice depends on the synchrony between respiratory, phonatory and resonatory systems. Anatomical or physiological deviations in any of these systems would lead to a voice disorder. Cleft palate is one of the anatomical deviation of speech mechanism. This alters the resonatory system of speech production mechanism affecting both resonatory and phonatory aspects of voice.

The presence of voicecdisorders in individuals with cleft palate have been documented by many investigators (McDonald and Koeppnaker, 1951; Westlake, 1953; Hess, 1959; Bzoch, 1964; McWilliams et al, 1969; Marks, 1971 and D'Antonio, 1988). These investigators have reported phonatory problems such as hoarseness (both with and without vocal cord pathology), breathiness, reduced loudness, deviant - pitch restricted pitch range and tensed strained vocal quality.

As there were very limited number of studies which tries to evaluate the vocal fold functions objectively in cleft palate individuals, the present study was attempted.

The present study was designed to investigate the laryngeal functions and amount of noise component in the voice of cleft palate and lip individuals and to compared them with normals using pitch perturbation, intensity perturbation and LTAS measurements.

In this study 13 cleft palate and lip subjects (6 males and 7 females) in the age range of 8 years to 25 years were studied using laryngograph (Kay Elemetric Corporation), PC-AT Computer and Philips F6121 stereo audio recording deck with AKG D222 Cordoid dynamic microphone.

The measurement for the following parameters were obtained for three vowels /a/ , /i/ , and /u/ phonated at comfortable pitch and loudness and for Kannada voice passage;

1) Relative average perturbation for,frequency
2) Directional perturbation factor for frequency
3) Relative average perturbation for intensity
4) Directional perturbation factor for intensity
5) Alpa, beta and gama ratio of LTAS.

The data obtained was compared with normative data. Basic descriptive statistical analysis and Mann Whitney "U" Test was carried out to findout the significance of difference between normals and cleft palate and lip individuals in all the above para-meters.

The following results were obtained ?

1. There is no significant difference between cleft palate individuals and normals for vowels /a/ , /i/ , and /u/ in terms of relative average perturbation for frequency and intensity.

2. There is no significant difference between cleft palate and lip individuals and normals for vowels /a/ , /i/ and /u/ in terms of directional perturbation factor for frequency and intensity.

3. There is no significant difference between cleft palate and lip individuals and normals in terms of alpha, beta and gama ratio for Kannada voice passage for LTAS measurements.

The absence of significant difference in voice perturbation and LTAS between normals and individuals with cleft palate and lip may indicate normal laryngeal function in individual with cleft palate and lip. This may be attributed to(a) Absence of higher glottal resistance due to the absence of a separate side tube in cleft palate and lip cases. (b) Improper therapeutic to reduce nasality and improper learning of compensatory mechanisim may cause hoarseness. Thus leading to higher voice perturbation. In the present study non of the subjects had undergone therapy. However, the results of Zajac and Linville(l989) were showed significant difference between normals and individuals with VPI in terms of pitch and intensity perturbation. The authors attributed this difference between means to increased glottal resistance, increased respiratory effort and altered air flow dynamics. The contradictory results obtained by Zajac and Linville (1989) warrens further detailed study of laryngeal behaviour in cleft palate and lip individuals with VPI and

individuals with cleft palate and lip as a separate group. It also suggest that to study voice perturbation before and after the therapy for the reduction of hypernasality.

LIMITATIONS :

1. Only 13 cleft palate and lip subjects were studied.
2. Repaired and unrepaired cleft palate and lip were studied together.
3. Objective measurements were not compared with perceptual evaluation of hoarseness and nasality.

SUGGESTION :

1. TO study the laryngeal behaviour of cleft palate and lip individuals with VPI and individuals with cleft palate and lip as a separate group.
2. To study the voice perturbation before and after the therapy for the reduction of hypernasality.

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