SPEECH DIMENSIONS IN DYSARTHRIA WITH LESIONS IN DIFFERENT CEREBELLAR LOCI

Thesis submitted to the University of Mysore for the Degree of

DOCTOR OF PHILOSOPHY (PhD)

IN

SPEECH AND HEARING

by

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To AMMA without whose grace this thesis couldn't have been completed.

Dedicated to my parents, husband and brother

Certificate

This is to certify that the thesis entitled **SPEECH DIMENSIONS IN DYSARTHRIA WITH LESIONS IN DIFFERENT CEREBELLAR LOCI** submitted by Ms. Vandana.V.P for the degree of Doctor of Philosophy to the University of Mysore, Mysore was carried out at the All India Institute of Speech and Hearing, Mysore.

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Certificate

This is to certify that the thesis entitled **SPEECH DIMENSIONS IN DYSARTHRIA WITH LESIONS IN DIFFERENT CEREBELLAR LOCI** submitted by Ms. Vandana.V.P for the degree of Ph.D in Speech and Hearing, in the University of Mysore, Mysore, is the result of the work done by her at All India Institute of Speech and Hearing, Mysore, under my guidance. I further declare that the results of this work have not been previously submitted for any degree.

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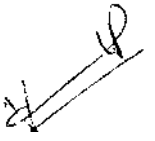
Declaration

I declare that the thesis entitled **SPEECH DIMENSIONS IN DYSARTHRIA WITH LESIONS IN DIFFERENT CEREBELLAR LOCI** which is submitted herewith for the award of the degree of Doctor of Philosophy (Speech and Hearing) at the University of Mysore, Mysore, is the result of work carried out by me at the All India Institute of Speech and Hearing, Mysore, under the guidance of Prof. R. Manjula, Department of Speech Pathology, A.I.I.S.H, Mysore.

I further declare that the results of this work have not been previously submitted for any degree.



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INTRODUCTION

Normal speech production requires good coordination between the various structures and its response to various intrinsic and extrinsic influences. Acoustic targets, aerodynamic values, positions of individual structures, overall vocal tract configuration and rates of muscle contraction are the primary variables controlled by the nervous system with respect to speech production (Kent, 2000). Normal speech is produced by the smooth coordination of five components of speech mechanism including respiration, phonation, resonance, articulation and prosody. When one or any combination of the five components is affected by a neuromotor disturbance, it leads to motor speech disorders such as dysarthria and apraxia. Motor speech disorders which encompass apraxia of speech and the dysarthrias are defined as disorders of speech resulting from neurologic impairment affecting the motor programming or neuromuscular execution of speech (Duffy, 1995).

Dysarthria: Types, assessment & classification

'Dysarthria' refers to a group of speech disorders characterized by disturbance in muscular control due to damage to the central or peripheral nervous system leading to paralysis, paresis, weakness, slowness, incoordination and/or altered tone of speech musculature (Darley, Aronson & Brown, 1969 a & b; 1975). Depending on the site of lesion in the nervous system, different types of dysarthrias have been identified and each of them presents a characteristic symptom c luster.

The types of dysarthrias are differentiated using various methods. These methods are broadly classified under the headings: perceptual, acoustic and physiologic. Perceptual methods are based primarily on the evaluation of auditory perceptual attributes of speech. Acoustic method involves acoustical quantification and description of clinically perceived deviant speech using sophisticated instrumentation. While the perceptual and acoustic analysis focus on the speech signal which is already produced by the activation of the speech musculature, the physiologic analyses describes the activity of the speech musculature while it is being produced. The physiologic analyses is again dependant on the use of instruments which help quantify the movements of the speech structures and the air in the vocal tract, the dynamics of movement in specific muscle groups, the temporal parameters and their relationship with the central processes which control speech.

The perceptual method of classifying dysarthria is considered the "gold standard" for clinically differentiating the types of dysarthria (Ludlow & Bassich, 1983; Portnoy & Aronson, 1982; Sheard, Adams & Davis, 1991). Darley et al., (1969 a & b; 1975) pioneered the use of perceptual classification system to identify clusters of salient perceptual characteristics in speech that are associated with lesions in the central and peripheral nervous system. This system is also popularly referred to as the "Mayo Clinic Dysarthria Research" (Darley et al., 1969a & b; 1975). Many researchers who investigate the acoustic and physiologic bases of dysarthrias also use the perceptual classification system. Darley et al., (1975) identified six major types of dysarthrias. They include (a) Spastic dysarthria (lesion in upper motor neuron) (b) Flaccid dysarthria (lesion in the

lower motor neuron) (c) Hypokinetic dysarthria (lesion in the basal ganglia and associated brainstem nuclei) (d) Hyperkinetic dysarthria [quick hyperkinesias and slow hyperkinesias] (lesion in the basal ganglia and associated brainstem nuclei) (e) Ataxic dysarthria (lesion in the cerebellum and / or its connections (f) Mixed dysarthria (lesion involving any of the structures in the nervous system).

Ataxic / Cerebellar dysarthria

Ataxic dysarthria is caused due to damage to the cerebellum. Heterogeneous etiological factors can cause damage to the cerebellum or cerebellar control circuit pathways. The general etiologic categories include degenerative, inflammatory, neoplastic, toxic, metabolic, traumatic and vascular diseases (Duffy, 1995).

Three deviant clusters of abnormal speech characteristics were reported by Darley et al., (1969 a & b; 1975) in ataxic dysarthria. They include (a) articulatory inaccuracy (b) prosodic excess (c) phonatory - prosodic insufficiency. Many investigators observed additional symptoms other than the ones reported in the classic Darley et al., (1975) classification system for ataxic dysarthria. These included (a) Telescoping (Duffy, 1995) (b) Explosive loudness (Kammermeier, 1969) (c) Voice tremor (Aronson, 1990; Ackermann & Ziegler, 1991).

Cerebellar control for speech

The cerebellar control for speech is a highly complex mechanism. Understanding this complex control is rendered difficult due to the reciprocal connections between the cerebellum and cerebrum in the form of cerebellar inflow

and outflow tracts, including cortico - cerebellar pathways. The anatomical details of cerebellar control of speech, has been deliberated upon from decades. Like in the cerebral lobe, a discrete and localized control of motor functions in the cerebellum is suggested based on the lesion studies (Diener & Dichgans, 1992). From a functional standpoint, the cerebellum is presumed to receive information of intended speech movements from the cerebral cortex and monitor the adequacy of movement outcomes based on feedback received from muscles, tendons and joints. It influences the subsequent cortical motor output based on the feedback and information received from cortex about upcoming movement goals. This permits the cerebellum to make modifications that are required for smooth coordination of speech movement (Kent & Netsell, 1975; Netsell & Kent, 1976; Kent, Netsell & Abbs, 1979). The specific role of cerebellum in speech motor control in normals is described from the point of (a) short loop and long loop revisory function of the cerebellum (Kent, 1990) and (b) motor planning, programming and execution (Vander Merwe, 1997). According to Kent (1990), the role of the cerebellum is to update the provisional, but intact output motor plan from higher centres (i.e. motor cortex) as a short loop revisory function, in order to eliminate the need for immediate sensory feedback from higher centres (i.e. the motor cortex). In cerebellar disease, the long loop revisory system is used for motor programming instead of short loop revisory system. This leads to increased time for processing and results in longer speech segments which are the common characteristics of speech in subjects with cerebellar dysarthria. According to Vander Merwe (1997), cerebellum is involved in the motor programming stage of speech production, which involves selection and sequencing of motor programs of articulatory muscles (including vocal folds) and specification of muscle action in

terms of spatio-temporal and force dimensions such as muscle tone, rate, direction and range of movements. Cerebellum is also functionally associated with the ability to utilize afferent information appropriately in transforming the feedback messages (for novel task) into more skillful feed forward messages (for non novel task) (Kent, Kent, Duffy & Weismer, 1998; Spencer & Rogers, 2005).

The specific neuromuscular basis for ataxic dysarthria was summarized by Kent et al., (1979) as follows:

1) *Physiological basis for cerebellar dysfunction:* Presence of generalized hypotonia which is cited as an important feature of ataxic dysarthria, leads to delay in generation of muscular forces, reduced rate of muscular contraction, and reduced range of movements. These features are reflected in ataxic dysarthric speech as prolongations, slow movements and telescoping (Kent & Netsell, 1975).
2) *Dysfunction in tactile & proprioceptive feedback:* A dysfunction in tactile and proprioceptive feedback affects the temporal dimension of speech. According to Brown, Darley & Aronson (1970) and Kent & Netsell (1975), the stage during which tongue moves to any target locus is guided by proprioceptive sense and the stage during which the tongue is in contact with the target locus is guided by tactile sense. According to Bowman (1971), instances where stop consonants are produced with repetitive contacts and fricatives produced with prolonged constrictions in subjects with ataxic dysarthria are due to dysfunctions in the second stage when articulatory contact is made between the target loci.

3) *Dysfunction in sensory motor integration i.e. biasing of muscle spindles:* Kent and Netsell (1975) opined that cerebellar speech dysfunctions could also be due to inadequate muscle spindle information to higher motor centres during the performance of the movement. They attributed the characteristics of ataxic dysarthria like articulatory prolongations and reduced articulatory rate to disturbance in spindle bias and hypotonia (Kent & Netsell, 1975).

4) *Dysfunction in integration and interpretation of afferent signals:* Even if correct muscle spindle information is relayed to the cerebellum, the cerebellum may fail to process signals with adequate rate, timing, direction and range, resulting in poor integration and interpretation of afferent signals. In ataxic dysarthria, this is reflected as telescoping (reduced range of movements) and prolonged speech segments (Kent & Netsell, 1975).

5) *Modification and execution of motor commands by motor cortex:* The commands released by the motor cortex are refined and precisely controlled by the cerebellum, before actual speech production takes place. According to Kent and Netsell (1975), speech errors like imprecise articulation and prolongation of vowels and consonant constrictions can be attributed to dysfunction at this level.

Functional localization in the cerebellum and cerebro - cerebellar interactions

Functional localization in the cerebellum is explained in terms of cerebral functional control and there are many studies / hypothesis proposed in this regard. Lechtenberg and Gilman (1978) attributed the prosodic disturbances seen in subjects with left cerebellar hemispheric lesion, to the dissociation between right cerebral hemisphere and the left cerebellar hemisphere. Boutsen and Christman

(2001) and Boutsen and Christman (2002) inferred the contralateral cerebro - cerebellar findings based on intrinsic prosodic cues such as syllabic durations, interword durations, voice onset time (mediated largely through left cerebral hemisphere) and extrinsic prosodic cues such as overall speech rate (mediated largely through right cerebral hemisphere). The cerebro-cerebellar connections are also explained based on spectral aspects (controlled by right cerebral hemisphere) and temporal aspects (controlled by left cerebral hemisphere), in the 'Differential cue lateralisation hypotheses' proposed by van Lancker & Sidtis (1992).

Localization of speech function in the cerebellum

Review of literature shows that differences in speech characteristics have been reported in subjects with cerebellar lesions due to different neuropathologies such as tumours (Lechtenberg & Gilman, 1978) and cerebellar infarcts (Amarenco & Hauw, 1990; Amarenco, Roullet, Goujon, Cheron, Hauw & Bousser, 1991; Ackermann, Vogel, Peterson & Poremba, 1992; Stangel, Stapf & Marx, 1999). Murdoch and Theodores (1998) reported that in cerebellar disorders where the lesion is slowly progressive like cerebellar tumours, symptoms of cerebellar dysfunctions tend to be much less severe than in conditions where the lesion develops acutely (e.g. cerebrovascular accidents, traumatic injuries, infarctions etc), and recovery is much faster in cases of acute lesion. Few studies have commented about the presence / absence of dysarthria based on lesion size (Lechtenberg & Gilman, 1978; Ackermann et al., 1992). Ackermann et al., (1992) stated that more than the size of lesion, the location of lesion matters in determining the presence / absence of dysarthria.

The interesting fact to a Speech - language Pathologist is that ataxic dysarthria is not always reported as a common feature in all subjects with cerebellar lesions. In other words, only certain areas of the cerebellum are speculated to be involved in speech motor control. Symptom clusters, including or excluding ataxic dysarthria are reported by some investigators, depending on the lesion site in the cerebellum. A lesion in the midline zone (superior and inferior vermis) is reported to result in dysarthria along with disordered stance and gait, truncal titubation, rotated or tilted head postures and oculomotor abnormalities. A lesion in the lateral hemispheric zone (hemispheres and paravermal regions) is reported to result in dysarthria along with hypotonia, dysmetria, dysdiadochokinesis, ataxia, tremor and oculomotor abnormalities (Gilman, Bloedel & Lechtenberg, 1981; Chiu, Chen & Tseng, 1996). Lesions in certain other areas of the cerebellum were not observed to be associated with ataxic dysarthria. For example, Victor, Adams & Mancal (1959), noted that dysarthria is unusual in chronic alcoholic patients, who tend to have extensive degeneration in the anterior lobe of the cerebellum, including superior portions of the vermis. This suggested that superior vermis and anterior portion of the anterior lobes are not crucial for speech. Brown et al., (1970) confirmed this observation and further stated that ataxic dysarthria is usually seen in bilateral or generalized cerebellar diseases.

Several studies have reported ataxic dysarthria in subjects with focal cerebellar tumours or small cerebellar infarcts in specific sites in the cerebellum. Focal cerebellar lesions in the paravermal (posteromedial) areas and lateral hemispheres of the cerebellum have been implicated with ataxic dysarthria

(Lechtenberg & Giltnan, 1978; Amarenco, Chevrie-Muller, Roullet & Bousser, 1991; Ackermann et al., 1992; Timmann, Kolb & Diener, 1999; Urban et al., 2003; Urban et al., 2006). Based on clinical evidence, lesions in the superior cerebellar peduncle (von Cramon, 1981) and superior cerebellar artery (Ackermann et al., 1992; Barth, Bogousslavsky & Regli, 1993) have been reported to be associated with ataxic dysarthria. Dysarthria was also reported to be present in subjects with lesions in the superior cerebellar vermis, bilateral cerebellar hemispheres, paravermal and lateral aspects of the hemisphere and left paravermal area (Lechtenberg & Gilman, 1978; Gilman & Kluin, 1992; Ackermann et al., 1992; Urban et al., 2003; Urban et al., 2006). Others observed dysarthria when the regions supplied by rostral basilar artery including superior cerebellar artery (Chaves et al., 1994; Erdemoglu & Duman, 1998), rostral paravermal area (Timmann, et al., 1999; Kang, Lee, Bae, Han, Yoon & Roh, 2000) and paramedian regions of the superior cerebellar hemispheres (Ackermann & Ziegler, 1992) were involved. Urban et al., (2006) identified that right paravermal region within the territory of superior cerebellar artery was more frequently involved in dysarthria than left sided cerebellar lesions.

One interpretation of these lesion data is that different speech functions seem to be represented in different regions of the cerebellum, either because of individual variations in regional cerebellar function (population heterogeneity) or because speech is multiply represented in the cerebellum (distributed representation) (Kent, Duffy, Slama, Kent & Clift, 2001). It is possible that different parts of the cerebellum are involved in the control of different motor systems within speech production mechanism or they are involved with different

functions of motor control (Kent et al., 2001). Another point to be noted is that identification of lesion is complicated by the possibility of diaschisis, in which a lesion to one brain structure causes a functional disturbance in a remote structure that itself is not damaged (Botez et al., 1991). On similar lines, Cisneros and Braun (1995), hypothesized that differences in the severity of ataxic dysarthria could be related to lesions in structures other than the cerebellum, like brainstem lesions. Some authors have tried to delineate the speech characteristics associated with ataxic dysarthria, when the brainstem is not involved. In this context, Urban et al., (2003) reported that lesions in the superior paravermal region of the right cerebellar hemisphere may lead to dysarthria that is unrelated to (often concomitant) with brainstem infarctions. To summarize, for a full understanding of ataxic dysarthria, one must account for the remote effects of lesion as well as for its local effects.

Another aspect of cerebellar control of speech, which is widely debated by investigators, is that speech functions might be lateralized within the cerebellum, similar to the lateralization of speech and language within the cerebral hemispheres. Even decades ago, Holmes (1917) and Holmes (1922) observed that more apparent speech dysfunctions were present only when both the cerebellar hemispheres and vermis were damaged and hence vermis is an important region responsible for the regulation of speech. Holmes (1917) and Holmes (1922) also noted that speech usually improves rapidly when lesions are unilateral. The role of cerebellar vermis in controlling speech functions is also supported by Mills and Weisenburg (1914) and Chiu et al., (1996). In contrast, Brown (1949) and Brown (1959) and Amici, Avanzini & Pacini (1976) observed that locus for speech

(1959) and Amici, Avanzini & Pacini (1976) observed that locus for speech dysfunction is in the cerebellar hemispheres and not in the vermis. In opposition to the view of vermal involvement in cerebellar speech motor control, several other studies have either implicated the left cerebellar hemisphere (Lechtenberg & Gilman, 1978), right cerebellar hemisphere (Urban et al., 2003) or bilateral cerebellar regions (rostral paravermal and paramedian regions of superior cerebellar hemisphere) (Ackermann & Ziegler, 1992; Timmann et al., 1999) in controlling speech functions. Lechtenberg and Gilman (1978) summarized that speech was most frequently disordered when there were lesions in the superior portion of the left cerebellar hemisphere, and speech was more strongly represented in the cerebellar hemispheres than in the vermis. They also expressed that because prosodic disturbances are prominent in ataxic dysarthria, the "dominance" of the left cerebellar hemisphere is logically related to its strong cerebellar-cortical ties to the right cerebral hemisphere and its important role in prosodic functions. Gilman et al., (1981), also cited differences in the incidence of ataxic dysarthria in subjects with right and left cerebellar hemisphere lesions, supporting the notion of possible existence of subtypes of ataxic dysarthria. They state, " — It is unlikely that only one cerebellar locus could be responsible for all facets of speech disorder occurring with cerebellar disease".

Ataxic dysarthria as a function of speech task

The characteristics of ataxic dysarthria are also reported to vary depending on the type of speech task performed by the subjects. This is reasoned on the basis of differential demands placed on the motor control mechanism of the speech subsystems by these tasks (Kent, Kent, Rosenbek, Vorperian, & Weismer, 1997).

Kent et al., (1997) emphasized that task based speech profiles help in: (a) classifying dysarthria (b) describing subgroups and variations (c) indicating pathophysiology in the subsystems of speech mechanism (phonatory, articulatory, prosodic and linguistic). They speculated that task specific findings may emerge based on the age of onset, etiology, neuroanatomic area of involvement, speech valves involved (respiratory, laryngeal, pharyngeal, velar, lingual, dental, labial), speech events involved (neural, muscular, structural, aerodynamic, acoustic and perceptual) and the perceptual characteristics (pitch, loudness, voice quality, respiration, prosody, articulation).

Very few speech tasks are reported as sensitive in identifying characteristics of ataxic dysarthria. The tasks of phonation and diadochokinesis (DDK) were reported to be sensitive in identifying the characteristics of ataxic dysarthria (Kent et al., 1997; Kent, Kent, Duffy, Thomas, Weismer & Stuntebeck., 2000). The tasks of sentence repetition and conversation are generally reported to be difficult in dysarthrias, but no reports are available about the sensitivity of these tasks in ataxic dysarthria. Single word intelligibility tasks were not reported to be effective in ataxic dysarthria (Kent et al., 1997). Kent et al., (1997) questioned the effectiveness of scanning index [proposed by Ackermann and Hertrich (1994)] in quantifying speech characteristics in ataxic dysarthria.

These findings suggest the need to identify tasks that are sensitive in differentiating characteristics of ataxic dysarthria. It is also suggested that languages selected per se may have a contributing effect in the dysarthric speech characteristics (Kent et al., 2000). Ataxic dysarthria has been studied extensively

in English and other languages (Duffy, 1995; Kent et al., 1997; Kent et al., 2000; Ackermann et al., 1992; Ozawa, Shiromoto, Ishizaki & Watamori, 2001; Urban et al., 2003; Urban et al., 2006). Studies on ataxic dysarthria in Indian languages are scanty (Vandana, 2000; Jose, 2001; Mili, 2005) and have been carried out on ataxic dysarthric subjects with nonfocal / diffuse lesions.

Vandana and Manjula (2006) suggested differential speech features in subjects with lesions in different areas of the cerebellum, based on the tasks of 'relational speech timing' (changes in the duration of vowel in the initial syllable of the base word with the addition of suffixes). The preliminary findings indicated that the left and right superior paravermal regions may be involved in durational control based on a 'relational speech timing' paradigm. Vandana and Manjula (2007) reported some preliminary findings based on the performance of ataxic dysarthric subjects with lesions restricted to different cerebellar loci on the tasks of phonation and diadochokinesis [Alternating motion rate (AMR) and Sequential motion rate (SMR)]. The findings revealed some speech task specific findings with differences in functional localization of phonatory parameters [Variation in amplitude (vAm) and Smoothed amplitude perturbation quotient (SAPQ)] and diadochokinetic measures. Neural correlates of phonation pointed to the involvement of left superior paravermal, left anteroinferior and superior vermis regions of the cerebellum, whereas those related to diadochokinesis seemed to be lateralized mainly to the right superior paravermal and right anterosuperior cerebellar regions.

Subgroups in atoxic dysarthria

The clinical evidence in ataxic dysarthria proposed by Darley et al, (1969 a) suggested that although prosodic deviations of excess and equal stress and explosive loudness are reported as cardinal features, they are not evident in all ataxic speakers. On similar lines, Duffy (1995) observed that although the perceptual and acoustic studies indicate that slow speech rate and durational variability are common in subjects with ataxic dysarthria, these deviations may occur independently or together. They opined that subgroups of subjects with ataxic dysarthria may exist based on the presence, absence or coexistence of these features. Duffy (1995) was of the opinion that subgroups may exist in whom speech is slow but not dysrhythmic, dysrhythmic but not slow, or simultaneously slow and dysrhythmic.

The possibility of subgroups in ataxic dysarthria was explored by Ackermann and Hertrich (1994), by measuring the tendency towards 'scanning type' of speech in acute and chronic ataxic dysarthric subjects. They measured this in a sentence context. They defined scanning as the absence of durational variability between syllables in the sentence. They did not observe any difference in the scanning index of subjects with acute and chronic ataxic dysarthria. Boutsen, Bakker and Duffy (1997) were of the opinion that ataxic dysarthria may not be a homogenous disorder, and hence describing the characteristics of ataxic dysarthria based on dysarthric severity scales alone may not reveal the differences in speech characteristics in subjects with ataxic dysarthria. They stated that differences in specific loci of lesions were more responsible than etiology & / or severity for the differences observed in speech characteristics among the subjects.

They suggested the possibilities of identifying the subgroups in ataxic dysarthria based on the performance of subjects on diadochokinetic speech task. Suggesting that acoustic basis, more than any other approach would be sensitive in describing the subtypes if any in ataxic dysarthria, they stated that "…the acoustic basis of subtypes may lie in measures of variability, such as DDK rate, and such acoustic variability, relative to identification of subtypes, may derive from or be more apparent in impairments in some speech subsystems than in others". They identified that the syllables /ta/ and /ka/ that involves the tongue tip and tongue blade are more affected than the syllable /pa/ which primarily involves the lip muscles.

Many investigators have carried out acoustic analysis to describe the subsystem errors in the speech of subjects with cerebellar dysfunctions. Some of these studies have been based on analysis of the speech in subjects with lesions in specific sites in the cerebellar structures, but most of the others are observations made without reference to the site of lesion in the cerebellar structure. Few studies have investigated ataxic dysarthria based on acoustic and physiologic analysis including respiratory measures (Abbs, Hunker & Barlow, 1983; McClean, Beukelman & Yorkston, 1987; Deger, Ziegler & Wessel, 1999), phonatory measures (Kammermeier, 1969; Gentil, 1990; Ackermann & Ziegler, 1991; Ackermann & Ziegler, 1994; Hertrich, Spieker & Ackermann, 1998) and articulation, rate and prosodic measures (Kent & Netsell, 1975; Kent et al., 1979; Kent & Rosenbek, 1982; Gentil, 1990a; McNeil, Weismer & Adams, 1990; Ackermann, Hertrich, & Scharf, 1995; Ackermann & Hertrich, 1997; Kent et al., 1997; Kent et al., 2000; Ozawa et al., 2001; Ziegler, 2002). Duffy (1995)

interpreted and generalized within certain limits due to following reasons:

a) The studies summarized included only one or few speakers
b) Not all subjects with ataxic dysarthria exhibited all the symptoms
c) The characteristics observed were not necessarily unique to ataxic dysarthria

Extending the thought of cerebellar lateralization, Duffy (1995) observed that different "types" of ataxic dysarthria may exist depending upon the lateralization of cerebellar lesions. Gilman et al., (1981) also opined that "……it is unlikely that only one cerebellar locus could be responsible for all facets of speech disorder occurring with cerebellar disease". In the light of increasing evidences proposed to postulate that there are subgroups in ataxic dysarthrias and the acoustic analysis is the most sensitive tool to identify the subgroups, there is a need to readdress the issue keeping in mind the evidence proposed to support the discrete and localized nature of speech motor control in the cerebellar hemispheres.

To summarize, nearly three decades ago, Darley et al., (1969a) proposed clinico-anatomic correlations for seven perceptual types of dysarthria, including ataxic / cerebellar dysarthria. These investigators proposed hypotheses, which related the site of lesion to type of dysarthria. Their study was primarily based on clinical observations, especially the perceptual attributes of speech samples of subjects with different neuropathologies. However, these findings were not well supported by neuroimaging findings (Kent et al., 1998). There was a different

scenario with the advent of neuroimaging methods. Urban et al., (2003) used fMRI technique to study dysarthric characteristics in subjects with cerebellar infarctions and reported that lesions in the right superior paravermal region lead to dysarthria that is unrelated to brainstem lesions. Schoch, Dimitrova, Gizewski, & Timmann (2006) studied possible differential role of cerebellar cortex and nuclei in functional outcome by examining somatotopy in the cerebellar cortex using fMRI findings. They found that nonspeech and speech functions were differentially localized in the cerebellum. They found that limb ataxia was significantly correlated with lesions of the interposed and part of dentate nuclei and ataxia of posture and gait correlated with lesions of the fastigial nuclei. Upper limb ataxia was correlated with lesions of vermal, paravermal and hemispheral lobules IV - V & VI, lower limb ataxia with lesions of vermal, paravermal and hemispheral lobules III and VI, dysarthria with lesions of paravermal and hemispheral lobules V and VI and ataxia of posture and gait with lesions of vermal and paravermal lobules II, III and IV.

As highlighted in the previous sections, it can be seen that inconsistency in the speech characteristics associated with ataxic dysarthria is a common finding. There is no common consensus and support as to the existence of typical or common set of speech clusters associated with ataxic dysarthria. The variations in the dimensions of speech as reported by acoustic studies also suggest the possibility of existence of subgroups in ataxic dysarthria depending on the area in the cerebellum that is implicated. There is increasing evidence put forth to suggest that only some anatomical regions in the cerebellum could be involved in speech motor control (Amici et al., 1976; Amarenco et al., 1991; Ackermann et al., 1992;

Lechtenberg & Giltnan, 1978; Urban et al., 2003). There is also evidence put forth by proponents of the perceptual analysis method who suggest that the dysarthria seen when there is lesion in different sites of the cerebellum is associated with different symptom clusters, and there is no single symptom complex in ataxic dysarthria (Victor et al., 1959; Brown et al., 1970; Gilman et al., 1981).

Need for the study

Investigations which have postulated subgroups in ataxic dysarthria are few in number (Lechtenberg & Gilman, 1978; Amarenco et al., 1991; Ackermann et al., 1992; Duffy, 1995; Boutsen et al., 1997; Kent et al., 2000). Majority of these findings were based on perceptual findings only (Fischer, Picard, Polak, Dalai & Pojemann, 1965; Amici et al., 1976; Lechtenberg & Gilman, 1978; Amarenco et al., 1991; Ackermann et al., 1992; Chiu et al., 1996). Few investigations which have postulated subgroups in ataxic dysarthria (Ackermann & Hertrich, 1994; Boutsen et al., 1997) have been based on the acoustic and perceptual measures in ataxic dysarthria subjects with diffuse or nonfocal lesions. Acoustic analyses in these studies were confined to temporal measures, only. There are very few studies, which have aimed to determine the characteristics of ataxic dysarthria, based on the different lesion sites in the cerebellum (Ackermann et al., 1992; Amarenco, Kase, Rosengart, Pessin, Bousser & Caplan, 1993; Urban et al., 2003). There is need for a study that includes both acoustic (spectral as well as temporal) and perceptual measures to evaluate the performance of subjects with lesions restricted to different cerebellar loci, in order to see if there is region specific speech motor control in ataxic dysarthria. There are very few studies on dysarthria in the Indian context (Basava Raj, 1998; Vandana, 2000; Jose; 2001;

Mili, 2005). Studies by Vandana (2000) and Mili (2005) included ataxic dysarthric subjects with diffuse or nonfocal lesions and had limited number of subjects. Studies on ataxic dysarthria due to lesions restricted to the cerebellum only in Indian context are scanty (Vandana & Manjula, 2006; Vandana & Manjula, 2007). Vandana and Manjula (2006) studied Malayalam speaking subjects with lesions restricted to different regions of the cerebellum. The preliminary studies pointed to the differential localization of durational control in speech within the cerebellum. The study by Vandana and Manjula (2007) also pointed to the possibility of differential role of cerebellar regions based on tasks of sustained phonation and diadochokinetic repetitions [alternating motion rate (AMR) and sequential motion rate (SMR)]. There is a need for a detailed study probing into the differential speech subsystem involvement including different speech tasks and both perceptual as well as acoustic paradigm to identify speech motor control by specific region in the cerebellum. The study is planned to fulfill this objective. Malayalam language was chosen for the study as the investigator was a native speaker of Malayalam and also because of the accessibility and availability of subjects with lesions in different cerebellar loci from different hospitals in Kerala state of India where Malayalam is spoken by majority of people.

This study attempted to investigate the voice and speech dimensions in Malayalam speaking ataxic dysarthric subjects with lesions in different cerebellar loci. Only subjects with specified variety of lesions (tumour) were included. Stringent inclusion and exclusion criterias specified in the study restricted the number of subjects and there were limited number of subjects with lesions in

different cerebellar loci. Only subjects with cerebellar tumours were chosen in the study so as to maintain homogeneity within the cerebellar groups. This was done as lesions associated with early cerebellar tumours are restricted to cerebellum, whereas lesions associated with cerebellar infarcts involve extensive regions of the cerebellum as well as regions adjacent to the cerebellum. Literature also reports of differences in symptom manifestations based on whether the cerebellar lesion is due to tumour or infarct (Amarenco & Hauw, 1990; Amarenco et al., 1991; Ackermann et al., 1992; Murdoch & Theodoras, 1998; Stangel et al., 1999; Kang et al., 2000; Urban et al., 2003).

There is also a need to identify and establish a protocol of tasks which are sensitive to tap the differential function of different cerebellar loci. It is difficult to assess in an unbiased manner which speech task is sensitive in identifying the functional control of different cerebellar loci. Hence a protocol of tasks which included phonatory, articulatory, prosodic and perceptual measures were selected in Malayalam language for the study to suit the Indian context. Although speech tasks that principally involve phonatory, articulatory as well as prosodic mechanisms were selected in this study, major focus was on testing the phonatory and articulatory subsystems. Certain tasks that were included in the protocol had to be developed in Malayalam language. There are only preliminary studies on prosodic aspects in Malayalam, and the prosodic structure of Malayalam is not completely understood. The inclusion of tests in the protocol was guided by the reported sensitivity of these tasks in other studies (Kent et al., 1997; Kent et al., 2000). This study also attempted to investigate the perceptual attributes in the speech of subjects with lesions in different cerebellar loci, by characterizing the

speech based on 38 dimensions as suggested by Darley et al., (1975). Studies have often discussed the nature of speech characteristics in ataxic dysarthria using perceptual and / or acoustic methods only. Hence these measures alone were included in this study. The study is undertaken to explore, describe and categorize various speech and voice dimensions in Malayalam speaking ataxic dysarthric subjects with lesions in different loci of the cerebellum, using acoustic and perceptual methods.

Aims of the study:

- To analyse and differentiate some aspects of voice and speech in selected speech tasks in subjects with ataxic dysarthria due to lesions in various sites of the cerebellum using acoustic and perceptual analysis
- To compare the results obtained in individuals with ataxic dysarthria against those of normal control group

Hypothesis:

- It is hypothesized that there would be a difference in voice and speech characteristics of subjects with cerebellar lesions when compared to that of normal control subjects.
- It is hypothesized that there would be differences between groups of subjects with lesions in different cerebellar loci (left superior paravermal (LSP), left anteroinferior (LAI), superior vermis (SV), right superior paravermal (RSP), right posterosuperior (RPS) and right anterosuperior (RAS)].

Method:

The experimental group included seventeen subjects with ataxic dysarthria. This group included subjects with lesions in left superior paravermal (LSP), left anteroinferior (LAI), superior vermis (SV), right superior paravermal (RSP), right posterosuperior (RPS) and right anterosuperior (RAS) regions of the cerebellum. The control group included thirty number of subjects matched for age and sex of the experimental subjects. A cross sectional standard group comparison research design was used for the study. The speech tasks included in the study aimed to analyse the function of: A) Phonatory mechanism, B) Articulatory mechanism and C) Prosodic mechanism.

Material and recording

A task protocol was compiled to study the dysfunction in the domain of A) Phonation, B) Articulation and C) Prosody.

To study for dysfunction in each domain, a task protocol was selected which included some standard tasks and some new tasks. There were three tasks under phonatory, three tasks under articulatory and one task under prosodic domain. The subjects in the experimental and control group were instructed to perform the various tasks in the selected protocol. The voice and speech samples of the subjects were audio recorded using a digital tape recorder Sony MZ-55. The subjects were tested in individual setup.

Analysis

The recorded corpus of speech was subjected to acoustic and perceptual analysis. Acoustic analysis for the phonatory samples were carried out using Multi Dimensional Voice Profile (MDVP) Program in Computerized Speech Lab 4400 (CSL - 4400), Kay Elemetrics software. Other speech parameters were analysed using spectrogram, pitch contour and energy contour modules in Computerized Speech Lab 4400 (CSL - 4400), Kay Elemetrics software. Perceptual analysis was carried out for 38 dimensions of speech based on the method proposed by Darley et al., (1975). The speech samples were rated on a seven point rating scale by three judges. Inter and intra judge reliability was computed.

The data were compiled and discussed under spectral, temporal and perceptual measures for the various tasks.

The implications of the study:

An attempt is made to throw some light on the differential contribution of different cerebellar regions to speech motor control. Very few studies in the past have focused on analysis of lesion specific characteristics of ataxic dysarthria. As a preliminary attempt, this study has aimed to reflect on speech motor control in the different regions of cerebellum, based on a speech task based profile of subjects with ataxic dysarthria with lesions in different regions of the cerebellum.

Limitations of the study:

The study has been carried out with a small number of dysarthric subjects with lesions in different cerebellar loci, which restricts the generalization of the research finding. Equal representation of male and female subjects could not be obtained. Also this study in itself has considered only few tasks of varying complexities. Physiologic analysis was not included in the design of the study. Spontaneous speech samples have not been included. The possible contribution of cerebellar nuclei to dysarthria could not be considered. Future studies incorporating these variables are necessary.

REVIEW OF LITERATURE

Dysarthria comprises of a group of speech disorders resulting from dysfunction in neuromuscular control. Damage to the central or peripheral nervous system, leads to varying degrees of weakness, slowness, in coordination, or altered muscle tone in the speech mechanism affecting speech production (Darley et al., 1969a, b; 1975). Clinically, different types of dysarthrias are identified based on the site of lesion. Each type of dysarthria exhibit characteristic set of perceptual symptoms. The speech subsystems such as respiratory, phonatory, articulatory, resonatory and prosodic systems may be affected differently and to varying degrees in different types of dysarthrias (Kent, 2000). Based on the lesion, different types of dysarthria are identified.

Table 1: Types of dysarthria and the respective lesion sites (Darley et al., 1969 a, b; 1975)

Type of dysarthria	**Lesion**
Spastic dysarthria	Upper motor neuron lesion
Flaccid dysarthria	Lower motor neuron lesion
Hypokinetic dysarthria	Basal ganglia and brainstem nuclei
Hyperkinetic dysarthria	Basal ganglia and brainstem nuclei
Ataxic dysarthria	Cerebellum and / or its connections
Mixed dysarthria	Any combination of structures mentioned above

Ataxic dysarthria is a motor speech disorder associated with lesions in cerebellum or its connections. Cerebellum is mainly concerned with coordination of muscles and muscle groups of the body. Cerebellum does not directly initiate any muscle contractions, but coordinates the timing of muscle group contractions, so that movements in the skeletal muscles are performed smoothly and accurately. The cardinal sign of cerebellar damage is 'ataxia'. This term is suggestive of

incoordination of movements, poor temporal coordination in the execution of complex multicomponent sequential movements as in speech and walking etc. As a result of cerebellar incoordination, complex movements tend to be broken down or decomposed into simple independent sequential movements. These movements are in turn executed with inappropriate force, amplitude and timing, leading to incoordinated movements (Murdoch & Theodoras, 1998). It also plays a major role in planning, learning, and performing skilled motor acts (Daum & Ackermann, 1997; Topka, Massaquoi, Benda & Hallet, 1998; Schulz, Dingwall & Ludlow, 1999).

The speech in ataxic dysarthria is characterized as scanned, slurred, staccato, explosive, hesitant, slow, altered accent and garbled speech (Gilman et al., 1981). However, investigations reveal that all speech symptoms are not seen in all subjects and different clusters of speech symptoms are observed in subjects with lesions in different regions of the cerebellum (Boutsen et al., 1997; Kent et al., 2001). Hence an understanding of region specific anatomical and functional correlates of cerebellum has an important bearing in speech motor control of the cerebellum.

1.0. Neuroanatomical correlates of the cerebellum

Cerebellum comprises of a medial portion called 'vermis' and two 'lateral hemispheres'. Each hemisphere is divided into anterior, posterior and the flocculonodular lobe by deep fissures (Murdoch & Theodoras, 1998). In a superior view, the anterior portion refers to that portion of the cerebellum, which lies anterior to a deep fissure called primary fissure, and this corresponds largely

to the part of the cerebellum called paleocerebellum. The anterior lobe regulates body posture (Murdoch & Theodoros, 1998). The posterior lobe lies in between the anterior and flocculonodular lobes and is the largest and most important portion of the cerebellum. It regulates the voluntary movements of the body and coordinates speech movements (Murdoch & Theodoros, 1998). Posterior lobe is also referred to as the neocerebellum since it is phylogenetically the newest portion of the cerebellum. The flocculonodular lobe is located within the white matter on either side of the midline and is concerned with equilibrium. As a landmark, it consists of four deep nuclei, which include the dentate, globose, and emboliform nuclei (together known as interpositus) and fastigial nucleus. The flocculonodular lobe is the phylogenetically oldest portion of the cerebellum and is also called archicerebellum. The cerebellum also has extensive connections with other parts of the nervous system through the cerebellar peduncles (inferior, middle and superior) (Murdoch & Theodoros, 1998). Schoch et al., (2006) speculated on the possible differential functional role of cerebellar cortex and nuclei by examining the somatotopic organization in cerebellar structures. They found that limb ataxia was significantly correlated with lesions of the interposed and part of the dentate nuclei and ataxia of posture and gait with lesions of the fastigial nuclei. Upper limb ataxia was correlated with lesions of vermal, paravermal and hemispheral lobules IV, V and VI; lower limb ataxia with lesions of vermal, paravermal and hemispheral lobules III and VI and ataxia of posture and gait with lesions of vermal and paravermal lobules II, III and IV.

2.0 .Cerebellar lesions and dysfunctions

Ataxic dysarthria is caused due to different neuropathologies and a variety of focal or diffuse lesions. Several etiological factors such as Cerebellar agenesis, Multiple sclerosis, Friedreichs ataxia, Trauma, Posterior fossa tumours, Ischaemic lesions etc lead to cerebellar insult (Murdoch & Theodoras, 1998; Duffy, 1995). It is generally observed that slow and progressive (e.g. cerebellar tumour cases) lesions lead to less severe symptoms than acute lesions (Murdoch & Theodoros, 1998).

Clinical symptoms of cerebellar lesions include difficulty in execution of alternating ballistic movements, delay in initiation of movement, increased movement durations, reduced speed of movement and impaired rhythmic tapping (Inhoff, Diener, Rafal & Ivry, 1989; Ivry & Keele, 1989; Hallet, Berardelli, Matheson, Rothwell & Marsden, 1991; Ackermann & Hertrich, 1994). Any of these characteristics may be manifested along with dysarthria. In addition to the gait disturbances, truncal instability, titubation, nystagmus, oculodysmetria, hypotonia, impaired check, excessive rebound, dysmetria, decomposition of movement, dysdiadochokinesis, dysynergia, intention or kinetic tremor and pappilledema may also be seen (Duffy, 1995). Dysmetria, a common sign of cerebellar disease, refers to inappropriate range of movement, and is characterized by overshoot or undershoot of targets and abnormalities in speed. The decomposition of movement refers to errors in sequencing and speed of component parts of a movement, with a resultant lack of coordination. 'Dysdiadochokinesis' is a manifestation of decomposition of movement that occurs in cerebellar disease (Duffy, 1995). 'Dyssynergia' is the inability to

perform components of movements and is characterized by abnormalities in rate, rhythm, amplitude and precision. This is reflected in subjects with cerebellar lesions as separation of a series of voluntary movements that normally flow smoothly and in sequence into a succession of mechanical or puppet like movements. Delayed initiation or termination of movements may also be a manifestation of dyssynergia (Murdoch & Theodoras, 1998). Difficulties in standing and walking are the most common signs of cerebellar disease. The gait is often referred to as "ataxic" and is usually broad based and associated with truncal instability leading to falls. On the "Romberg test", subjects with cerebellar disease show a sway towards the side of the lesion (Daube, 1986). A rhythmic tremor of the body or head can also occur in association with cerebellar disease. This is called 'titubation' and is usually manifested as rocking of the trunk or head forward or back, side to side in a rotary motion, several times per second (Duffy, 1995). Cerebellar dysfunction may also be associated with nystagmus (rapid oscillation or jerky back and forth movements of the eyeballs at rest or with lateral or upward gaze) and occulodysmetria (small rapid eye movements as the patient attempts to fix on a visual target and attempts to correct for inaccurate fixation) (Gilman et al., 1981). Another characteristic motor symptom includes a decrease in resistance to passive movement and is referred to as hypotonia.

The location of lesion in the cerebellum dictates the type of speech clusters evidenced in the subject (Lechtenberg & Gilman, 1978; Amarenco et al., 1991; Ackermann et al., 1992; Gilman & Kluin, 1992; Boutsen et al., 1997; Kent et al., 2001). Distinct clusters of symptoms are reported in cerebellar lesions in the midline zone or in the lateral hemisphere (Lalonde & Botez, 1990).

2.1. Lesions in the midline zone of the cerebellum

In lesions of the midline zone (verm is, flocculonodular lobe, fastigial nucleus) of the cerebellum, different cluster of signs and symptoms are reported. These include disordered stance and gait (Mancall & McEntee, 1965; Maurice-Williams, 1975), truncal titubation (Dow & Moruzzi, 1958), rotated postures of the head (Amici et al., 1976), and disturbances of extraocular movements (Dow & Moruzzi, 1958). Holmes (1922), in his pioneering observation, considered dysarthria as a sign of midline cerebellar disease. Studies conducted later by Lechtenberg and Gilman (1978), demonstrated that dysarthria resulted mainly from lesions of cerebellar hemispheres, specifically the left hemisphere.

2.2. Lesions in the lateral (hemispheric) zones of the cerebellum

The cerebellar hemispheres constitute the lateral cerebellar zones with the dentate and interposed nuclei also included. The intermediate cerebellar zone, consisting of the paravermal portions of the cerebellar cortex is also included in the lateral zone (Gilman, 1986). Lesions associated with damage to the lateral hemispheric zone gives rise to hypotonia, dysarthria, dysmetria, dysdiadochokinesis, ataxia, tremors, rebound, limb weakness, oculomotor disorders and decomposition of movement (Gilman, 1986).

3.0. Neuromuscular basis for atoxic dysarthria

The neuromuscular problems in ataxic dysarthria are explained on the following basis: (1) physiological deficits (2) tactile and proprioceptive feedback deficits and (3) deficits in sensory motor integration. The reported deficits are

based on the function of the lateral cerebellum (Kent et al., 1979). Findings specific to the midline cerebellar regions are scanty.

3.1. Physiological deficits

A generalized hypotonia is cited as a primary feature in many subjects of ataxic dysarthria. Hypotonia results in a delay in the generation of muscular forces, a reduced rate of muscular contraction and reduced range of movements. These physiological deficits reflect in speech as prolongation, slowness of movement and telescoping (Kent & Netsell, 1975). Assynergia and other specific movement disorders are also said to be due to poor integration of afferent signals or poor revision of cortical motor commands (Kent & Netsell, 1975).

3.2. Deficits in tactile and proprioceptive feedback

In an experiment reported by Bowman (1971), a series of movement cycles in lingual articulation, were analysed. Two temporal stages were identified in each movement cycle which were explained on the basis of tactile and proprioceptive feedback. The former stage during which the tongue is moved upto the target locus is guided by proprioception and the latter stage during which the tongue is in functional contact with the locus is guided by the tactile sense. In subjects with cerebellar lesions, the terminal stage of the movement cycle is reported to be affected, specifically, the time when contact is established between articulatory surfaces. This was not agreed by Brown et al., (1970) and Kent and Netsell (1975), who were of the opinion that even the first stage, during which the tongue is moved upto the target locus of the movement cycle is disturbed in cerebellar subjects and this results in abnormalities in both the rate and direction

of motion. They were of the opinion that Bowman's (1971) notion of movement segmentation in cerebellar dysfunction did not explain all of the speech movement disorders in ataxic dysarthrics. They further speculated that some movement abnormalities like repetitive contacts for stop consonants, and prolonged constrictions of fricatives can be attributed to a defective processing of tactile information, but other movement abnormalities, such as reduced velocities, incorrect sites of constriction, and missed contacts, could not be explained on the grounds of inadequate mediation of tactile signals.

3.3. Deficits in sensorimotor control

Cerebellum is suggested to be responsible for biasing the muscle spindles, to insure that spindle information of an appropriate nature is supplied to the higher centres as movement is performed. An abnormality in spindle bias was reported to be the cause for hypotonia and velocity insensitivity (abnormally slow movements) that is seen in cerebellar disorders (Kent et al., 1979). Hypotonia was explained as due to inadequate tonic alpha motoneuron discharge and velocity insensitivity was explained as due to compensatory mechanism in response to a lack of proprioceptive feedback about the evolving movement. It was explained that due to velocity insensitivity, the subjects attempt to prevent dysmetria by performing deliberate, slow and cautious movements. This further explains the prolonged articulation and reduced rate of movement that are often evidenced in cerebellar subjects. Kent et al., (1979) reasoned that if muscle spindle information is correctly relayed to the cerebellum, there could be a further failure in integration and interpretation of afferent signals as required for motor control leading to poorly controlled movements, with errors in rate, timing, direction, and

range. Kent and Netsell (1975) opined that the telescoping (irregular articulatory breakdown giving speech a transient accelerated character) in speech could be the result of inadequate muscular contractile forces and prolonged segments due to delays in afferent processing and formulation of corrective motor commands.

Functional control of the cerebellum also includes the revisory control over the motor cortex, which is imprecise and provisional and requires refinement. Deficits in revisory control leads to lack of precision in gestures with regard to direction, range or timing as observed in ataxic dysarthria (Kent et al., 1979).

In summary, Kent et al., (1979), suggested three functions of the lateral cerebellum. The first is the biasing of muscle spindles to ensure that the spindle information of an appropriate nature is supplied to the higher centres as a movement is performed. The second is the integration and interpretation of afferent information. Disturbances in this function results in poorly controlled movements, with errors in rate, timing, direction and range. The third is the revisory control over the commands issued by the motor cortex giving refined and precise control.

4.0. Speech motor control and cerebellum

4.1. Speech motor control in cerebellar disorders

Speech motor control in the cerebellum is delineated to the levels of motor planning, motor programming and motor execution (Vander Merwe, 1997). According to this model, there are four stages of speech production and they are identified as linguistic-symbolic planning which is a non-motor (or premotor)

process, motor planning, motor programming, and execution. According to Vander Merwe (1997), different anatomical regions are involved during different stages of speech production, the cerebellum being involved in the speech programming stage. From the available literature (Vander Merwe, 1997), the programming of speech movements involves the selection and sequencing of motor programs of the muscles of the articulators (including the vocal folds) and specification of the muscle-specific programs in terms of spatio-temporal and force dimensions such as muscle tone, rate, direction and range of movements.

Few studies have cited that "Sequence length effect" reflects the amount of preparation required for an upcoming movement sequence and it specifically serves as an indicator of advanced speech motor programming (Klapp, 1995, 2003; Harrington et al., 2000; Verwey, 2003; Verwey & Eikelboom, 2003). "Sequence length effect" refers to the changes in initial vowel length or initial syllable duration with addition of syllables to a base word. Spencer & Rogers (2005) studied the "sequence length effect" using a reaction time protocol in five speakers with cerebellar ataxia and Friedreichs ataxia and compared this with fifteen control subjects. The changes in reaction time for utterances of varying length and complexity was studied. It was observed that the speech reaction times of subjects with ataxic dysarthria did not increase linearly with an increase in utterance length, suggesting a difficulty in advance programming in this group. The reduced sequence length effect in ataxics was explained as due to deficient use of feedforward information. This was similar to the explanations given by Kent, et al., (1998) and Spencer & Rogers (2005), which stated that the poor performance of ataxic subjects was due to deficient use of feedforward

information, resulting in errors in programming in which the speaker programs only a portion of the utterance as the motor system waits for feedback before progressing to the next syllable.

The characteristic scanning speech quality and disrupted rhythm in ataxic dysarthria was also attributed to deficient programming of utterances by Darley et al, (1969a); Hartelius, Runmarker, Andersen and Nord (2000) and Kent et al., (1998). In an experiment involving ataxic dysarthria subjects in speech tasks like sustained phonation, diadochokinetic rate, sentence repetition and conversation (Kent et al., 1997), speech programming was implicated.

4.2. Short loop and long loop revisory function of the cerebellum

Motor control of cerebellum also includes revision of the motor plans from higher motor centres (i.e. motor cortex). The revisory functions of the cerebellum are described as short loop revisory (does not rely on feedback from motor cortex) and long loop revisory (relies on feedback from motor cortex) function by Kent (1990). According to Kent (1990), the role of the cerebellum is to update the provisional, yet intact output motor plan from higher centres (i.e. motor cortex) as a short loop revisory function, in order to eliminate the need for immediate sensory feedback from higher centres (i.e. the motor cortex). Kent et al., (1979), Kent and Netsell (1975) and Hirose (1986) reasoned that the short loop revisory function is active only for learned motor movements and not for novel motor movements. In cerebellar disease, the long loop revisory system is used for motor programming instead of short loop revisory system. This leads to increased time, which inturn leads to longer speech segments which are commonly seen in the

speech of cerebellar dysarthrias. This hypothesis draws support from the findings of Ackermann and Hertrich (1993), who attributed the prolongation of vowels in cerebellar dysarthric subjects to activation of long loop system in case of disease to the cerebellum.

4.3. Speed and precision of speech movement in cerebellar lesions

Speech motor control in the cerebellum is also explained from the point of view of the speed and precison of speech movements involved. Netsell (1982) reported slow and discontinuous muscle movements in ataxic dysarthria, producing muscle contraction of increased and uniform duration, resulting in disproportionate lengthening of short vowels or lengthening of unstressed syllables in speech (Kent et al., 1979; Ackermann & Hertrich, 1993).

5.0. Localization of speech function in the cerebellum

Several studies have suggested that different regions of the cerebellum contribute differently in speech motor control (Holmes, 1917, 1922; Mills & Weisenberg, 1914; Brown, 1949, 1959; Fischer et al., 1965; Amici et al., 1976; Lechtenberg & Gilman, 1978; Amarenco et al., 1991; Ackermann et al., 1993; Chiu et al., 1996; Timmann et al., 1999; Urban, 2003). Studies reporting the differential function of the cerebellum are reviewed in the following sections.

5.7. *Lesions in cerebellar vermis and speech dysfunctions*

As early as in 1917, in a classic report by Holmes, inconsistent occurrence of dysarthria was reported in subjects with gunshot and shrapnel wounds to cerebellar hemispheres. From case studies, Holmes (1917, 1922) made an

important observation that more apparent speech dysfunctions were present only when both the hemispheres and vermis were damaged and hence vermis is an important region responsible for the regulation of speech. This observation was supported by a case study by Mills and Weisenburg (1914), who reported dysarthria of sudden onset in subjects with haemorrhage in the superior cerebellum, especially vermis. Much later, Chiu et al., (1996), who studied speech and motor functions in fifteen subjects with cerebellar disease, reported that the cerebellar vermis and fastigial nuclei are important for speech motor control

5.2. Lesions in cerebellar hemispheres and speech dysfunction

In contrast to studies (Holmes, 1917, 1922; Mills & Weisenburg, 1914) supporting the role of vermis in speech motor control, Brown (1949, 1959) was of the opinion that the locus for speech dysfunction was in the cerebellar hemispheres and not the vermis. Susequently, Brown et al., (1970) reported dysarthria in subjects with extensive damage to the cerebellum following alcohol abuse. This observation raised some questions as to the importance of vermis in speech motor control. They found no dysarthric features to be associated with lesions of superior vermis and anterior portion of anterior lobe.

Lechtenberg and Gilman (1978) observed that speech was more strongly represented in cerebellar hemispheres than in vermis. Supporting this view, Amici et al., (1976), reported that lesions in the paravermal and lateral regions of the cerebellar hemispheres lead to dysarthria. They did not find dysarthria in subjects with cerebellar tumours in the vermis region. Other studies by Timmann et al.,

rostral paravermal lesions (either in the left or right cerebellar hemisphere) and paramedian regions of the superior cerebellar hemisphere.

Some studies have implicated unilateral hemispheric lesion, especially the left cerebellar hemisphere, in dysarthria (Amici et al., 1976; Lechtenberg & Gilman, 1978; Amarenco et al., 1991). According to Amici et al. (1976), nonavailability of information on the lateralization of speech function in the cerebellum in earlier studies (Brown, 1949, 1959; Brown et al., 1970) might have masked the importance of left cerebellar hemisphere in speech functions. Based on the observation of 31 subjects, Lechtenberg & Gilman (1978) specified that the paravermal superior portion of the left cerebellar hemisphere is involved in controlling speech related functions. They found dysarthria in subjects with left cerebellar disease which was characterized by slurred, slow and scanning speech as a result of articulatory breakdown, excess and equal stress, imprecise consonants, prolonged phonemes, prolonged intervals and slow rate. The retrospective study design and the possibility of perifocal oedema with possible brainstem involvement could have biased the result of their investigation. Amarenco et al., (1991) also reported a subject with dysarthria following a small infarct in the left paravermal zone of the rostral cerebellum.

Few studies did not corroborate the notion of an exclusively left sided cerebellar speech motor control. Ackermann et al., (1992); Cruz-Martinez & Arpa (1997) and Urban et al., (2003) implicated right cerebellar hemisphere in dysarthria. Ackermann et al., (1992) specifically cited the right paravermal region in the superior cerebellar portion as the site responsible for dysarthria. This was

based on the study of three subjects with right cerebellar ischaemia who presented irregular articulatory breakdown as the core speech deficit. This was in contrast to the feature of "excess and equal stress" reported as the core feature in ataxic dysarthria by Darley et al., (1969a, b; 1975). Further support came from Cruz-Martinez & Arpa (1997) who studied subjects with cerebellar stroke in the right cerebellar hemisphere using transcranial magnetic stimulation. Urban et al., (2003) also reported that lesions in the superior paravermal region of the right cerebellar hemisphere may lead to dysarthria that is unrelated to (often concomitant) with brainstem infarctions. Schoch et al., (2006) associated the presence of dysarthria with lesions of paravermal and hemispheral lobules V and VI.

5.3. Speech function in subjects with different neuropathologies of cerebellum

5.3.1. Dysarthria in progressive conditions of the cerebellum

Appearance of dysarthria in progressive cerebellar lesions such as tumours and abscesses allows for observation of localization of speech functions in the cerebellum. Lechtenberg and Gilman (1978) observed that in subjects with cerebellar tumors and recurrent abscesses, dysarthria developed as the lesion extended into the left cerebellar hemisphere and in vermal disease requiring paravermal resections, dysarthria developed after resection of the left paravermal area. Specifically, the superior paravermal segment of the left hemisphere about Larsell's lobules HVI and HVII were implicated in cerebellar speech control. They drew support for this observation based on the reported findings of Lam and Ogura (1952) and Wolfe (1972), who reported that Larsell's lobules HVI and

v-

HVII receives inputs from auditory cortex and has indirect connections with laryngeal nerve.

5.3.2. Dysarthria in cerebellar infarcts

Dysarthria has been reported in subjects with cerebellar infarcts of all vascular territories. Infarctions in the SCA territory leading to dysarthria were reported in 56% (Amarenco & Hauw, 1990) and 75% (Stangel et al., 1999) of cerebellar subjects. In infarctions restricted to the posteroinferior cerebellar artery (PICA) territory, dysarthria was found in 0% to 39% of subjects (Amarenco et al., 1991; Ackermann et al., 1992; Kang et al., 2000). These differences are most likely due to the retrospective study design that was followed, differences in the extent of brainstem involvement, and the absence of a standardized procedure used for the evaluation of dysarthria. Ackermann et al., (1992) specifically observed that dysarthria occurred when there was an infarct in the superior cerebellar artery (SCA) without the involvement of dentate nucleus. In such subjects, dysarthria was characterized by articulatory errors such as reduced articulatory precision, slurred production of single consonants / consonant clusters and inconsistent articulatory errors. In one subject with right SCA infarct and involvement of dentate nucleus, a combination of phonatory as well as articulatory disturbances was observed. One of the subject in this study with bilateral cerebellar infarct (more extensive lesion on the left side) and dentate lesion with no associated brainstem infarct also demonstrated mild phonatory as well as more pronounced articulatory errors. Articulatory errors included reduced articulatory precision (slurred production of single consonants / consonant clusters / distorted vowels) and inconsistent articulation. On the contrary, a subject with right SCA

infarct showed predominant phonatory errors, strident voice with fluctuations of pitch, loudness and voice tremor, but only mild articulatory disturbances. Since this subject also had unilateral brainstem involvement, the role of brainstem in the emergence of phonatory features was not ruled out.

According to Amarenco and Hauw (1990), the brainstem is generally involved in SCA infarcts, posteroinferior cerebellar artery (PICA) and anteroinferior cerebellar artery (AICA) infarcts. They observed dysarthria in four out of 13 subjects with AICA territory infarcts, with brainstem involvement. They observed that most SCA infarcts were associated with rostral brainstem and occipitotemporal infarcts. Urban et al., (2003) showed that the rostral paravermal region of the anterior lobe in the territory of right SCA, was linked with the coordination of articulatory movements of the tongue and orofacial muscles, leading to dysarthria that is unrelated to brainstem infarcts. Urban et al., (2006) found dysarthria to be associated with both right as well as left sided SCA lesions. However, the occurrence of dysarthria was more pronounced in the right sided SCA lesions (77.7 %) compared to left sided SCA lesions (22.3%). They identified the paravermal region within the territory of SCA to be affected in all these lesions. The cerebellar dysarthria was characterized by impaired consonant articulation, slow speaking rate, prolonged phonemes and syllables, articulatory inaccuracy, reduction of phonemes and syllables and imprecise vowels and /or distorted vowels.

Most of the investigators have observed the occurrence of dysarthria in clients with lesions in medial superior cerebellar artery (mSCA) territory and / or

lateral superior cerebellar artery (1SCA). Amarenco et al., (1991) observed dysarthria in a subject with infarct in the left mSCA of the cerebellum [the area implicated by Lechtenberg & Gilman (1978) as the most frequently involved in cerebellar dysarthria]. The symptoms were characterized by slurred speech, explosive staccato scanning vocalization, and wavering voice modulation. The dysarthric characteristics in subjects with 1SCA was mild or characterized as slurred speech, and they were often associated with ipsilateral dysmetria of the limbs, vertigo or unsteadiness, limb or trunk ataxia, and nystagmus, rather than speech symptoms. The posteromedial (paravermal) part of rostral cerebellum (supplied by mSCA) was suggested to play a major role in vocalization and speech compared to anterior part of rostral cerebellum (supplied by 1SCA).

Ackermann et al., (1992) were of the opinion that the degree of dysarthria and the speech subsystems involved depended more on location of the lesion than the size of lesion. They reported dysarthria in a subject with SCA infarct in the right paravermal cerebellar area posterior to anterior fissure, which was smaller than the lesion reported by Lechtenberg and Gilman (1978). Another subject with bilateral infarction of cerebellum (infarction more on the left side) showed marked articulatory deficits in addition to phonatory disturbances which were mild.

In general, studies have suggested that all SCA infarctions are strongly associated with dysarthria. The occurrence of articulatory or phonatory errors in dysarthria varied depending on the site of lesion within the cerebellum and also on the extent of involvement of the brainstem.

5.4. Summary of findings with respect to localization of speech functions in the cerebellum

Decades ago, a proposed vermal origin of speech (Holmes, 1917; Mills & Weisenberg, 1914) was questioned by Brown et al., (1970). Subsequent clinicoanatomic studies of ataxic dysarthria implicated a variety of cerebellar lesions, including the superior cerebellar vermis, bilateral cerebellar hemispheres, paravermal region, lateral region of the hemispheres, and left paravermal area (Amici et al., 1976; Lechtenberg & Gilman, 1978; Amarenco et al., 1991; Ackermann et al., 1992; Urban et al., 2003). Kent et al., (2001) speculated that that different speech functions are represented in different regions of the cerebellum, either because of individual variations in regional cerebellar function (population heterogeneity) or because speech is multiply represented in the cerebellum (distributed representation). Kent et al., (2001) were of the opinion that different parts of the cerebellum are involved in the motor control of different motor systems within speech mechanism or they may be involved in different aspects of functional motor control and the disruptions may be associated with certain regions more than the others.

The left paravermal area has been implicated in the control of speech more often and is based on clinical reports of subjects with progressive lesions or reported speech sequelae subsequent to tumour resection (Lechtenberg & Gilman, 1978). Interesting observation was also made in terms of lesion size and involvement of cerebellar nuclei (Ackermann et al., 1992). In view of the fact that most of the cerebellar infarcts (AICA, PICA and SCA) were associated with brainstem lesions (Amarenco & Hauw, 1990), it was speculated that a combination of cerebellar lesions and brainstem lesions may lead to dysarthria

(Urban, Hopf, Visbeck, Fleischer & Andreas, 1996). Most of the reported studies however exercised caution in interpreting dysarthric characteristics based on lesion type. Rapid changes in signs of neurologic disease in cerebellar hemorrhage and cerebellar infarction are a major limitation in drawing conclusion on specific lesion sites being responsible for the different clinical signs. Studies on subjects with isolated cerebellar lesions without associated brainstem lesions are necessary to gain insight about cerebellar speech motor control.

The varied opinion on occurrence of dysarthria in subjects with lesions localized to different regions in the cerebellum may be limited due to the small and / or varying subject sample. Further, it is influenced by the cooccurrence of brainstem lesion, and variation in evaluation procedures used for description and assessment of dysarthria. There are many studies which have focused on motor characteristics in localized lesions of the cerebellum than on the speech characteristics associated with dysarthria. Dysarthria, as a symptom has not been characterized for specific speech errors in the phonatory, articulatory or prosodic subsystems in the speech production mechanism.

Table 2: Summary of the lesion specific studies in ataxic dysarthria

Investigators	**Lesion**	**Speech findings**
Holmes (1917, 1922)	Cerebellar hemisphe-res & vermis	Vermis important in regulation of speech Dysarthria seen in hemispheric lesions
Mills & Weisenberg (1914)	Superior cerebellum (especially vermis)	Dysarthria present
Dow &Moruzzi (1958)	Vermis	Dysarthria absent
Brown (1949, 1959)	Cerebellarhemispheres	Dysarthria present

Victor etal., (1959)	Superior vermis and anterior portion of anterior lobe	Dysarthria absent
Geschwind (1975); Fischer etal., (1965)	Unilateral cerebellar hemispheric lesion	Dysarthria present
Amici etal., (1976)	Paravermal and lateral regions of cerebellar hemispheres and vermis	Dysarthria present
Lechtenberg & Gilman (1978)	Paravermal superior portion of left cerebellar hemisphere	Dysarthria present & characteried by slurred, slowed and scanning speech as a result of articulatory breakdown, excess and equal stress, imprecise consonants, prolonged phonemes and intervals and slow rate
Thach (1980)	Right and left cerebellar hemispheres and anterior region of vermis	Dysarthria present
Amarenco et al., (1989)	Posterior inferior cerebellar artery	Dysarthria seen occasionally
Amarenco & Hauw (1990)	Anterior inferior cerebellar artery and posterior inferior cerebellar artery associated with brainstem infarction	Dysarthria common in AICA.
Ackermann et al., (1992)	• Paravermal region of superior cerebellar portion and right or left cerebellar lesion • With and without dentate involvement • Bilateral infarctions	• Lesion in cerebellar cortex without involvement of dentate nucleus causes dysarthria i.e. articulatory impairments • Lesion in dentate nucleus is associated with marked phonatory abnormality in addition to articulatory disturbances • Bilateral lesions (both cerebellar hemispheres (more severe deficits)
Ackermann et al., (1992) & Gilman & Kluin(1992)	Paramedian region of superior cerebellar hemisphere	Dysarthria present
Amarenco et al., (1993)	Rostral cerebellar infarcts	Dysarthria present

Barthetal.,(1993)	Superior cerebellar infarction	Dysarthria present
Chiu et al., (1996)	Vermis and fastigial nuclei	Dysarthria present
Urban(2003)	Superior paravermal area of right cerebellar hemisphere with brainstem infarction	Dysarthria present

6.0. Cerebro-cerebellar connection and its role in speech motor control

Cerebro-cerebellar connections play an important role in motor speech control. Lechtenberg and Gilman (1978), cite two cortico-cerebellar pathways as important in speech motor control: (a) the frontopontocerebellar tract originating in Brodmann area 10, and (b) the tract connecting the orofacial area of motor cortex with the paravermal segment of the contralateral cerebellar hemisphere. Lesions within cerebellum can influence the function of these tracts. This may be due to 'diaschisis'. In diaschisis, a lesion to one brain structure (the cerebellum in this case) causes a functional disturbance in a remote structure that is not damaged. Diaschisis, therefore causes difficulty in identification of the lesion.

Prosodic disturbances in cerebellar disease raise interesting issues regarding the role of the cerebellum in speech motor control. Dysarthria is reported to occur more often when the lesion is in the left cerebellar hemisphere than the right hemisphere (Lechtenberg & Gilman, 1978). This hemispheric effect was explained as due to the influence of contralateral cerebrocerebellar connections. Prosodic functions are controlled by right cerebral hemisphere. Due to strong contralateral connections, it has been hypothesized that prosodic disturbances are indicative of the dissociation between right cerebral hemisphere

and the left cerebellar hemisphere in ataxic dysarthria (Lechtenberg & Gilman, 1978). However, Ackermann et al., (1992) had a different opinion. They observed that cerebellar dysarthria occurred when there were lesions in either the left or the right paravermal region of superior cerebellar hemisphere and hence both hemispheres of the cerebellum are equally involved in speech motor control.

The contribution of contralateral cerebello-cerebral connections to speech motor control calls for a review of the functions of the right and left cerebral hemispheres.

6.1. Cerebral hemispheric control and prosody

Speech gestures are often classified into fast gestures as in phoneme production and slow gestures like those related to prosody (Perkell et al., 2000; Mobius & Dogil, 2002). In the same way, some have proposed a distinction between intrinsic and extrinsic prosody (Boutsen, 2001, 2003; Boutsen & Christman, 2001, 2002). Intrinsic prosody is reported to be mediated largely through left cerebral hemisphere and includes aspects such as (a) syllabic plan (successive syllables), syllabic magnitudes and syllable durations, basic consonantal and vocalic gesture specifications (front-back, transition extent & others) (b) Microsegmental rate (movement transition, inter word intervals, gesture durations). That is, intrinsic prosody is mainly related to segmental aspects of speech. It is processed primarily by the left cerebral hemisphere. Extrinsic prosody is processed primarily by the right cerebral hemisphere and involves manipulation of intonation across longer utterances, variations in phrasal intonation, tonic stress, and overall speech rate that are message-driven and satisfy

the communicative needs of both a speaker and a listener. Thus, left cerebral hemisphere, more than the right, is dedicated to short sensorimotor speech activities including voice onset time (VOT) and production of transients (Boutsen & Christman, 2001, 2002; Boutsen, 2001, 2003).

6.2. Cerebellar control in laryngeal and supralaryngeal functions

Cerebellum is said to be crucial in controlling and maintaining the temporal aspects of any motor act. Temporal regulation by cerebellum may be as precise as a measure in milliseconds (Ivry, 1996) or seconds (Malapani, Dubois, Rancruel, & Gibbon, 1998). Boutsen & Christman (2002) speculated that the right cerebellar hemisphere is involved in the coordination of supralaryngeal and laryngeal movements as is required for voice onset time and in the maintenance of syllable integrity. They also speculated that left cerebellum is likely to be involved in controlling the tonal aspects of speech over a span of several syllables.

Apart from the observations of Boutsen and Christman (2002), other studies have suggested a distinct acoustic-perceptual process for timing versus pitch characteristics (van Lancker & Sidtis, 1992) associated with the left and the right cortical hemisphere. The 'Differential cue lateralisation' hypothesis proposed by van Lancker & Sidtis (1992) contends that acoustic cues to prosody are lateralised to different cerebral hemispheres with fundamental frequency (F0) parameters processed by right cerebral hemisphere and temporal parameters by left cerebral hemisphere in speech production as well as speech perception. Considering the role subserved by the contralateral cerebro-cerebellar connection, the assigned functions of the cerebral hemispheres have a contralateral

representation in the cerebellar hemispheres. In this context, Lechtenberg and Gilman (1978) suggest that, given the vast documentation of laterality effects on cerebral-cortical functions for speech, and the ample connections between cerebral and cerebellar hemispheres, laterality should be considered an important factor in cerebellar functions.

After reviewing studies on localization of speech functions within the cerebellum, cerebellar function control and role of dominant and nondominant cerebral hemispheres in cerebellar speech functions, it is pertinent to review studies on the speech characteristics associated with ataxic dysarthria. Vast differences in symptoms / characteristics of ataxic dysarthria are reported as a function of selected speech task (Kent et al., 1997). Ataxic subjects In most of the studies, ataxic subjects included were those with diffuse lesions or lesions which were not restricted to the cerebellum alone. Very few studies have addressed the characteristics of ataxic dysarthria associated with specific lesions in cerebellum. Unlike the speculation made from the 'Differential cue lateralization hypothesis' for cerebral control, no specific hypothesis has emerged with regard to the cerebellar control. However, observation based on few studies with specific involvement of cerebellar areas does throw some light in this regard.

7.0. Differential speech characteristics of cerebellar dysarthria

Speech characteristics associated with ataxic dysarthria have been described using perceptual, acoustic, and physiologic techniques in different languages (Ackermann & Ziegler, 1992). The neural mechanisms of the cerebellum that underlie the coordination, temporal regulation, and automatic

control of respiratory, phonatory and articulatory movements for speech are reported to be impaired in ataxic dysarthric subjects (Duffy, 1995; Ziegler & Wessel, 1996; Kent et al, 1997; Kent et al., 1998; Kent & Kent, 2000; Kent et al., 2000; Kent et al., 2001). Some studies have described the perceptual characteristics in ataxic dysarthria (Darley et al., 1969a, 1969b; Duffy, 1995; Urban et al., 2003; 2006) and some have described the acoustic characteristics (Kent et al., 1997; Kent et al., 2000). There are inconsistent findings reported in the speech characteristics associated with ataxic dysarthria. According to Duffy (1995), ataxic dysarthria may affect any or all of the respiratory, phonatory, resonatory and articulatory levels of speech. But, articulation and prosody are maximally affected in ataxic dysarthria. Urban et al., (2006) reported similar findings and indicated that in cerebellar lesions (isolated ischaemic stroke in the right or left SCA region) parameters of articulation and prosody are more pronounced than phonatory characteristics. Darley et al., (1969a, 1969b) and Duffy (1995) reported imprecise consonants, excess and equal stress (reduced stress contrasts), irregular articulatory breakdowns, distorted vowels, harsh voice, prolonged phonemes and intervals, and voice tremor as the most frequently affected perceptual characteristics in ataxic dysarthria. However, not all these perceptual features can be seen in all ataxic dysarthric subjects. The acoustic features of ataxic dysarthria, such as variation in fundamental frequency (vFo) and the standard deviation of fundamental frequency (STD), variability in energy measures in diadochokinetic (DDK) task, slow speaking rate and formant frequency ranges in sentences are also said to be varied (Kent et al., 2000). Increased voice onset time (Kent et al., 1997) and disproportionate length of lax vowels (Kent et al., 1979; Kent & Rosenbek, 1982; Ackermann et al., 1995) have

also been reported in acoustic studies on ataxic dysarthria. These perceptual and acoustic findings may have a physiologic basis as studies on ataxic dysarthria have documented slow movements, errors of direction and range of movements, impaired muscular forces in production of rapid movements, and reduced or exaggerated range of movements in respiratory, phonatory and articulatory systems (Duffy, 1995; Kent et al., 2000). These abnormalities may be exaggerated due to the underlying motor symptoms characterizing cerebellar dysfunction, such as hypotonia, dysmetria, tremor and dysdiadochokinesis (Duffy, 1995; Kent et al., 2000).

Breakdown in the articulatory and prosodic aspects of speech is typically reported as a predominant characteristic of ataxic dysarthria (Brown et al., 1970; Duffy, 1995). This inference was drawn after identifying the salient perceptual speech characteristics in cerebellar subjects with varying etiologies like tumours, multiple sclerosis, cerebellar degeneration, spinocerebellar degeneration, toxic effects of alcohol, strokes and effects of trauma. According to Brown et al., (1970), the most characteristic of ataxic dysarthria include three clusters.

a) *Articulatory inaccuracy:* characterized by imprecise consonant production, irregular articulatory breakdowns and distorted vowels. Articulatory inaccuracy was thought to arise from abnormalities in the control of rate, range, direction, force and timing of voluntary movements (Kent & Netsell, 1975) and from the inaccuracy of individual movements and dysrrhythmia of repetitive movement (Kent, 1990) and ataxia of the respiratory and oral-buccal-lingual musculature (Brown et al., 1970).

b) *Prosodic excess:* characterized by excess and equal stress, prolonged phonemes, prolonged intervals and slow rate. These characteristics were attributed to slowness of individual and repetitive movements, indicating that the overall time program for speech is distorted (Kent & Netsell, 1975).

c) *Phonatory prosodic insufficiency*, characterized by harshness, monopitch and monoloudness. According to Kent & Netsell (1975), hypotonia is believed to be responsible for this cluster of deviations.

The characteristics that are generally agreed upon as being common in ataxic dysarthria are the scanning pattern of speech, disturbed articulation of both consonants and vowels, and abnormal voice quality (Kent et al., 1997) These characteristics are evident in a variety of speaking tasks, like sustained phonation, diadochokinesis and conversation (Brown et al., 1970; Kent & Kent, 2000; Kent, et al., 1997).

Task based analysis of dysarthria has gained importance with the study reported by Kent et al., (1997). A task-based analysis provides insight into the nature of the dysarthria and helps to (a) classify the type of dysarthria (b) identify and describe subgroups and individual variations within a dysarthric type (c) indicate pathophysiology in the subsystems of speech production (phonatory, articulatory, prosodic and linguistic features) and (d) explain task-dependant nature of the dysarthria in cerebellar disease. According to Kent et al., (1997), task specific patterns seen in ataxic dysarthria may be because of the difference in the speech subsystem involvement / speech motor control demands involved in these

tasks. According to them, there is evident difference in the degree of motor programming involved in different speech tasks. In addition, different tasks pose different degrees of demands on the cognitive-linguistic processing. Also, the involvement of the subsystems of speech production mechanism may also differ depending on the speech tasks.

Task based performance differences in dysarthric subjects have been reported in both perceptual (Zeplin & Kent, 1996) and acoustic measures (Kent et al., 1997). Zeplin & Kent (1996) replicated the Mayo clinic perceptual study of dysarthria and reported that the most deviant and frequent perceptual dimensions for a given form of dysarthria included the differences between the tasks of syllable repetition and passage reading. They named these deviant dimensions as 'core features'. The core features of ataxic dysarthria were reported to be imprecise consonants and reduced stress. The core features of monoloudness and monopitch could be identified for the task of passage reading but not for syllable repetition task. On similar lines, Kent et al., (1997) reported a profile after task based analysis of ataxic dysarthria using acoustic measures. The tasks included sustained vowel phonation, syllable repetition, word intelligibility, sentence recitation and conversation. The premise of the study was the fact that though ataxic dysarthria was defined globally as having slow rate, scanning pattern of syllable timing, inconsistent articulatory errors and breakdowns, the prominence of these signs varied with the speaking task. The rationale for each of these tasks was described by them as follows:

a) Sustained phonation: Phonation was described as the 'simplest task for isolating the respiratory-phonatory system for speech' (Duffy, 1995). Kent

et al., (1997) reported higher values of shimmer measures and long term variability of fundamental frequency (FO) in the cerebellar group compared to normal control group.

b) Diadochokinesis: Alternating motion rate (AMR) involves the rapid repetition of the same syllables whereas sequential motion rate (SMR) refers to the rapid repetition of syllable sequences (Forrest, Weismer & Turner, 1989; Tjaden & Watling, 2003). Slow rate of syllable repetition was characteristic of ataxic dysarthria compared to the normal control group (Kent et al., 1997).

c) Word intelligibility task: Single word intelligibility task was not considered sensitive in subjects with ataxic dysarthria (Kent et al., 1997). However, few errors could be identified in the severe dysarthric group.

d) Sentence recitation: This task allows for controlled vocabulary and syntax and also increases the complexity of speech task. The performance of ataxic dysarthric subjects on sentence recitation reflects on their performance on a complex speech task.

e) Conversation: A complex speech task such as the conversational task helps to examine speech motor control in relation to language formulation and communicative interaction (Kent et al., 1997). The patterns of prosody and paralinguistic features can also be identified from this task.

Many investigators have speculated the existence of subgroups in ataxic dysarthria based on the reported variability in the cluster of speech symptoms in subjects with ataxic dysarthria. Duffy (1995) speculated the possibility of subgroups in ataxic dysarthria based on the pattern of syllable repetition and the

prominence of excess and equal stress. Duffy (1995) reported that subjects with relatively stable syllable repetition rates most often showed excess and equal stress as a predominant feature, whereas this feature was less frequent in subjects with prosodic excess and irregular syllable repetition rates. All ataxic dysarthric subjects did not exhibit the features of excess and equal stress and explosive loudness. According to Darley et al., (1969), excess and equal stress was not evident in 30% of ataxic dysarthric subjects and explosive loudness was not evident in 66% of the ataxic speakers, in a conversational task. Ackermann and Ziegler (1994), studied subjects with pure cerebellar atrophy and olivopontocerebellar atrophy. They reported that only a subgroup of both these subject groups showed large pitch fluctuations, increased jitter values, or both. They attributed the phonatory disturbances to asymmetrically distributed motor deficits at the laryngeal level and altered the reflexes in the laryngeal or respiratory muscles. The observation was particularly important for potential subtyping of subjects because the phonatory deficits frequently reported were either monopitch or monoloudness on the one hand and excessive pitch variability and explosive loudness on the other.

A review of the studies on characteristics associated with ataxic dysarthria in general, becomes important before we review the characteristics of subgroups in ataxic dysarthria. The characteristics, both acoustic as well as perceptual are presented under each of the speech subsystems of (a) respiration (b) phonation (c) articulation (d) resonance (e) prosody.

8.0. Speech subsystem dysfunction in atoxic dysarthria

A single subsystem of speech or a group of subsystems of speech may be affected depending on the areas in the cerebellum that are involved (Ackermann et al., 1992).

8.1. Respiratory dysfunctions in atoxic dysarthria

A number of studies have reported the perceptual correlates of respiratory inadequacy in ataxic dysarthria (Darley et al., 1969a; Kluin, Gilman, Markel, Koeppe, Rosenthal & Junck, 1988; Chenery, Ingram & Murdoch, 1990). All these studies were carried out on subjects with nonfocal cerebellar lesions. Studies have reported significant reduction in respiratory support for speech as well as sudden forced inspiratory and expiratory sighs (Chenery et al., 1990); audible inspiration (Kluin et al., 1988) and excessive loudness variations (Darley et al., 1969a) in subjects with ataxic dysarthria. Ebert, Hefter, Dohle & Freund (1995), noted a considerable variability in minimum and maximum energy measures (especially the energy minimum) for each peak in the diadochokinetic task and this was reasoned to reflect the respiratory instability or dyscoordination. Also, shorter utterances on a breath group was seen in ataxic subjects compared to the control subjects, in a conversational sample (Kent et al., 1997), reflecting indirectly on the respiratory dysfunction in ataxic dysarthria.

Several studies attempted to understand the physiological basis for respiratory dysfunction in cerebellar dysarthria (Brown et al., 1970; Abbs et al., 1983; Yorkston, Beukelman & Bell, 1988; Murdoch, Chenery, Stokes & Hardcastle, 1991). Brown et al., (1970) reported reduced respiratory rate and vital capacity in subjects with nonfocal cerebellar lesions and this was attributed to

poor temporal coordination between respiration and phonation. Based on the study of circumferential size changes of the rib cage and abdomen as observed in a subject with ataxic dysarthria, Abbs et al., (1983) attributed respiratory dysfunction to incoordination in the movement of the chest wall and ribcage. Yorkston et al., (1988); Cisneros & Braun, (1995) and Deger et al., (1999) reported reduced and variable lung volume levels that were inadequate for the purpose of speech in cerebellar subjects with nonfocal lesions. Murdoch et al., (1991) noticed abrupt changes in the movements of the rib cage and abdomen and irregularities in chest wall movements during sustained vowel phonation and syllable repetition in some of their subjects with nonfocal cerebellar lesions.

8.2. Phortatory dysfunction in ataxic dysarthria

Phonatory disturbances are reportedly conspicuous in ataxic dysarthria with nonfocal cerebellar lesions. Perceptual attributes of phonatory dysfunction (Darley et al., 1969a; Joanette & Dudley, 1980; Gilman & Kluin, 1992) as well as acoustic attributes (Ackermann & Ziegler, 1991; 1994) were described in ataxic dysarthria due to isolated cerebellar disease (Ackermann & Ziegler, 1994) Friedreichs ataxia (Joanette & Dudley, 1980) and spinocerebellar degeneration (Gilman and Kluin, 1992). Phonatory abnormalities commonly described in ataxic dysarthria included monopitch, monoloudness and harshness (Darley et al., 1969a, 1969b); harshness, pitch level and pitch breaks (Joanette & Dudley, 1980) and alternating loudness, fluctuating pitch, transient harshness, transient breathiness, voice tremors and audible inspiration (Gilman & Kluin, 1992). Darley et al., (1975), were of the opinion that although the reported features were solely attributed to phonatory dysfunction; many of them could also partially be the

result of dysfunction at other levels of the speech production mechanism (e.g. the respiratory system).

The findings on the perceptual variability of pitch and loudness in ataxic dysarthria seem to be equivocal. Inconsistencies are reported with reference to pitch and loudness. It is reported to be reduced (Brown et al., 1970), increased (Joanette & Dudley, 1980) or alternating (Kluin et al., 1988) in ataxic dysarthria. Chenery et al., (1990), observed that, in general, the features of phonatory-prosodic insufficiency i.e. monopitch and monoloudness are the most frequent which are associated with severe forms of speech deviations than abnormal pitch and loudness variations. Acoustic studies have especially pointed to an increased short-term and long-term variability in phonation leading to phonatory dysregulation (Gentil, 1990a; Zwirner, Murry & Woodson, 1991; Kent et al., 1997; Hertrich et al., 1998). However, methodological differences across studies make it difficult to specify the nature of voice dysfunction in ataxic dysarthria.

Acoustic studies have most often supported the findings of Joanette and Dudley (1980), that ataxic dysarthria is characterized by higher pith level. Based on the acoustic measures of mean F0, Ackermann and Ziegler (1994) reported that five of their 20 subjects with cerebellar disorder who had a history of cerebellar atrophy had an overall mean F0 above the normal range, while the other 15 subjects exhibited values within the normal range. Three of the five subjects with increased pitch level had atrophy confined to the cerebellum and two had olivopontocerebellar atrophy (OPCA) i.e. non focal cerebellar lesions. Thus they concluded that increased pitch level could be present in focal as well as nonfocal

cerebellar lesions. The increased pitch level was reasoned to be due to altered sensory feedback from the laryngeal structures. They also observed an increased vocal effort in these subjects and this was attributed to a compensatory mechanism employed by the subjects to overcome the sensory disturbances. They however expressed that the finding needs to be confirmed with additional studies. None of the subjects with cerebellar disorders reported in Ackermann and Ziegler (1994) study showed consistent pitch lowering. They however noticed an intrinsic pitch effect, with reduced FO for /a/ as compared to high vowels /i/ and /u/. Zwirner et al., (1991) found no significant difference between the mean FO values of male ataxic subjects [123 Hz (SD 32, range 83-176)] compared to the mean FO values of normal male controls [118 Hz (SD 15, range 99-147)]. They found that the standard deviation of FO (reflecting long term phonatory instability) was significantly higher in ataxic dysarthrics than in the control group. The pitch fluctuations that were commonly seen in cerebellar disorders could be a reflection of the disruption in the proprioceptive loops mediated through extracerebellar structures (Mackay & Murphy, 1979; Ackermann & Ziegler, 1994).

Vocal tremor has occasionally been reported to be present in some subjects with cerebellar lesions. Vocal tremor refers to long-term quasi-periodic modulation in frequency and/or amplitude (Horii, 1979, 1983). Ackermann and Ziegler (1994) found a quasi-rhythmic modulation of the FO contour at a frequency of 2.8 Hz in an ataxic dysarthric subject, who demonstrated predominant pitch fluctuations. They explained the cerebellar voice tremor as postural tremor due to isometric contraction of the internal laryngeal muscles. They did not observe any significant between - trial pitch variations; with the

exception of one ataxic subject who showed a value exceeding the normal range for high vowels. According to them, vocal tremor may not be the most deviant dimensions in ataxic dysarthria. Boutsen, Duffy and Dimassi (2004) found abnormally high vocal tremor in vowel prolongations of ataxic-dysarthric (with variable etiology) compared to normal speakers. They speculated a possible association between the presence of vocal tremor and extracerebellar pathology as eight out of thirteen subjects who had vocal tremor showed associated extracerebellar pathology. Vocal tremor was seen in only one out of five subjects with pathology confined to the cerebellum. They further observed that the rate of tremor (5 Hz in normal speakers and 3 Hz in ataxic dysarthric speakers) was the only distinguishing factor between normal and ataxic dysarthric subjects. According to Duffy (1995), instability in intensity and pitch during vowel prolongation were some of the most deviant speech characteristics of ataxic dysarthria.

Kent et al., (1997) and Kent et al., (2000) also reported increased long-term variability of amplitude (vAm) and long-term variability of fundamental frequency (vFO) for cerebellar group compared to the normal control group. Cannito and Marquardt (1997) observed that overshoot or undershoot of pitch and loudness could be a reflection of the underlying dysmetria in ataxic dysarthria. Gremy, Chevrie-Muller and Garde (1967) and Kent et al., (2000) reported some gender based differences based on acoustic parameter of jitter. They observed increased jitter in females compared to male subjects with cerebellar atrophy.

Phonatory dysfunction in ataxic dysarthria is also studied by observing the pattern of voice onset time in these subjects compared to normal control subjects. Voice onset time (VOT) reflects the coordination of timing between laryngeal and upper airway (Smith, Hillenbrand & Ingrisar, 1986). Extensive reports are available on VOT measures in ataxic dysarthria (Gandour & Dardarananda, 1984; Ivry & Gopal, 1993; Ackermann & Hertrich, 1997). Kent et al., (1997) reported prolonged VOT's (for the syllables /ta/ and /ka/) in cerebellar dysarthria subjects when compared to normal controls. They also noticed larger standard deviations for the cerebellar group than the control group, especially at fast rate of speech. Ackermann and Hertrich (1997) stated that variability in VOT is due to impaired temporal computations (increased overall duration) and not due to central timing abnormalities.

VOT in voiced and voiceless sounds in the speech of cerebellar dysarthria subjects is also addressed. Kent et al., (1979), observed longer VOT's for all voiceless stop consonants, in cerebellar dysarthrics with degenerative causes. In subjects with diffuse cerebellar lesions, when repetitions of initial stop consonants were produced as part of a word (Gandour & Dardarananda, 1984) or syllable (Ivry & Gopal, 1993), the average VOT's tended to be longer for all stop consonants. The increased VOT was especially noticeable for voiceless aspirated stops. Some studies report that the categorical separation of VOT for voiced and unvoiced stop consonants (Ackermann, Graber, Hertrich and Daum, 1999) is reduced in ataxic speech. Ackermann and Hertrich (1997) reported on the VOT values for voiced and voiceless consonants in ataxic dysarthric subjects with diffuse lesions based on the severity of dysarthria. They observed distinct VOT

values in subjects with mild insult, whereas subjects with severe dysarthria showed complete assimilation of voiced and voiceless stops.

Using both acoustic and perceptual methods, Hertrich et al., 1998), studied gender specific differences in subjects with cerebellar atrophy. They found significantly increased values on almost all parameters, particularly those involving long-term instability scores ('pitch' and 'loudness fluctuations,' 'quiver', vFo and vAm) in females, but soft phonation index was remarkably low in female subjects compared to male subjects. In the perceptual domain, harshness was reportedly more prominent in males and harshness, breathiness, strained, quivering, pitch and loudness fluctuations were more prominent in females.

In summary, the acoustic and perceptual studies on ataxic dysarthria indicate inconsistency in the findings of laryngeal measures. In the reported studies, increased long and short term pitch and loudness fluctuations and reduced FO were observed in ataxic dysarthria. The phonatory deviations, in general, may be due to underlying dysmetria in subjects with cerebellar lesion.

8.3. Resonatory dysfunction in ataxic dysarthria

Resonatory dysfunction evidenced as hypernasal speech and nasal emission is not a commonly reported feature of ataxic dysarthria (Murdoch & Theodoros, 1998). Duffy (1995) studied cerebellar subjects with nonfocal lesions and reported rare occurrences of mild hyponasality. This was attributed to improper timing of velar and articulatory gestures for the utterance of nasal consonants.

8.4. Articulatory and prosodic dysfunctions in atoxic dysarthria

Breakdown in articulatory and prosodic aspects of speech are most prominent in ataxic dysarthrias (Darley et al., 1969a; 1969b). Improper formation and separation of individual syllables in speech due to imprecise articulation leads to reduced speech intelligibility in ataxic dysarthrics. This is also associated with prosodic disturbances seen in the form of loss of texture, stress, tone and rhythm of individual syllables (Murdoch & Theodoras, 1998).

Articulatory and prosodic errors include articulatory inaccuracy (comprised of imprecise consonants, irregular articulatory breakdowns and vowel distortions), prosodic excess (comprised of excess and equal stress, prolonged phonemes, prolonged intervals and slow speech rate), and phonatory-prosodic insufficiency (comprised of harshness, monopitch and monoloudness). Brown et al., (1970) described these errors as a byproduct of ataxia of respiratory and oral-buccal-lingual musculature, slow movements and hypotonia. Zyski and Weisiger (1987) and Zeplin and Kent (1996) replicated Darley et al., (1975) study and attempted to identify the salient perceptual dimensions of ataxic dysarthria. Zyski and Weisiger (1987) identified irregular articulatory breakdowns, vowel distortion and excess and equal stress as characteristic features in cerebellar dysarthria. Zeplin and Kent (1996) identified task based differences and found that the features of imprecise consonants and excess and equal stress were salient in syllable repetition and passage reading, whereas monopitch and monoloudness

were salient in a passage reading task. Enderby (1983, 1986) examined the performance of ataxic speakers on the Frenchay Dysarthria Assessment battery and expressed that the perceptual correlates of articulatory and prosodic inadequacy like poor intonation, poor tongue movement including alternating movement, lateral movement and elevation of the tongue in speech, reduced rate of speech and poor movement of the lips including the alternating movements were more prominent than the other features.

Perceptual evidence for articulatory and prosodic errors has been observed as a function of linguistic task, using different speech stimuli which included (a) single word tasks (b) diadochokinetic task, (c) sentence and conversation tasks

(a) Single word tasks

Perceptual speech errors at word level in ataxic dysarthria have been addressed by several investigators (Zyski & Weisiger, 1987; Odell, McNeil, Rosenbek & Hunter, 1991; Zeplin & Kent, 1996; Kent et al., 1997). Cerebellar subjects demonstrated more errors in the noninitial position of words and substitution errors were more than distortion errors (Odell et al., 1991). Zyski and Weisiger (1987) and Zeplin & Kent (1996) observed distortion of vowels and imprecise consonants in words of ataxic dysarthrics. Mc Neil, Odell, Miller & Hunter (1995) observed that location of errors in words was consistent from trial to trial and overall, there was reduced variability of error type seen in ataxic dysarthria. In a word intelligibility task reported by Kent et al., (1997), error pattern analysis revealed very few errors in the form of voiced-voiceless contrast in syllable initial position in subjects with ataxic dysarthria.

(b) Diadochokinetic task (DDK)

According to Kent et al., (1997) and Kent et al., (2000), DDK tasks may be universally used to characterize ataxic dysarthria as they are insensitive to language properties. Diadochokinetic tasks involve either alternating motion rate (AMR) and / or sequential motion rate (SMR). AMR tasks include rapid syllable repetition of syllables /pa/, /ta/ and /ka/ and SMR tasks include rapid repetition of syllable sequence /pataka/ (Tjaden & Watling, 2003). Darley et al., (1975) found that syllable repetition rates of ataxic speakers were slow and irregular compared to the subjects with other types of dysarthria.

Variability in repetition rate, loudness and pitch in DDK task is considered a crucial feature in the diagnosis of ataxic dysarthria (Brown et al., 1970; Chenery et al., 1990; and Murdoch & Theodoras, 1998). Ziegler and Wessel (1996) reported a good correlation between the absolute rate in DDK, speech intelligibility and severity measures. They found that, the rate of DDK repetition accounted for 59% of variance in speech intelligibility and 69% of variance in speech severity ratings.

(c) Sentence recitation and conversation tasks

Scanning or staccato rhythm, altered stress patterns and irregular articulatory breakdowns are some of the salient features observed in ataxic dysarthria in tasks of conversation or sentence recitation (Brown et al., 1970). However, the extent to which each of these symptoms are prominent in the speech of ataxic dysarthria is reported to be different across studies. Brown et al., (1970) and Kluin et al., (1988) described the feature of excess and equal stress to be

highly prominent in ataxic dysarthria whereas Ackermann et al., (1992) observed irregular articulatory breakdown as the core speech error.

In general, inconsistency in the perceptual findings of ataxic dysarthria are high and these differences are often explained as due to variations in the severity or etiology of the disease, differences in the languages studied, time post onset, different degrees of involvement of the brainstem and other factors.

Sheard et al., (1991), reported a high intercorrelation between the identified perceptual speech dimensions (imprecise consonants, excess and equal stress, irregular articulatory breakdown, distorted vowels and harsh voice) in ataxic dysarthria. Inspite of this, Sheard et al., (1991) also observed that variability in perceptual rating of speech dimensions were subject to listeners' bias which inturn could affect the rating of speech dimension. Due to this and other variables, perceptual analysis alone of speech in ataxic dysarthria is not considered to be adequate. Kent et al., (1997) observe that "inconsistencies in perceptual description heighten the need for quantitative acoustic analysis of the speech disorder associated with cerebellar disease and although some progress has been made in this area, the need remains for an efficient and sensitive set of acoustic measures. In this direction, number of investigators (Ziegler & Wessel, 1996; Kent et al., 1997; Kent et al., 2000) have used either acoustic and / or physiological procedures to study the articulatory and prosodic errors of ataxic dysarthria.

8.4.1 Acoustic analysis of articulatory errors in atoxic dysarthria

Deficits in speech timing in the form of increased duration of phonemes, syllables, words and sentences are often reported in subjects with cerebellar disease (Kent & Netsell, 1975; Schonle, Dressier, & Conrad, 1990; Ackermann & Hertrich, 1993; 1994; Kent et al., 1997). Acoustic studies by Ackermann et al., (1995) and Kent et al., (1997) revealed abnormal transitions from consonants to vowels and reduced speech rate in some of the subjects with cerebellar damage. Increased articulatory duration with variable velocities and abnormal timing patterns of muscle force development required for speech were cited as reasons for the slow rate in ataxic dysarthria (Kent & Netsell, 1975; Hirose, Kiritani, Ushijima & Sawashima, 1978; Kent et al., 1979; Ackermann & Hertrich, 1993, 1994; Kent et al., 1997). Reports also suggest a task dependant effect in cerebellar subjects, i.e. longer utterances are more affected than single-word productions in cerebellar pathology (Kent et al., 1979; Kent et al., 1997).

a) Vowel errors in ataxic dysarthria: Duration of both tense and lax vowels in ataxic speech is often reported to be increased, but the lax vowels are reportedly more prolonged than tense vowels (Kent et al., 1979; Kent & Rosenbek, 1982; Ackermann et al., 1995). Formant structure is also addressed by few studies. Kent et al., (1979) observed no significant difference in the formant structures of vowel in a CVC syllable structure produced by normal controls and ataxic dysarthric subjects. However, the ataxic subjects exhibited prolonged syllable durations and formant transitions when compared to normal subjects. This was reasoned as due to time availed by dysarthrics as a strategy to reach the vowel target positions and for correct production of the vowels. Kent and Netsell (1975) and

Netsell and Kent (1976) analysed the articulatory position and movements in ataxic subjects using cineradiography procedure. They observed that the movements of the lips, tongue and jaw were coordinated in ataxic dysarthria. However, they observed that poor and slow adjustments of anterior-posterior tongue movements during vowel production characterized ataxic dysarthria and they attributed this to the perceived vowel distortions.

b) Syllable duration in ataxic dysarthria: Prolonged CVC syllables (Kent et al., 1979) and utterance durations (Ackermann & Hertrich, 1994) have been reported as a cause for reduced speech rate in ataxic dysarthria. Charcot (1877) used the term 'scanning speech' to characterize the speech of individuals with multiple sclerosis with cerebellar lesions. Scanning speech is reported to be the most perceptible and easily recognized speech characteristic associated with subjects with cerebellar disease (Kent & Netsell, 1975; Kent et al., 1979). The 'scanned' or 'staccato' speech in ataxic dysarthria is described as including (a) excess and equal stress (b) prolonged phonemes & (c) slow rate. According to Kent et al., (1979), this is more evident in the form of a syllable-by-syllable motor production control strategy. This is subsequently associated with increased duration of the unstressed syllable in ataxic dysarthria (Kent et al., 1979).

The presence of 'scanned speech' in ataxic dysarthria has been investigated acoustically, and an index suggested explaining the variability of syllable duration on a temporal scale (Ackermann & Hertrich, 1994; Hartelius et al., 2000). They suggest that this derived measure helps to infer information on

'syllable isochrony' in ataxic dysarthrics, evidenced as equalized syllable durations which is the perceptual dimension of scanned speech.

The scanning index (SI) is computed using the formula given by Ackermann and Hertrich (1994) as follows

$$SI = \frac{S1 \times S2 \times S3 \times \ldots\ldots\ldots\ldots Sn}{[S1 \times S2 \times S3 \times \ldots\ldots\ldots\ldots Sn/n]n}$$

Where, S= duration of the syllable & n= number of syllables considered.

Provided that all of the syllables have equal length, the index amounts to unity or '1'. If the speech is variable or less 'scanned', then the index should be less than unity or '1'.

Few investigations were conducted on the speech of ataxic dysarthria using the measure of SI (Ackermann & Hertrich, 1994; Hartelius et al., 2000). These studies concluded that ataxic dysarthric subjects show reduced speech rate in terms of syllable duration as well as utterance durations. They also noticed a tendency for syllable isochrony for certain measures like intrautterance variation and SI .The utility of SI has however been questioned by Kent et al., (1997), because even if one of the syllables is shorter than the others, the SI shows a value less than one.

c) Durations and formant structure in longer utterances (words, phrases, sentence and conversation): Longer word duration (Hertrich & Ackermann, 1999), uniform lengthening of the consonant clusters and nucleus portion of the words (Kent et al., 1979) are commonly reported in ataxic speech in comparison to normal controls. More recently, Liss,

Spitzer, Caviness, Adler & Edwards (2000) observed significantly longer phrase durations in ataxic speech when compared to control subjects. The range of formant frequency in sentences and conversation were reported to be similar to normal speaking subjects by Kent et al., (2000) and Kent et al., (1997) respectively. It was explained that the slow speaking rate typical of speakers with cerebellar disease facilitated increased time required for greater amplitude of articulatory movements and hence the formant frequency ranges matched with that of normal subjects. Kent et al., (2000) compared syllable rates for AMR, sentence recitation and conversation and found that syllable rate was reduced for sentence recitation and conversation compared to AMR. They however, reported that the difference was not significant between the three tasks.

8.4.2. Variability in repeated speech tasks in ataxic dysarthria

Increased variability of syllable durations have been reported in ataxic spee- ch during rapid syllable repetitions (Kent et al., 1979; Portnoy & Aronson, 1982; Hirose, 1986). Increased variability following cerebellar damage have also been reported in the control of limb and speech movements with respect to direction, force, velocity, amplitude, rate, movement onsets, and terminations (Hallet et al., 1991; Schonle & Grone, 1993; Ackermann & Hertrich, 1994). Findings and inferences have differed across the studies. Variability was reported to be increased in slower limb movements and speech movements (Crystal & House, 1988a, 1988b). Contrary to this observation, Kent et al., (1997) found increased variability during fast syllable repetition in cerebellar subjects and

attributed this to an inherent inability of the cerebellar subjects to achieve a faster rate.

8.4.3. DDK measures in atoxic dysarthria: Acoustic correlates

Ataxic dysarthrics often exhibit deviant characteristics in diadochokinetic (DDK) task (Kent et al., 1997; Kent et al., 2000; Ozawa et al., 2001). Maximum syllable repetition rates of ataxic speakers were typically slow and irregular compared to those of speakers with other types of dysarthria (Darley et al., 1975). Diadochokinetic measures (alternating motion rates) in ataxic dysarthria showed a differential lengthening effect of segment durations, that is a differential contribution of the syllable duration or the gap duration to total duration (which is a total of the syllable duration and the closure duration) (Ozawa et al., 2001). Ozawa et al., (2001) operationally defined syllable duration in the AMR task as the period ranging from stop burst to vowel formant offset and gap duration as the period ranging from stop burst to vowel formant offset. Total duration was considered as the total of the syllable duration and gap duration. Ozawa et al., (2001) observed that the reduced mean total duration in the ataxic dysarthric subjects correlated with gap duration. Kent et al., (1997) compared the diadochokinetic rates (alternating motion rates) in ataxic dysarthric subjects and normal control subjects. They studied the pattern of changes in voiceonset time, vowel duration and intersyllable gap duration in both the groups. They reported that the control subjects showed abnormally long vowel durations in the syllable compared to intersyllable duration and voice onset time, whereas, ataxic dysarthric subjects showed a different trend with maximum duration for intersyllable duration followed by vowel duration and voice onset time. Much

later, Kent et al., (2000) attributed the slow rate of speech in ataxic dysarthric subjects to lengthening of both syllable and intersyllable durations. However, they cautioned that the variability was high for both syllable and intersyllable durations.

Another aspect that has been addressed with regard to diadochokinesis in ataxic dysarthric subjects is a differential involvement of different syllables (syllable /pa/ which is a lip related syllable and syllables /ta/ and /ka/ that are principally tongue related) in alternating motion rate task. According to Kent et al., (1998), the extent of involvement of peripheral nerves and the different neural regulatory loops involved in the production of each syllable varies, leading to different manifestations in speech diadochokinesis. Kent et al., (1997) reported that the ability to produce faster repetition rates was affected in the cerebellar group, especially for syllable /ka/. Ozawa et al., (2001) reported that tongue related sounds (/ta/ and /ka/) were more prolonged than the other sound (/pa/) in subjects with degenerative cerebellar disease. Ziegler (2002) reported that among the three syllables, /pa/ was fastest, followed by /ta/ and then /ka/; and cerebellar subjects showed increased temporal variability of syllable repetition. This is consistent with the findings reported in the studies by Kent et al., (2000) and Ziegler and Wessel (1996).

Few studies report variable amplitude minima and maxima in ataxic subjects compared to normals in diadochokinetic tasks (Tatsumi, Sasanuma, Hirose & Kiritani, 1979; Kent et al., 2000). Hence, Kent et al., (2000), stress the

need to include measures of amplitude variability along with the study of diadochokinetic rate and temporal variability in ataxic dysarthrics.

8.4.4. Duration of short and long vowels

A variety of other durational parameters are reported to be affected in the speech of cerebellar dysarthrics, one of them being the relative duration of phonologically short and long vowels [vowel length contrasts (VLC)] (Klatt, 1976). Phonological vowel length contrasts provide insight into supraglottal-laryngeal coordination. It principally requires control of the phonatory subsystem for adequate production of vowel contrasts that are phonologically relevant (Caruso & Burton, 1987). Ackermann et al., (1999), also observed poor vowel length contrasts in subjects with diffuse cerebeliar lesions and idiopathic cerebeliar atrophy. This is in contrast to the observation by Gandour and Dardarananda (1984), who reported that despite markedly prolonged vowel durations, the temporal relation of short and long vowel targets were maintained in a Thai speaker with ischaemic cerebeliar lesion.

8.4.5. Articulatory -prosodic errors in ataxic dysarthria

Prosodic characteristics in ataxic dysarthria are often described as either prosodic excess or as prosodic insufficiency (Darley et al., 1969a; 1969b). The dysprosody in ataxic dysarthria is also manifested in the form of (a) generally flat fundamental frequency (b) increased / decreased rate of speech (c) short phrases (Kent et al., 1979; Gentil, 1990a).

The perception of speech intensity is acoustically reflected in RMS energy while speech pitch is reflected in mean FO and FO range. Increases in RMS energy and FO are reflective of emphasis or stress in normal speech. According to Schlenck, Bettrich & Willmes (1993) mean FO is the acoustic equivalent to what is perceived as pitch and FO range corresponds to degree of monotony. Darley et al., (1969b) reported monopitch and monoloudness as characteristic of ataxic speech and this was acoustically reflected as variable and decreased FO range and intensity values. Kent et al., (2000) did qualitative analyses of unintelligible episodes in the conversation of ataxic subjects. They reported that these samples had a well defined syllable pattern, but the acoustic contrasts of consonant and vowel sequences within the syllable were not maintained in all the subjects. In addition, deficits in intelligibility were observed even in the presence of very distinct acoustic patterns in some ataxic subjects. Thus it was presumed that perceptual feature of unintelligibility may or may not be associated with abnormalities in acoustic patterns.

The fundamental frequency in the conversation of ataxic dysarthric subjects was reported as monotonous or consisting of excessive variation (Kent & Netsell, 1975). Kent et al., (1997) and Bunton, Kent, Kent & Rosenbek (2000) were of the opinion that cerebellar subjects often showed poor FO regulation in conversation, which was considered to be a reflection of poor cerebellar control. Kent and Kent (2000) also support the notion that cerebellum contributes to dysprosody, in the form of poorly regulated intonation and loudness, that is often evidenced in ataxic dysarthria. Bunton et al., (2000) addressed the contribution of prosodic features such as tone unit (words, duration) and F0 variation in speech

intelligibility of ataxic dysarthria subjects with diffuse lesions. They showed that, compared to normal control group, the ataxic dysarthric subjects presented more number of tone units and reduced duration of tone units. Moreover, the variations of FO in conversation were reduced in ataxic dysarthric subjects compared to normal control subjects. They also noticed a concomitant reduction in speech intelligibility when the intonation contours were flattened through acoustic synthesis. Reduction in FO range compared to normal control subjects has also been reported in other dysarthria types by Canter (1963); Kent and Rosenbek (1982) and Schlenck et al., (1993). A possible reason for the reduced FO range could be derived from the physiological basis, wherein factors such as declining subglottal pressure (Collier, 1974), tracheal pull (Maeda, 1976), or aspects of laryngeal FO control (Strik & Boves, 1992) might act to bring about a decrease in FO range. However, there is a raging controversy regarding whether it is the laryngeal muscles, or change in subglottal pressure, which controls FO (Ohala, 1978; 1990; Ladd, 1984). Spectrographic analysis of repeated phrase utterances of a single case with ataxic dysarthria by Kent & Netsell (1975) revealed monotonous as well as marked variations in FO pattern. These deviations were attributed to generalized hypotonia seen in ataxic dysarthria, characterized by delayed generation of muscul- ar forces, reduced rate of muscular contraction and reduced range of movements, as is required for normal speech.

The characteristics of speech in ataxic dysarthria, as reviewed in the previous sections reveal evident inconsistencies in reported features. The following sections present the reasons for this and report the studies which speculate the possibility of subgroups in dysarthria, in general.

9.0. Subgroups in dysarthria

Only a 'single form' or umbrella term of a disorder comes into picture during the initial descriptions of any disorder (Kent et al., 1998). Considerable nonhomogenity seems to be reported for various disorders. In this context, the use of a 'single form' or umbrella term does not reveal much about the characteristics associated with any disorder. Because of this heterogeneity, attempts to recognize subgroups come into picture. Ataxic dysarthria is no exemption to this.

The possibility and the question of subgroups in dysarthria has arisen due to factors such as (a) variation in neuropathology among patients who have the same medical diagnosis and / or the same classification of dysarthria (b) variation in subsystem involvement among patients who have the same classification (c) severity of impairment (d) gender and age (e) differences in time post onset etc. Preliminary findings reveal that subgroups may be present in all dysarthria types. Possibility of subgroups in flaccid dysarthria (Kent et al., 1998), spastic dysarthria (Delwaide & Young, 1985), mixed spastic-flaccid dysarthria (Shiffer, Brignolio, Chio, Leone, & Rosso, 1986), hypokinetic dysarthria (Poewe & Wenning, 1996) and hyperkinetic dysarthria with dystonia (Brin, Fahn, Blitzer, Ramig, & Stewart, 1992) have been metioned in literature. Few studies have deliberated upon the possibility of subgroups in ataxic dysarthria. The findings on subgroups may be useful for clinical description and theoretical interpretation.

9.1. Subgroups in atoxic dysarthria

The basic assumption for the presence of subgroups in ataxic dysarthria arises from the inconsistencies that are reported in the characteristics associated with ataxic dysarthria, studied both perceptually and acoustically.

Ackermann and Ziegler (1992) listed the primary speech disturbances in ataxic dysarthria as: articulatory movements, increased variability of pitch and loudness, monotonous, scanning speech and articulatory imprecision. It is noteworthy that these findings are language insensitive and similar descriptions of ataxic dysarthria are reported in subjects speaking different languages and dialects (Chenery et al., 1990; Duffy, 1995). Most studies implicated disruptions in phonatory and articulatory levels of speech. Despite this apparent universality of the major feature of ataxic dysarthria, there were also indications of individual variations in the speech disorder. The variability of speech symptoms among individuals with ataxic dysarthria was cited by Cannito & Marquardt (1997) as "there are sufficient discrepancies in findings, across studies and between patients within studies, of the speech characteristics of (ataxic dysarthria) to suggest the likelihood of differential impairments contributing to the variability of the dysarthrias exhibited by different (ataxic dysarthric) subjects." Using oscillographic and electroglottographic methods, Gremy et al., (1967), identified a severe form and a moderate form of ataxic dysarthria. The severe group had laryngeal irregularities, bradylalia and specific impairments of articulation (e.g., devoicing of voiced stops and exaggerated explosions of voiceless stops). Reduced phonetic contrasts were the only feature noticeable in the moderate group. Joanette and Dudley (1980), identified two distinct factors that related to

articulation and phonation, in their subjects with Friedreichs ataxia. "The general dysarthric factor", had the dimensions of imprecise consonants and prolonged phonemes as core features. The other factor called "phonatory stenosis", had the voice dimensions of harshness, pitch breaks, and pitch level as core features. In another group of subjects with Friedreichs ataxia, task based differences were reported in terms of inspiration/ expiration repetition task and a syllable repetition task (Cisneros & Braun, 1995). Both the tasks were affected in the group with severe involvement and with evident signs of atrophy of medullary and pontine structures. This was characterized by both respiratory insufficiency and articulatory / phonatory difficulties.

Speech characteristics of scanning or staccato rhythm, altered stress patterns and irregular articulatory breakdowns are not consistently reported in literature on ataxic dysarthria. The feature "excess and equal stress" was highly prominent in descriptions by Brown et al., (1970) and Kluin et al., (1988), but Ackermann et al., (1992) emphasized irregular articulatory breakdown as the core speech disturbance in ataxic dysarthria. Similarly, Ackermann et al., (1992) identified inconsistent articulatory deficits and slow speech tempo as the most common features in ataxic subjects. It was not clear if these differences could be explained by variations in severity or etiology of the disease, differences in the languages studied, retrospective design of studies, absence of a standard evaluation procedure for dysarthria, or some other factor, Sheard et al., (1991), obtained perceptual ratings of ataxic dysarthria for the five dimensions of imprecise consonants, excess and equal stress, irregular articulatory breakdown, distorted vowels and harsh voice. They observed substantial variation in

interjudge agreement across dimensions, with poorest agreement on the dimensions of irregular articulatory breakdown, distorted vowels and harsh voice. Sheard et al., (1991) reported that high intercorrelations between rated dimensions may be reflective of the fact that these dimensions were not perceptually independent. Based on this Sheard et al., (1991) concluded that: " the actual rating values assigned to apparently separate dimensions may infact reflect an overall perception of a number of concurrent, crucial / salient speech characteristics". These intercorrelations may contribute to the inconsistent findings in ataxic dysarthria.

The results of acoustic studies of ataxic dysarthria generally have confirmed the speech deviations uncovered in perceptual studies (Ludlow & Bassich, 1983). Boutsen et al., (1997) and Boutsen et al., (1997), reported that people with ataxic dysarthria produced abnormally slow and variable repetition rates. The results suggested that variability and rate of diadochokinetic performance may be two independent speech dimensions in ataxic dysarthria. They identified three subgroups based on patterns obtained for durational variability and the trends obtained for the syllables /pa/, /ta/ and /ka/. Uniform durational variability across utterance types characterized one group. Other two groups were identified based on the fact that the syllables /ta/ and /ka/ had increased durational variability compared to syllable /pa/. The findings were reasoned to reflect different subsystem impairm- ents. Ackermann and Hertrich (1994) investigated the possibility of subgroups by studying chronic and acute ataxia. They measured the tendency towards scanning in a sentence production task. Scanning was defined as the absence of durational variability between

syllables. Across subgroups, half of the cerebellar patients had a scanning index that was higher than the normal range. They reported that the scanning index of patients with acute and chronic ataxic dysarthria however was similar to each other.

In general, data from perceptual and acoustic studies indicate that slow speech rate and durational variability are common in subjects with ataxic dysarthria. They also suggest that these speech deviations may occur independently or together. That is, the association of slowness with dysrhythmia, dysrhythmia with slowness, or both may reveal the possibility of different subtypes.

It appears that many of the differences reported in the literature could reflect differential impairments in the subsystems of speech. Kent et al., (2000) stressed about the importance of identifying subgroups within the general category of ataxic dysarthria and how these subgroups relate to site of lesion or pathologic differences. They also opine that identification of subgroups may be important for clinical assessment and management. There is increasing evidence put forth to suggest that only some anatomical regions in the cerebellum could be involved in speech motor control (Amici et al., 1976; Lechtenberg & Gilman, 1978; Amarenco et al., 1991; Ackermann et al., 1992; Urban et al., 2003). It can be concluded from the literarture that although ataxic dysarthria has been investigated by a variety of methods in several different languages, there are inconsistent reports on the salient characteristics associated with this disorder. In particular, further work is needed to identify the most consistent features of the

disorder. It is also necessary to establish quantitative criteria which can help in identifying and describing various speech characteristics. Most of the studies on ataxic dysarthria do not provide a detailed description or classification of abnormal speech characteristics. Due to this ataxic dysarthria is usually reported in the literature as a 'single form' of disorder. This may mask the difference in speech characteristics, if any, based on different lesion sites. The perceptual and acoustic characteristic of ataxic dysarthria generally reported in the literature is not sufficient to give a wholistic view of ataxic dysarthria. These studies do not include and typically lack clear descriptions of deviant speech characteristics or classification of ataxic dysarthria, use perceptual attributes alone for the characterization of speech features, include subjects with degenerative changes or diffuse cerebellar lesions, use only limited acoustic measures, and select tasks that are language sensitive, use retrospective study designs and are inconsistent in the use of standard evaluation tools for dysarthria.

Moreover, ataxic dysarthria also can be portrayed with respect to task-based profile, in which functional deficits are described for specific aspects of speech production (Kent & Kent, 2000). Thus, from the available literature it is clear that more studies are required using a variety of speech tasks to describe the subsystem errors using acoustic as well as perceptual measures. There is a need for studies to identify and quantify the voice and speech dimensions in ataxic dysarthria caused due to lesions in various sites of the cerebellum, using acoustic and perceptual methods to help in differentiating subgroups if any in ataxic dysarthria.

METHOD

Ataxic dysarthria caused due to cerebellar lesion is now mostly recognized as a nonunitary disorder (Boutsen et al., 1997; Kent et al., 2000). The cluster of signs and symptoms of ataxic dysarthria are reported to be varied and this has been attributed to the different functional role of regions in the cerebellum for speech motor control. Further, the speech subsystems of respiratory, phonatory, articulatory and resonatory mechanisms are also attributed to different regional control by the cerebellum (Amarenco et al., 1991; Ackermann et al., 1992; Duffy, 1995;Urban et al., 2003). These findings are based on acoustic and perceptual analysis of speech in subjects with variable etiology and nonfocal lesions in the cerebellum. This study attempts to investigate the voice and speech profiles of subjects with lesions in specific sites of the cerebellum. It aims to understand the localization of speech characteristics and the differential subsystem involvement (phonatory, articulatory and prosody) through acoustic and perceptual analysis of voice and speech of subjects with lesions in specific sites of the cerebellum.

Aims of the study:

- To analyse and differentiate some aspects of voice and speech in selected speech tasks in subjects with ataxic dysarthria due to lesions in various sites of the cerebellum using acoustic and perceptual analysis.
- To compare the results obtained in individuals with ataxic dysarthria against that of normal control group.

The study uses a cross sectional standard group comparison design.

Hypothesis:

- It is hypothesized that there would be a difference in voice and speech characteristics of subjects with cerebellar lesions when compared to that of normal control subjects.
- It is hypothesized that there would be differences in voice and speech chaeacteristics between groups of subjects with lesions in different cerebellar loci [left superior paravermal (LSP), left anteroinferior (LAI), superior vermis (SV), right superior paravermal (RSP), right posterosuperior (RPS) and right anterosuperior (RAS)].

Subjects

Experimental group

Seventeen subjects with ataxic dysarthria due to lesions restricted to various sites in the cerebellum were included in the study. The subjects were selected based on neurological evaluation and diagnosis by a neurologist/ neurosurgeon/ neuroradiologist. The neurological evaluation was also supported with findings from neuroimaging investigations [(Computerized tomography (CT) & / or magnetic resonance imaging (MRI)]. The subjects fulfilled the following criteria:

a) They were diagnosed as having cerebellar tumour as the neuropathology along with the presence of dysarthric speech.
b) Absence / presence of dysarthria was tested by the experimenter on a proforma for dysarthria evaluation and proforma for speech examination which is a standard proforma used routinely in the clinical evaluation by Speech - language pathologists at the centre of study. The presence of

dysarthria was also confirmed by perceptual evaluation of the speech samples of subjects by another experienced speech - language pathologist.

c) They were in the age range of 20 to 51 years.

d) They were native speakers of Malayalam language which is a Dravidian language spoken in Kerala state in India.

e) The education qualification of the subjects varied from matriculation to graduation.

f) They had normal hearing threshold as tested by an Audiologist.

g) They did not have history of any other neurological illness or other type of speech problem, as confirmed by the Neurologist and Speech-language pathologist respectively.

h) The speech and voice samples of the subjects were recorded within 4 months of the onset of dysarthria.

i) All subjects were subjected to computerized tomography and / or magnetic resonance imaging investigations and the evidence for lesions in the cerebellum was established.

j) The severity of dysarthria was judged perceptually based on a 7 -point rating scale by three experienced judges (experimenter and two postgraduate students of speech pathology). The rating of 1 = normal (0% of dysarthric characteristics), 2 = mild (Just barely dysarthric < 10 % of dysarthric characteristics), 3 = mild to moderate (15 to 30 % dysarthric characteristics), 4 = moderate (30 to 50 % dysarthric characteristics), 5 = moderate to severe (50 to 70% dysarthric characteristics), 6 = severe (70 to 90 % dysarthric characteristics), 7 = Profound (> 90% dysarthric characteristics) was used as per Langmore & Lehman (1994). All the

dysarthric subjects had mild severity except for two dysarthric subjects with mild to moderate severity. All three judges agreed to this rating of severity.

Subjects with the following characteristics were not included in the study:

a) Subjects who had diffuse lesions involving the brainstem structures along with cerebellum.
b) Subjects who were using artificial dentures and who had no teeth.
c) Subjects who had any premorbid history of psychological problems as revealed through case history and bedside testing.
d) Subjects who had upper respiratory infection at the time of speech sample recording.
e) Subjects who had cognitive impairment as screened on Mini - Mental State Examination Scale (Folstein, Folstein & McHugh, 1975).
f) Subjects who had language impairment as screened on Western Aphasia Battery in Malayalam (Philip, 1992).
g) Subjects who had cerebellar etiology other than tumour.
h) Subjects who had associated medical conditions like hydrocephalus, ventricular compression, post surgical lesions etc.

The site of lesion within the cerebellum of the seventeen subjects were recorded based on CT & / or MRJ and the same was confirmed by three neurologists, three neurosurgeons and three neuroradiologists based on the neurological examination and reports of CT or MRI (Appendix 1). Based on the site of lesion, the experimental subjects were grouped into six groups. The neurological / CT or MRJ findings, pathophysiology and diagnosis of the subjects

in the experimental group are given in Appendix 1. The demographic details of the subjects are given in Table 3.

Table 3: Demographic details of the subjects

Groups	Subject	Age/ Sex	Site of lesion in the cerebellum	Neurodiagnosis & type of tumour in the cerebellum
I	OK	29/F	Left superior paravermal (LSP)	Cavernous angioma
	NB	23/F	Left superior paravermal (LSP)	Medulloblastoma
	TJ	45/F	Left superior paravermal (LSP)	Venous angioma
II	JA	37/F	Left anteroinferior (LAI)	Tuberculoma
	ST	27/M	Left anteroinferior (LAI)	Lymphoma
III	MK	36/M	Superior vermis (SV)	Medulloblastoma
	VN	27/M	Superior vermis (SV)	Astrocytoma
	BT	50/F	Superior vermis (SV)	Adenocarcinoma
	HR	42/F	Superior vermis (SV)	Pilocytic astrocytoma
	SD	25/F	Superior vermis (SV)	Hemangioblastoma
IV	SP	42/F	Right superior paravermal (RSP)	Astrocytoma
	ST	39/F	Right superior paravermal (RSP)	Cavernous angioma
	OK	31/F	Right superior paravermal (RSP)	Pilocytic astrocytoma
V	N	34/M	Right posterosuperior (RPS)	Hemangioblastoma
	JT	46/M	Right posterosuperior (RPS)	Tuberculoma
	RP	51/M	Right posterosuperior (RPS)	Hemangioblastoma
VI	RN	23/M	Right anterosuperior (RAS)	Astrocytoma

The data were collected from neurology / neurosurgery departments in four major hospitals in Kerala State. Informed consent in writing was obtained from all the subjects prior to the study. The subjects were explained the purpose and the nature of the study before taking the consent. The data was collected from these seventeen subjects over a time span of 21 months.

Control group:

A group of 30 normal control subjects, matched in age and gender to the experimental group were included in the study. This included two control subjects matched to each of the experimental subjects. Experimental subjects ST and VN

and subjects HR and SP matched in age and gender and hence only two age and gender matched controls were matched for each of these pair. This was carried out to establish confidence intervals for various tasks selected for the study. A total of 12 males and 18 females were selected as the control group. Normal subjects were in the age range of 23 years to 51 years with a mean age of 37.5 years. The criteria for selection of these subjects were as follows:

a) They were screened for any history of hearing or speech problem.

b) They had no history of any neurological disease.

c) They had no history of laryngeal pathologies and had no upper respiratory infections / pathology at the time of recording of the speech sample.

d) The education qualification of the subjects varied from matriculation to graduation.

e) They were native speakers of Malayalam language which is a Dravidian language spoken in Kerala State in India.

Material

1) Proformafor assessment of dysarthria

A detailed history was collected from each experimental subject by the investigator. The history included relevant information regarding the onset of speech problem, disease progression, family history, previous history of illness and other complications. The structure and functions of speech mechanism was examined including the vegetative functions, speech functions (voice, articulation, prosody), reading and writing skills and speech intelligibility. The findings were also confirmed by another experienced Speech - Language pathologist.

*2) Proforma for neurological examination **of** dysarthria*

Detailed case histories of the experimental subjects were also collected by a neurologist and / or neurosurgeon before and during the clinical examinations. Detailed case history about each subject was obtained and recorded through medical records and an interview with the subject or his / her family members. Neurological examination included evaluation of higher mental functions, cranial nerves, motor, sensory, cerebellar, spine and other functions. Symptoms like disordered stance/ gait ataxia, truncal titubation, head tilt, oculomotor abnormality, nystagmus, hypotonia, dysmetria, dysdiadochokinesia, headache, irritability, tiredness, pappilloedema, intention tremor, hydrocephalus, neck pain, poor balance, intermittent vomiting and any others were also probed.

*3) **Protocol for** voice and speech assessment*

The voice and speech samples of subjects were obtained when they performed various tasks. The tasks which were reported to be sensitive in revealing underlying region of neural control in the literature were included in the study. The task of sustained vowel phonation has been reported to be sensitive in determining ataxic dysarthric characteristics by Ackermann et al., (1992), Amarenco et al., (1993), Kent et al., (1997) and Kent et al., (2000). These studies have indicated increased short-term and long - term variability of phonation. Perceptual findings of phonatory dysregulation in ataxic dysarthria are supported by various investigators (Darley et al., 1969a, 1969b; Joanette & Dudley, 1980; Gilman & Kluin, 1992 & Hertrich et al., 1998). The measure of maximum fricative duration has not been specifically addressed in subjects with ataxic

dysarthria. The mechanisms underlying the production of voice onset time and vowel duration has been speculated to be localized to specific cerebellar areas (Boutsen & Christman, 2002). Diadochokinetic task (AMR) has also been reported to be sensitive in determining ataxic dysarthric characteristics. Various studies have specifically cited irregularities in temporal measures such as irregular syllable repetition (Duffy, 1995), slow repetition rate, irregular interval between syllables and prolonged closure duration in ataxic dysarthric subjects (Kent et al., 1979; Portnoy & Aronson, 1982; Gentil, 1990a, 1990b; Ackermann & Hertrich, 1994; Ziegler & Wessel, 1996; Boutsen et al., 1997; Kent et al., 1997). Boutsen et al., (1997) opined that variability of temporal pattern in speech diadochokinesis could be a defining characteristic of subgroups in ataxic dysarthria. Kent et al., (2000) reported slow speech rate, temporal and amplitude variability (energy minima and energy maxima) as characteristic features of ataxic dysarthria. Urban et al., (2003) expressed that diadochokinetic task could be a useful tool in revealing the underlying region of neural control. Specific findings with respect to the sensitivity of sequential motion rate (SMR) task and word repetition task are not available. There are only speculations regarding the sensitivity of longer utterances like samples obtained from a reading and narration sample (Boutsen & Christman, 2002), in determining the cerebellar control.

Hence these tasks were included in the present study and analysed using acoustic and perceptual methods. Temporal and / or spectral measures were obtained through acoustic analysis. Perceptual measures were obtained from the tasks of word repetition and narration. Phonetic transcriptions of recorded word samples were carried out and sound by sound error analysis was done in terms of

substitutions, omissions, distortions or additions. Thirty eight speech dimensions (given by Darley et al., 1975) were selected for perceptual judgement of the narrative sample.

An overview of the tasks included in the study is given in Table 4.

Table 4: Overview of the tasks and the measures obtained from these tasks

	Task	Dimensions measured		
		Spectral	Temporal	Perceptual
Phonatory tasks	Maximum phonation duration (MPD) for vowels /a/, /i/ and/u/	• Fundamental frequency • Frequency perturbation • Amplitude perturbation • Noise related	MPD(s)	
	Maximum fricative duration for /s/ and *Izl*		Maximum fricative duration and s/z ratio	
	Voice onset time (VOT) in the stimuli words */kAlAm/* and /gAlAm/		VOT for voiceless sound in the stimuli word /kAlAm/ (CVCVC) VOT for voiced sound in the stimuli word /gAl/\m/ (CVCVC)	

Articulatory tasks	Word repetition task			Analysis of phoneme articulation using SODA
	Diadochokinetic task • /pV,/Wand /kA/ in AMR • /pAtAkV in SMR	• Minimum intensity • Maximum intensity • Intensity range	• Syllable duration • Closure duration • Total duration • DDK rate	
	Duration of short and long vowels in the stimuli words /kAlAtn/ and /kA:lAm/		Duration of short vowel in the stimuli word **/kAlAm/** (CVCVC) Duration of long vowel in the stimuli word /kA:lAm/ (CVVCVC)	
Prosodic tasks	• Passage reading • Narration task		Speech rate (syll/s)	

Material developed in Malayalam

Acoustic analysis of normal speech in Malayalam language speakers is attempted by very few investigators. Most of the studies were conducted on a small group of subjects and hence there is dearth of established norms and standard scores. Studies have addressed various acoustic characteristics in Malayalam speakers such as fundamental frequency range in male and female speakers (Jose, 2000), VOT in stop consonants (Savithri & Sreedevi, 2002), duration of short and long vowels (Velayudan, 1975) and rate of speech (Savithri

& Jayaram, 2005). Standard word lists and passages are also not available, except for a diagnostic articulation test developed for children in Malayalam by Maya (1990). These facts called for preparation of material by the investigator for two tasks i.e. (a) word list and (b) Reading passage.

The word list was prepared in consultation with a qualified linguist. The word list consisted of eighty six words. A total of 28 words were prepared for testing vowels. Short vowels /a/, /i/, */u/*, */e/* and /o/ were tested in the initial (five words), medial (five words) and final (five words) positions. The schwa vowel /u/ is most frequently seen only in word final position and hence stimulus word was prepared with this vowel in the final position. Long vowels /a:/, /i:/,/u:/, */e:/* and /o:/ occur frequently in the word initial and word medial positions and were tested only in these positions (five words in the initial position and five words in the medial position). Diphthong /ai/ was tested in the initial (one word) and medial (one word) position.

Word list was also prepared for consonants in the initial position (total twenty four words) and in the medial position (total twenty eight words). Consonants do not occur in the final position in Malayalam and hence were tested only in the initial and final position. The consonants tested included bilabial stops (/p/, /p^h/, / b / & /b^h *I),* bilabial nasal (/m/), voiced labiodental frictionless consonant (/v/), dental stops *(/t/*,, /d/, /d^h/ & /t^h/), dental nasal (/n/), alveolar nasal (/n/), alveolar trill (/r/), alveolar flap (/R/), alveolar fricative (/s/), alveolar lateral (/I/), retroflex nasal (/n/), retroflex fricative (/s/), retroflex lateral approximant *(/l/)*, retroflex lateral (/|/), retroflex stop (/d,), palatal nasal (/n/), palatal fricative (/j/),

palatal affricate (/ʧ/, /ʤ/), palatal continuant (/j/), velar nasal (/n/), velar stops (/k/, /k^h/, /g/ & /g^h/) and glottal fricative (/h/). Words with consonant clusters (/ks/, /tr/, /st/ and /kr/) were also prepared. Phonemic inventory in Malayalam is given in Appendix 2.

The syllable structure of the words in the word list were of the following type: vcv, cvcv, cvvc, vvcv, cvcvc, cvvcv, cvccv, vcvcv, vcvccv, vvcvvc, cvcvcv, cvccvc, vcvcvvcv, vccvcvc, cvvcvc, cvcvvcv, cvvcvcvcvc, cvcvcvvcvc and cvcvcvcvcv. The word list is given in Appendix 3.

The selected words were also tested for familiarity. The words were written on individual cards and given to twenty adult native speakers of Malayalam. The speakers included for familiarity testing were from different regions in Kerala State and were literate (matriculation to graduation). They rated the words for familiarity based on a 4 - point scale (1= very familiar, 2 = familiar, 3 = not so familiar, 4 = not familiar). A word was retained in the final list only if 85% of the subjects rated it as very familiar. Face validity of the stimuli in the word list was checked by three experienced Speech - Language pathologists. They were asked to rate each of the words based on a binary scale (0 = agree that the word is suitable to test, 1= not agree that the word is suitable to test).

A 524 syllable passage incorporating all the most frequently occurring phonemes, consonant clusters, inflectional morphemes, words, word boundaries fused by morphophonemic alternations and words with different suffixes in Malayalam as given in Ghatage (1994) was developed by the investigator in

consultation with a qualified linguist. Care was also taken to include longer words and sentences in the reading passage. Many words were found to occur more than once in the passage depending on the semantic content of the passage. The syllable structure of the words in the passage included syllables varying in length from bisyllable to eight syllables. The passage was tested for familiarity by asking fifteen normal adult literate (matriculation to graduation) native speakers of Malayalam to rate the passage for familiarity, depending on the content, on a four point rating scale [1- very familiar; 2 - familiar, 3 - not so familiar, 4 -not familiar]. Thirteen speakers rated the passage as very familiar and two of them as familiar. Face validity of the passage was checked by three experienced Speech - Language pathologists. They were asked to rate the passage based on a binary scale (0 = agree that the passage is suitable to test, 1= not agree that the passage is suitable to test). The reading passage is presented in Appendix 4.

Recording

The voice and speech samples of the subjects were collected individually and recorded on to a Sony MZ-55 digital tape recorder. A constant microphone-to-mouth distance of 10 cm was maintained for all the subjects. The recording was carried out in a sound treated room in the respective hospitals. The recordings for each subject were carried out in two or three sittings. For each subject, it took approximately 60 to 90 minutes to record samples of the tasks selected for the study. Adequate rest periods were given between the recordings.

Instrumentation

Acoustic analyses of voice and speech samples were accomplished by using Computer Speech Laboratory (CSL) Model 4400 (Kay Elemetrics, Corp) software. Speech samples were preamplified, low-pass-filtered at 9.8 KHz, and the digitized data was fed to the CSL - 4400 at a sampling rate of 16 KHz, using an analog-to-digital convertor with 16 - bit resolution and window size of eight. Phonatory samples were digitized at a sampling rate of 50 KHz (as per the suggested criteria by Kent et al., 2000). The Multi Dimensional Voice Profile software in the CSL - 4400 was used for analysis of phonatory samples. Analysis of temporal parameters in the samples was done using the spectrogram module in CSL - 4400, with analysis size of 100 points, pre-emphasis of 0.8 and a hamming window. The frequency features were extracted using the pitch contour module in CSL - 4400 with the minimum and maximum setting at 70 Hz and 350 Hz respectively. A 25 ms frame was used with display range of 0 to 350 Hz. The energy contour module in CSL - 4400 was used for analysis of energy with a frame length of 20 ms and display range from 30 dB (minimum) to 80 dB (maximum).

Test protocol and Analysis

I. Acoustic Analysis

Different protocols were used for investigation of the following subsystems of speech production mechanism:

A. Protocol for Phonatory tasks

B. Protocol for Articulatory tasks

C. Protocol for Prosodic tasks

Each protocol included tasks which were sensitive to investigate the function of the respective subsystem of speech.

A. Test protocol for phonatory tasks

Test protocol was developed to evaluate the following phonatory features in the speech of the subjects as shown in Table 5.

Table 5: *Protocol for phonatory tasks*

Domain tested	Task	Purpose
I A	Sustained vowel prolongation of /a/, *Ii/* and /u/	To assess laryngeal function
II A	Sustained prolongation of/s/ and *Izl* and s/z ratio	To infer on laryngeal coordination.
A. Phonation		
III A	Voice onset time for stimuli /kV and /gA/ in the words kAlAm/ & /gAlAm/	To assess laryngeal - supralaryngeal coordination

I A. Sustained phonation of vowels

The subjects were instructed to phonate the vowels /a/, /i/ and/u/ as long as possible at a comfortable loudness level after taking a deep inhalation. Three trials were given for each vowel. The mean duration of the three trials was considered as maximum phonation duration for that vowel.

The samples were digitized to extract ten parameters (reported to be frequently affected in ataxic dysathria by Kent et al., 2000), using Multidimensional Voice Profile in the Computerized Speech Laboratory (Model-4400). Analysis of phonatory parameters was done at a sampling rate of 50 KHz (as per the suggested criteria by Kent et al., 2000). The calculation algorithms for each parameter were preset. The sample size of 2.5 s in the midportion of the phonation, discarding at least the first 25ms of phonation as well as the terminal

phase of phonation was selected for analysis. This was done to capture (a) relative stable effort and pitch in the sample of the subjects & (b) to control the effects due to phonation - onset and phonation - offset (as per criteria by Kent et al., 2000). Ten acoustic parameters analyzed from the phonation sample is shown in Table 6.

Table 6: Acoustic parameters extracted from phonation samples using MDVP software

No	***Parameter***	***Unit***
Fundamental frequency related parameters		
1	Average fundamental frequency	**F0 (Hz)**
2	Phonatory frequency range	PFR (semitones)
Frequency perturbation parameters		
3	Jitter percent	Jitt (%)
4	Smoothed pitch perturbation quotient	SPPQ (%)
5	Variation in fundamental frequency	vFO (%)
Amplitude perturbation parameters		
6	Shimmer percent	Shim (%)
7	Smoothed amplitude perturbation quotient	SAPQ (%)
8	Variation in amplitude	vAm (%)
Noise related parameters		
9	Noise to harmonic ratio	NHR
10	Soft phonation index	SPI

The definition for each parameters as given in the manual for Multi - Dimensional Voice Program (Model 5105) by Kay Elemetrics is presented below:

Fundamental frequency related parameters

- F0 [Average fundamental frequency (/Hz/)]: Average fundamental frequency denotes the average number of vocal cord vibrations in a second and is denoted by Hz

- PFR (Phonatory fundamental frequency range): PFR denotes range between highest and lowest fundamental frequency and is measured in semitones.

Frequency perturbation parameters

- Jitt (Jitter percent / *%/):* Jitter percent measures cycle-to-cycle variation in pitch. It is a very short term measure of variability of pitch. The voice break areas are excluded while measuring this parameter.
- SAPQ (Smoothed Amplitude Perturbation Quotient /%/): SAPQ also measures cycle-to-cycle variation in pitch. It is a very long term measure of variability of pitch, the smoothing factor selected being 55 periods. The voice break areas are excluded while measuring this parameter.
- vFO [Fundamental frequency variation (%)]: It is a long term measure for fundamental frequency variation. It measures the coefficient of variation of fundamental frequency (i.e. ratio of standard deviation of fundamental frequency and fundamental frequency).

Amplitude perturbation parameters

- Shim [Shimmer Percent (%)]: Shimmer percent measures cycle - to - cycle variation in amplitude. It is a very short term measure of variability of amplitude.
- SPPQ [Smoothed pitch period perturbation quotient (%)]: SAPQ measures cycle-to-cycle variation in amplitude. It is a long term measure of variability of amplitude, the smoothing factor selected being 55 periods.

- vAm [peak Amplitude Variation (%)]: It is a long term measure for amplitude variation. It measures peak-to-peak amplitude coefficient of variation (i.e. ratio of standard deviation of amplitude perturbation).

Noise related parameters

- SPI (Soft Phonation Index): This parameter measures average ratio of the lower-frequency harmonic energy in the range 70 - 1600 Hz to the higher-frequency harmonic energy in the range 1600 - 4500 Hz.
- NHR (Noise-to-Harmonic ratio): A vocal signal is quasi-periodic in nature. However, the quasi-periodic wave may be contaminated with randon noise. The degree of noise can be expressed as an aperiodic noise to harmonic ratio. NHR gives average ratio of the inharmonic spectral energy in the frequency range 1500 - 4500 Hz to the harmonic spectral energy in the frequency range 70 - 4500 Hz. This is a general evaluation of noise present in the analyzed signal. The more the NHR, the greater the phonatory instability.

II A. Prolongation of /s/ and /z/ and s/z ratio

Samples of sustained productions of /s/ and /z/ sounds were collected from the subjects. The subjects were asked to prolong a /s/ sound (a measure of expiratory control) and a /z/ sound (a measure of sustained phonation). Three trials each were given for individual subjects for the prolongation of /s/ and *Izl* respectively. The s/z ratio was calculated by dividing the time taken for /s/ by time taken for /z/ measure. The s / z ratio was calculated for all three trials. The mean of the three values were considered. Typical s/z ratios of normal-speaking subjects

approximate 1.0 (Kitajima & Gould, 1976; Boone, 1977), indicating that voiceless expiration time closely matches the maximum phonation time.

III A. Voice onset time (VOT)

Voice onset time is defined as the interval between the release of an oral constriction and the initiation of glottal pulsing (Lisker and Abramson, 1964). On the spectrogram, VOT is defined as the time equivalent of the space from the onset of stop-release burst to the first vertical striation representing glottal pulsing (Lisker and Abramson, 1964, 1967; Zlatin, 1974).

Depending on the voicing status, stop sounds may have lag (for voiceless stops) or lead VOTs (for voiced stops). In English, voiced stops fall under the short lag group and voiceless stops in the long lag group (Lisker and Abramson; 1964, 1967). Savithri and Sreedevi (2002) studied Voice Onset Time in Malayalam. They studied ten native normal speakers of Malayalam using bisyllabic words with stop consonants in initial and final positions. They reported that voiced stops in Malayalam correspond to voice lead (negative VOT) and voiceless stops to short lag (positive VOT), in Malayalam language. They reported VOT of -97 ms for voiced and VOT of + 19 ms for voiceless, in Malayalam language.

The minimal pair /kAlAm-gAlAm/, differing in terms of the voicing of the initial stop consonant /k/ (velar voiceless unaspirated stop) and /g/ (velar voiced unaspirated stop) was considered for the analysis of VOT. The VOT was measured from the onset of the burst to the beginning of the striations for vowel

for the voiceless (+) (short lag VOT) and from the beginning of the voicing to the onset of the burst for the voiced (-) (lead VOT) sound.

Each of the words in the minimal pair [/kA|Am/ - /gAlAm/] were embedded in a carrier phrase /ippo nan.....ennu paRa ju/ (I say.....now) written one each on a card. The words were embedded in a carrier phrase to allow for naturalness and also to control rate of speech. The cards were presented to the subjects in a random order (within the minimal pairs) and the subjects were asked to read them in their natural reading rate and style. The subjects were required to read the carrier phrase with each minimal pair embedded in it, 5 times each in random order.

Speech sample for spectrographic analysis (to extract VOT) was digitized at a sampling rate of 16 KHz. VOT measurements was done by moving and placing the cursors on the wideband spectrogram. Clues from audio playback of the relevant speech segment were obtained while fixing the cursors for identification of the correct speech segments.

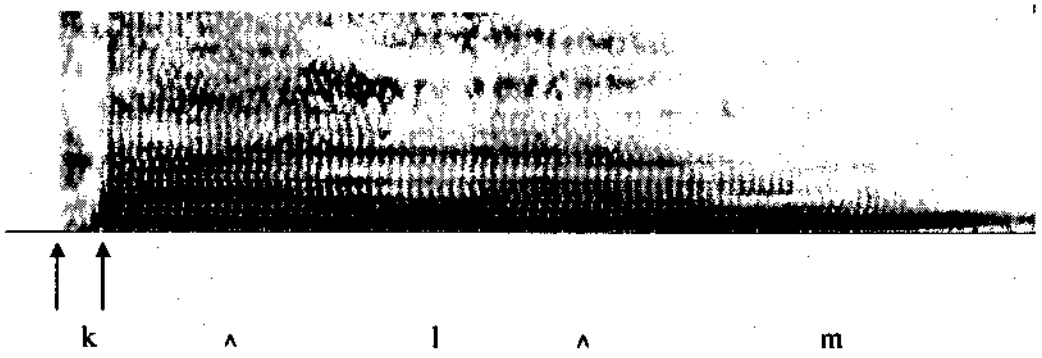

Figure 1 illustrates the spectrogram for measurement of lag VOT (for /kAlAm/) for normal control subject.

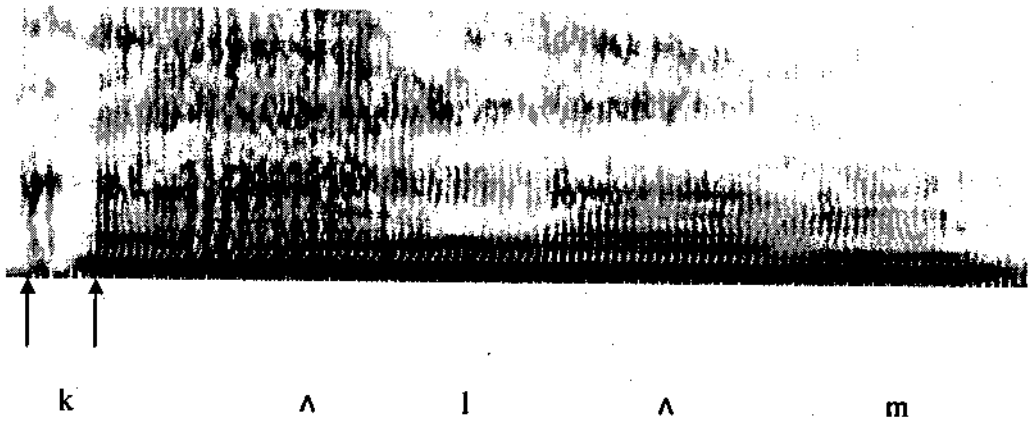

Figure 2 illustrates spectrogram for lag VOT (for /kʌlʌm/) for a subject with right superior paravermal lesion

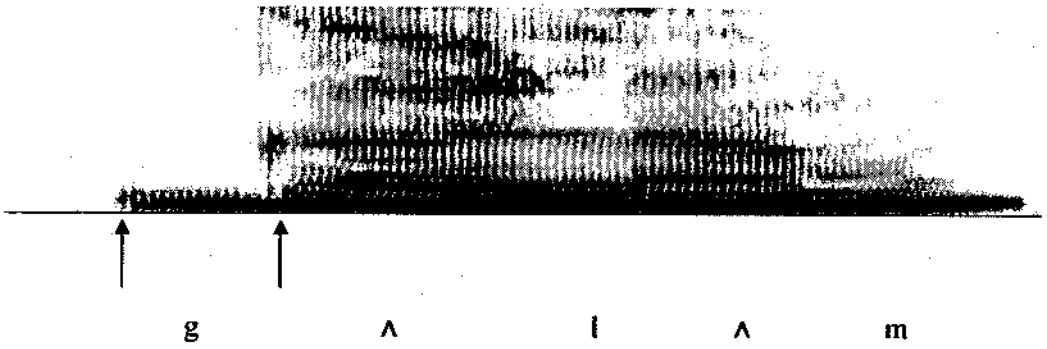

Figure 3 illustrates the spectrogram showing lead VOT (for /gʌlʌm/) for normal control subject

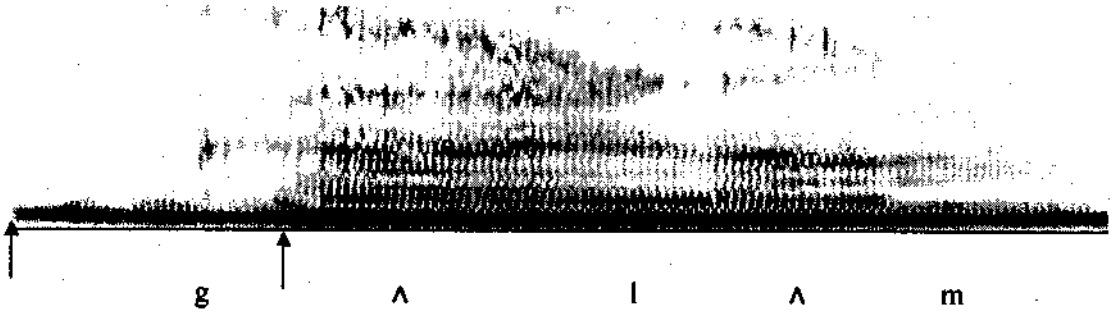

Figure 4 illustrates the spectrogram for lead VOT (for /gʌlʌm/) for a subject with right superior paravermal lesion.

B. Protocol for Articulation tasks

Three tasks were included to investigate articulatory functions and the details are shown in Table 7.

Table 7: Protocol for testing articulatory tasks

Domain tested	Task	Purpose
I B	Word repetition task	To test the articulation of subjects at word level
II B B. Articulation	Diadochokinesis - (a) Alternating motion rate (AMR) for CV syllables /pA/, /tA/ and /kA/	To assess the articulation of subjects on fast repetition of alternating motion task
	Diadochokinesis - (b) Sequential motion rate (SMR) for CVCVCV syllable sequence /pAtAkA/	To assess the articulation of subjects on fast repetition of sequential motor task
III B	Duration of short and long vowels /A/ (* short, low, back unrounded vowel) and /A:/ (long, low, back, unrounded vowel) in /kAd_Am/ and /kA:dAm/	To understand the supraglottal - laryngeal coordination, principally requiring phonatory subsystem

*[as per description of vowels in Malayalam by Syamala Kumari (1972)]

I B. Word repetition task

The word list prepared for this task is given in Appendix 3. This task was used to test the articulatory performance of the subjects. The most frequently occurring words in Malayalam were chosen by the investigator based on the work by Ghatage (1984). The words belonged to the noun or adjective category and they were culturally appropriate and unambiguous. The selected words were also phonemically balanced as checked and confirmed by a qualified linguist.

All the words were chosen by the experimenter based on the frequency of occurrence in Malayalam (Ghatage, 1984). The words in the list served to test eleven vowels, thirty three consonants, one diphthong and four consonant clusters in Malayalam. The short vowels were tested in the initial, medial and final

positions. Long vowels and diphthong were tested only in the initial and medial positions. Consonants and consonant clusters were tested in the initial and medial positions.

The words were written in Malayalam on individual cards and were presented to the experimental and control subjects with the instruction to read each word. The words were randomized and presented one at a time. A gap of 40 s was given after the subjects' response, before the next card was presented. The verbal responses of the subjects were recorded using a digital tape recorder.

The speech samples of the experimental and normal control subjects were mixed and randomized based on a random table. The speech samples were then given to three judges for assessment of misarticulations. The experimenter was one among the judges and the other two judges were post graduate students in Speech - language pathology with clinical experience in transcription and analyzing errors based on SODA. International Phonetic Alphabet (Revised Edition, 1994) was used for phonetic transcription of recorded speech material. From the transcribed speech sample sound-by-sound analysis was done in the word-initial, word-medial and word-final positions. Errors in the subjects' response on the given target sound were described as substitutions, omissions, distortions or additions. All the errors identified were of distortion type and hence only a sound-by-sound analysis and error classification based on SODA was used in the study. The results were recorded in the following format as given in Table 8.

Table 8: Format for recording the analyzed speech sample

Target Sound (1)	Stimulus word (2)	Syllable structure of(2) (3)	Subjects' production (4)	Syllable structure of(4) (5)	Error type SODA (6)

The analysis of the transcribed speech sample was carried out as follows

- The syllable structure of the stimulus words and the subjects response was analyzed.
- Distortion errors were not considered for further analysis.

Percent agreement between the first and second judge, second and third judge and first and third judge was calculated using the formula:

$$\% \text{ agreement} = \frac{\text{Total number of sounds in agreement}}{\text{Total number of sounds}}$$

where, Total number of sounds = Total number of agreements + total number of disagreements.

The same three judges reanalyzed the samples of 5 experimental subjects which were randomly selected after 3 months and responses were recorded. The percent agreement between the first and second judge, second and third judge and first and third judge was calculated again. The findings were similar to that observed in the first analysis.

// *B. Diadochokinetic task - Alternating motion rate (AMR) and Sequential motion rate (SMR)*

Model utterances for fast repetition of the syllables /p/\/, /tA/, /kA/ and syllable sequence /pAtAkA/ uttered by the investigator were recorded and these were played to individual subjects who were then asked to utter the syllables and syllable sequence in a similar manner. The model samples were played inorder to maintain uniformity in the model given to each of the subjects. The subjects were to start the repetition 30 seconds after the presentation of the model utterance. The subjects were instructed to repeat the syllables /pA/, /tA/ and */lkA/* which are presented seperately in an alternating motion rate task (AMR) and the syllable sequence /pAtAkA/ in a sequential motion rate task (SMR). They were to repeat these sounds as rapidly and precisely as possible on a single breath. The order of production of the four DDK tasks was maintained the same for all subjects (/pA/, /tA/, /KA/ & /pAtAkA/) (inorder to maintain uniformity). Two trials for each of these tasks were given and samples recorded.

The experimental subjects were able to produce an average of fourteen syllables in one breath for AMR and SMR task. All the syllable productions were however not selected for analysis. This criteria was adopted as per the reports of Ozawa, Shiromoto, Takeuchi & Watamori (1996), that AMR rates were relatively stable after three seconds from the beginning of syllable train. Hence ten syllables after three seconds from the beginning of repetition were chosen for analysis of speech sample for AMR task. The same criteria (Ozawa et al., 1996) was followed for chosing the syllables for analysis from SMR task. Three consecutive /pAtAkA/

sequences after three seconds from the beginning of syllable train were chosen for analysis. Three consecutive syllable sequences (/pAtAkA/) [Total nine syllables (three */pA/*, three /tA/ and three /k/\)] were chosen as this was the minimum number of sequences that few of the experimental subjects could produce in one breath in the SMR task.

The speech samples of diadochokinetic repetitions of syllables (/p/\, /tA/ & /KA/) and syllable sequence (/pAtAkA/) were digitized at a sampling rate of 16 KHz. Spectrographic analyses was done using wide band spectrogram using the spectrogram module of CSL - 4400 software. The syllables were measured spectrographically to obtain syllable duration ('sd'), closure duration ('cd'), total duration of the syllable ('td') and DDK rate (as per the suggested criteria by Ozawa et al., 2001) for the AMR and SMR tasks.

The temporal acoustic measure of DDK rate (syll / s), was measured for the syllables /p/\, /tA/, /kA/ and the syllable sequence /pAtAkA/ following the suggested formula of Kent et al., (1999) and Ozawa et al., (2001). All the measures were calculated for both the trials.

- DDK rate for AMR and SMR tasks was operationally defined as the number of syllables uttered in a second. Number of syllables per second (syll / s) for the AMR and SMR tasks were made by inspecting the acoustic signal and also using the waveform display.

- Syllable duration was operationally defined as the temporal interval between the vertical spike corresponding to burst release and the offset of glottal pulsing for the following vowel.
- Closure duration was operationally defined as the interval between the offset of glottal pulsing for a vowel and the burst release for a consonant. For voiceless stop consonants, the closure duration is silent; because the vocal tract is occluded and the vocal folds are not vibrating (voicing energy is absent).
- Total duration ('td') was operationally defined as the sum of syllable duration ('sd') and closure duration ('cd') for the syllable ('td'= 'sd'+ 'cd') (as per the criteria by Ozawa et al., 2001).

The energy contour module in CSL - 4400 was used to analyse minimum and maximum intensity values for syllables /pA/, /tA/ & /kA/ in AMR task and syllable sequence /pAtAkA/ in SMR task. The minimum and maximum intensity measures for the syllables /pA./, /tA/, /KA/ and syllable sequence /pAtAkA/ were obtained by placing the cursors on the trough of the energy contour for a syllable and then on the corresponding peak for that syllable (as per Kent et al., 1999). These measures were obtained for 10 consecutive syllables for the AMR task and for three consecutive syllable sequences in the SMR task. Energy minima (minimum intensity) and energy maxima (maximum intensity) were averaged for each speaker. All the measures were calculated for both the trial.

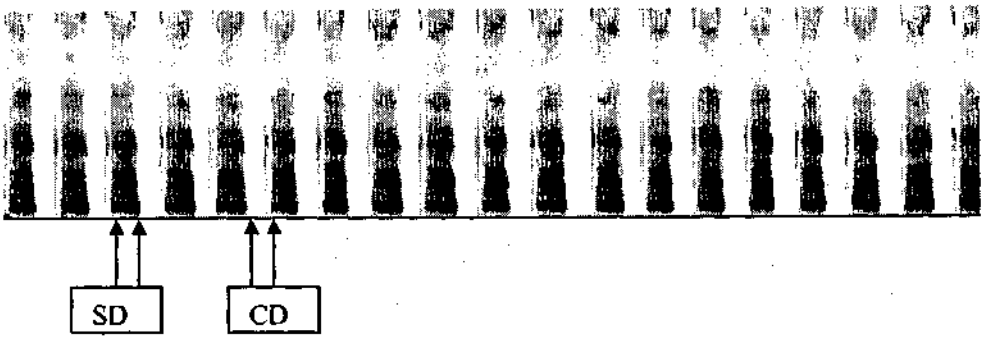

Figure 5 illustrates spectrogram for normal control female subject subject, for the syllable /pʌ/ showing syllable duration ('sd'), closure duration ('cd') and total duration ('td').

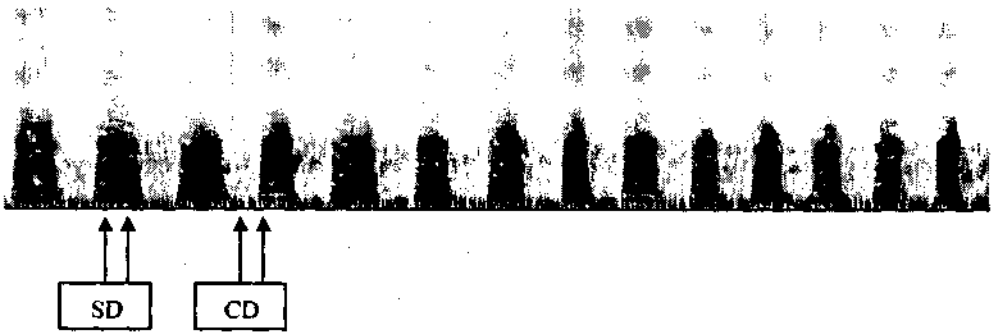

Figure 6 illustrates spectrogram for subject with right superior paravermal lesion, for the syllable /p/v/ showing syllable duration ('sd'), closure duration ('cd') and total duration ('td').

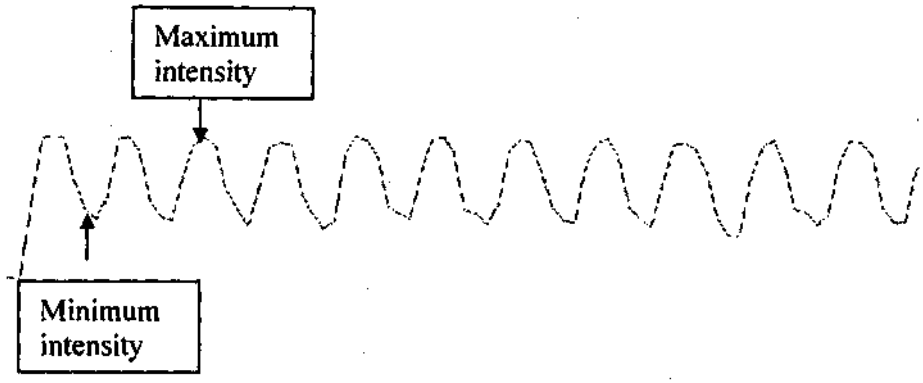

Figure 7 illustrates the energy contour for /pV showing maximum and minimum intensity for a normal control male subject (the arrows indicate the maximum and minimum intensity)

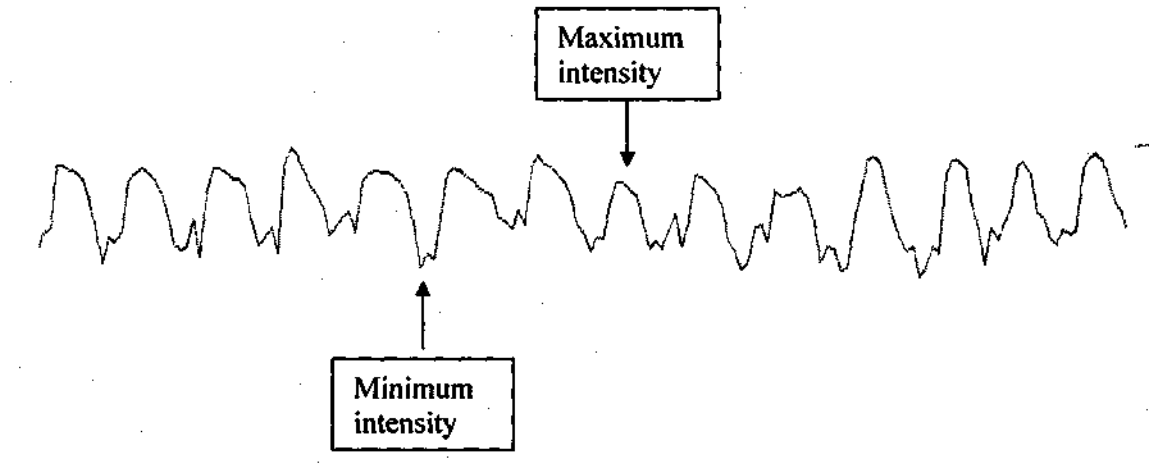

Figure 8 illustrates the energy contour for /p/\/ showing maximum and minimum intensity for a subject with superior vermis lesion (the arrows indicate the maximum and minimum intensity)

/// *B. Duration of short and long vowels*

The minimal pairs /*kAdAm/* - /kA:dAm/ were used for the measurement of duration of short (/A/) and long (/A:/) vowels. Each of the words in the minimal pairs /kAdAm/ and /kA:dAm/ were embedded in a carrier phrase / ippo nA:n.....

ennu PARA JU/ written one each on a card. The subjects were required to read the carrier phrase 5 times each in random order.

The vowel durations were measured from the onset of the formant energy for the vowel in the initial syllable of the word to the offset of formant energy for that vowel (Ackermann, Graber, Hertrich and Daum, 1999). Vowel duration was defined as the onset of periodic energy to the change in waveform associated with the initiation of the word medial retroflex /d. / in /kAdAm/ - /kA:dAm/.

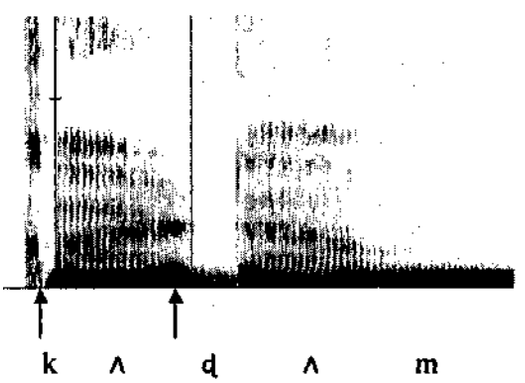

Figure 9 illustrates spectrogram (for /kAd.Am/) for normal control subject

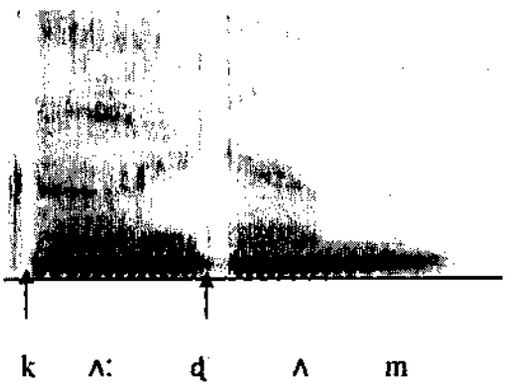

Fig 10 illustrates spectrogram (for / kAdAm/) for subject with superior vermis lesion

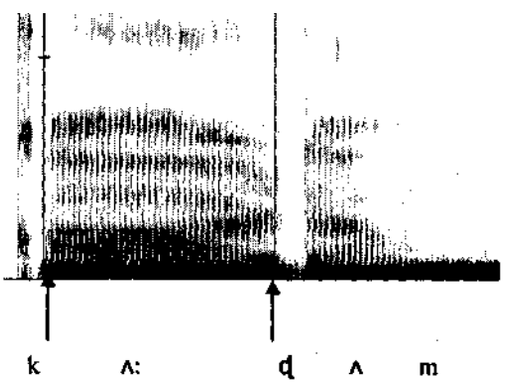

Figure 11 illustrates spectrogram (for /kA-.dAm/) for normal control subject

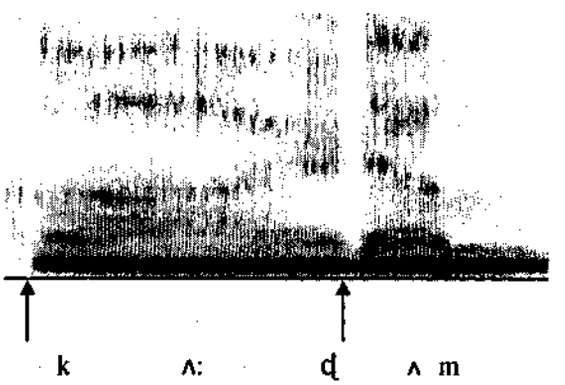

Fig 12 illustrates spectrogram (for / kA:dAm/) for subject with superior vermis lesion

C. Protocol for Prosodic tasks

One task was included to investigate prosodic function and the details are given in Table 9.

Table 9: Protocol for testing Prosodic tasks

Domain tested	Task	Purpose
I C C. Speech rate	Passage reading	To assess speech rate in a reading task
(syll / s) H C	Narration of two picture cards from Linguistic Profile Test (LPT) in Kannada by Karanth (1980)	To assess speech rate in a narration task

/. *Speech rate*

The prosodic system was tested by measuring Speech rate (number of syllables produced in a second) in a passage reading and narration task. The speech samples were obtained from reading passage (Appendix 4) and narration task (Appendix 5). A 524 syllable passage incorporating all the frequently occurring phonemes, consonant clusters, inflectional morphemes, words, word boundaries fused by morphophonemic alternations and words with different suffixes in Malayalam as per Ghatage (1994) was developed by the investigator in consultation with a qualified linguist. The syllable structure of the contents in the passage varied from simple to complex (as stated earlier). The reading passage is presented in Appendix 4. The reading passage was typed in bold letters on a card and was presented to the subjects who were instructed to read them aloud at comfortable loudness level and in their habitual reading rate and style.

Two pictures which formed a part of the "Linguistic Profile Test" in Kannada language by Karanth (1980) depicting a 'village scene' and 'school scene' were used for eliciting the narrative sample (Appendix 5). Subjects were

presented the pictures and were asked to narrate the events, things, actions etc depicted in the picture as naturally as possible. After the presentation of the picture, subjects were given one minute to prepare before they commenced narration. They were asked to narrate everything in the picture in as detail a manner as possible in their natural rate and speaking style.

I a. Speech rate in passage reading sample

Speech rate was calculated from the entire passage. The total number of syllables as read by the subjects was counted by the investigator. The total duration of the speech sample was obtained by measuring the total time taken for the passage to be read from the waveform obtained. Speech rate (syll / s) for passage reading was obtained by the formula:

$$\text{Speech rate (syll / s)} = \frac{\text{Total number of syllables in the passage}}{\text{Total time taken for the sample (s)}}$$

/ b. Speech rate in narration sample

Speech rate was calculated for the first 45 s sample of the narrated speech. This duration was selected as this was the minimum sample duration for which some of the experimental subjects spoke without pausing for speech. The total numbers of syllables in the narration sample of the subject was counted by the investigator. Speech rate (syll / s) for narrative sample was obtained by the formula:

$$\text{Speech rate (syll / s)} = \frac{\text{Total number of syllables in narration}}{\text{Total time taken for the narrative sample (s)}}$$

Distortion of syllables and disintegrity of syllables were only mild in all the subjects and hence no attempt was made to exclude the pauses (silent as well as articulatory pauses) while calculating the speech rate. Moreover, calculation of speech rate by excluding pauses does not provide a naturalistic picture of the disorder and hence was not included in the analysis.

II. Perceptual Analysis

One minute sample was identified from the corpus of narrative speech from the sample of the subjects. This duration was found to be representative for perceptual rating after listening to the speech samples of all the experimental subjects. The selected speech samples were randomized using a random table and transferred to a different tape. A total number of 47 samples were recorded. A 50 s silent interval was inserted between each of the samples. Three native female speakers of Malayalam (investigator and 2 Post graduate students in Speech - language pathology) served as the judges for perceptual analysis of speech. All three judges had prior experience / exposure in perceptual rating of dysarthric speech. Perceptual analysis of 38 dimensions used by Darley et al., (1975) (Appendix 6) was done to obtain perceptual data for the sample of narration.

For perceptual analysis, the recorded samples of both normals and dysarthric subjects were presented in random order. The samples were judged independently by the 3 judges (experimenter and two other judges). The two judges were blind to the purpose of the study. No identity was revealed about the subject, except information on age and sex. The speech samples were played on a tape recorder at a comfortable loudness level for the judges. The samples were

played in a silent room and the judges were allowed to listen to the speech sample, any number of times they wanted. Adequate time was given to the judges for rating the speech samples. All three judges rated the speech samples in four sessions (each session was for two to two and half hours). The three judges completed the perceptual ratings within two days.

The judges rated only one dimension of speech at a time and recorded the severity of each sample with regard to that dimension. After listening to all the samples on the tape for one dimension, the judges listened to the series again, and then rated the samples for the next speech dimension. In this way, attention was focused on one dimension at a time in the series of speech samples. The judges were given frequent rest periods during the perceptual analysis.

A seven point severity rating scale was adopted for perceptual judgement. The scale was as follows:

1 = normal speech
2 = mild
3 = mild to moderate
4 = moderate
5 = moderate to severe
6 = severe
7 = very severe

Measures of inter judge reliability were calculated for the perceptual ratings. Item by item reliability was calculated between the first and second judge, second and third judge and between first and third judge. Also, ten samples were randomly chosen and re-rated by the same three judges after 3 months to check for intrajudge reliability. Item by item reliability was calculated between the first and

second ratings of the first, second and third judge to obtain measures of intrajudge reliability.

Statistical analysis

A) Acoustic data:

The study included cross sectional standard group comparison design. This method is often suggested for medical and biomedical investigations by several statisticians (Oakes, 1986; Gardner and Altman, 1986; Everitt & Arnold, 1994). There were limited number of subjects in each of the experimental groups, which warranted the use of confidence interval as statistical procedure as against the statistical tests used for equality of means like t-test. Confidence intervals are value limits within which value of population mean is logically deduced. Any value which falls outside the limits of the confidence interval is considered to be different from population mean. The interval to be constructed is called a confidence interval and the values describing the boundaries of such an interval are the confidence limits. The degree of "confidence" in the proposition that the stated interval actually contains the population mean is indicated by a probability value. A 95% confidence interval is most commonly used in biological studies. Confidence intervals combine information on location and precision and can often be directly used to infer significance levels, and are thus, the best reporting strategy (APA manual, pp. 22, 5th Edition).

The mean scores of the experimental subgroups for different speech tasks analysed acoustically were compared with confidence intervals obtained for normal control subjects to see whether there is any difference from normal control

subjects. Comparison was done separately for the male and female subject groups, wherever gender based differences were expected. For this comparison, confidence intervals were obtained separately for male and female normal control subjects.

A confidence interval is calculated using t^1, the appropriate percentage point of the / distribution with (n-1) degrees of freedom.

Small-sample CI = x- (t' X s/$\sqrt{n}$) to x + (t' x s/ Vn),

where CI = confidence interval, x = Sample Mean, s = standard deviation of the sample, n = Sample size

B) *Perceptual data:*

Percent agreement was calculated for the perceptual analysis of word repetition task by the three judges. Item by item reliability was calculated for the perceptual evaluation of the narration sample for all the thirty eight dimensions given by Darley et al., (1975). Reliability coefficient alpha ($\propto$) was obtained separately for the ratings between three judges. A contingency table for the reliability ratings between the three judges was prepared for all the thirty eight dimensions and a cut off point was operationally defined.

Descriptive statistics was obtained using the statistical software SPSS (version 10) and Microsoft Excel.

RESULTS AND DISCUSSION

The aims of the study are to analyze the speech and voice dimensions in subjects with ataxic dysarthria due to lesions in various sites of the cerebellum, using acoustic and perceptual methods. Seventeen cerebellar dysarthrics served as subjects of the experimental group. They are grouped into six categories based on the site of lesion in the cerebellum and this is shown in Table 10.

Table 10: *Details of experimental subjects including site of lesion in the cerebellum*

[LSP = left superior paravermal, LAI' — left anteroinferior, SV = superior vermis, RSP = right superior paravermal, RPS = right posterosuperior and RAS = right anterosuperior]

Lesion in the cerebellum	Age & Sex	Total
Left superior paravermal	29/F	3
	23/F	
	45/F	
Left anteroinferior	37/F	2
	27/M	
Superior vermis	36/M	5
	27/M	
	50/ F	
	42/F	
	25/F	
Right superior paravermal	42/ F	3
	39/F	
	31/F	
Right posterosuperior	34/M	3
	46/M	
	51/M	
Right anterosuperior	23/M	1

Phonatory, articulatory and prosodic features were investigated in the speech of experimental and control subjects. The analyses included extraction of spectral, temporal and perceptual measures. The details of the tasks and the

measures obtained are presented in Table 11, as categories I, II and III (Phonatory, Articulatory and Prosodic). This is followed by results of perceptual analysis (Category IV).

Table 11: *Brief overview of the tasks involved in the study and the measures obtained from these tasks*

	Tasks	*Spectral*	*Temporal*	*Perceptual*
I *Phonatory tasks*	Maximum phonation duration (MPD) for vowels /a/, /i/ and /u/		MPD (s)	
	Maximum fricative duration for /s/ and /z/ and the s/z ratio		Maximum fricative duration and s/z ratio	
	Frequency, amplitude and noise measures of sustained vowels /a/,/i/ and/u/	Ten voice parameters extracted using Multi Dimensional Voice Profile (MDVP) software		
	Voice onset time (VOT) in the stimuli words /kAlAm/ and /gAlAm/		VOT for voiceless/ voiced sounds in the stimuli words /kAlAm/ and /gAlAm/	
II *Articulatory tasks*	Word repetition task			Analysis of errors based on SODA
	Diadochokinesis • /pA/, /tA and /kA/ in AMR • /pAtAkA/ in SMR	• Minimum intensity • Maximum intensity • Intensity range	• Syllable duration • Closure duration • Total duration • DDK rate	
	Duration of short and long vowels in the stimuli words /kAlAm/ and /kA:lAm/		Duration of short and long vowels	
HI Prosodic task	Reading and narration task		Speech rate (syll/s)	

The data obtained from the experimental groups is compared with the data obtained from age and gender matched control group. Only descriptive statistics (Mean, SD and confidence intervals) was employed in this study. Confidence intervals were obtained for the normal control group, with which the mean values obtained for the experimental groups were compared. Confidence intervals are established for the normal control group in order to infer about the population and hence the values of the experimental subjects were compared with the confidence intervals for normals. A 95% confidence interval was chosen in this study, as per the suggested criteria in most of the biological studies (Rozenboom, 1960; Gardner & Altman, 1986; Oakes, 1986). Any measure which is outside confidence interval is considered deviant. Small sample size of experimental subjects did not permit the use of paramateric as well as nonparametric statistics. The experimental subject groups were small due to the stringent inclusion and exclusion criteria that were followed. Caution was taken to include subjects with focal cerebellar tumours in cerebellar hemisphere or midline structures of the cerebellum, only. The control for variables such as age, language, exposure to speech therapy or medication, comorbid features etc. as established in the method chapter was followed.

The measured parameters in the experimental groups are compared between:

- Left superior paravermal lesions and normal control subjects
- Left anteroinferior lesions and normal control subjects
- Superior vermis lesions and normal control subjects
- Right superior paravermal lesions and normal control subjects

- Right posterosuperior lesions and normal control subjects
- Right anterosuperior lesions and normal control subjects

Specific comparisons were made between left superior paravermal and corresponding lesion in the right i.e. right superior paravermal lesions. Comparisons were also made between right posterosuperior and right anterosuperior lesions. No comparisons were made between other cerebellar lesions because of the absence of corresponding lesions.

I. Phonatory measures

A. Temporal measures

1. Maximum Phonation Duration (MPD)

Maximum phonation duration (MPD) is measured as the maximum time over which phonation can be sustained after a deep inhalation for a vowel sound. Maximum phonation duration was obtained for vowels /a/, *I'll* and /u/. The measure of Maximum Phonation Duration reflects on the coordination of respiratory and laryngeal processes.

The results in Table 12 reveal that the maximum phonation duration is reduced in all the experimental groups compared to normal controls. MPD places demands on sustaining good coordination of respiratory, laryngeal and supralaryngeal structures for a long time. Reduced MPD in all the experimental groups suggests poor coordination of respiratory, laryngeal and supralaryngeal structures as a general feature.

Table 12: *Mean (sec) and SD for measures of maximum phonation duration for /a/, /if and /u/ of normal controls (N) and experimental groups and confidence intervals (CI) for normal control subjects. [(*) indicates that the mean values are outside the confidence interval]*

	N	N	LSP	LAI	LAI	SV	SV	RSP	RPS	RAS
Gender	Male	Female	Female	Female	Male	Female	Male	Female	Male	Male
Vowel /a/										
Mean (s)	17.511 to	16.50	*10.21	*13.54	*14.21	•11.15	•12.21	•13.02	•14.55	•13.89
SD	9.41	6.91	12.03	8.62	8.47	7.54	8.55	11.21	5.42	4.53
CI	16.99 to 18.03	15.14 to 16.85								
Vowel /i/										
Mean (s)	17.47	16.46	*9.63	*12.71	*12.53	•10.64	•10.22	•12.14	•15.64	•14.53
SD	5.34	8.21	9.54	5.34	6.34	11.65	12.54	13.42	4.74	5.12
CI	17.16 to 18.78	15.37 to 16.75								
Vowel/i/										
Mean (s)	17.82	16.76	*11.04	* 14.44	*14.93	*9.58	•13.07	•14.53	•15.98	•10.51
SD	7.11	5.62	14.11	4.44	5.77	9.22	10.21	9.86	3.89	2.87
CI	17.29 to 18.96	15.49 to 17.01								

Abnormal loudness variation, shorter phrase lengths and loudness decay have been reported as indirect indices of respiratory abnormalities in ataxic dysarthria by Darley et al., (1975) and Ludlow and Bassich (1983). But these observations were made with reference to subjects with nonfocal cerebellar involvement. Brown et al., (1970) also observed reduced respiratory rate and vital capacity in ataxic dysarthric subjects with nonfocal lesions, which they attributed to reduced MPD and poor temporal coordination between respiration and phonation. Murdoch et al., (1991) reported irregularities in chest wall movements during tasks of sustained vowel phonation and syllable repetition in some subjects with ataxic dysarthria due to degenerative causes. Few investigators attributed phonatory abnormalities as the most deviant perceptual dimensions in ataxic

dysarthric subjects with nonfocal lesions due to hypotonia in the respiratory and laryngeal musculature (Brown et al., 1970; Darley et al., 1975; Chenery et al., 1990). Kent and Netsell (1975) stated that hypotonia accounts for the physiological deficits like delay in the generation of muscular forces, reduced rate of muscular contraction and reduced range of movements, which is inturn reflected in speech as prolongation and slowness of movement.

The results in Table 12 suggests that MPD is reduced in all the cerebellar subgroups of the study, that is in subjects with lesions restricted to left superior paravermal (LSP), left anteroinferior (LAI), superior vermis (SV), right superior paravermal (RSP), right posterosuperior (RPS) and right anterosuperior (RAS) regions. The reason for reduced MPD may only be speculated as due to hypotonia of the respiratory and laryngeal structures. Interestingly, in this study, in addition to hemispheric lesions (left superior paravermal, left anteroinferior, right superior paravermal, right posterosuperior and right anterosuperior), MPD was reduced in midline lesion of superior vermis also. These preliminary findings may be suggestive of the fact that hypotonia may be a generalized symptom seen in subjects with cerebellar lesion irrespective of the lesion site. The findings contradict the report by Mancall and McEntee (1965), Maurice-Williams (1975), Amici et al., (1976) and Gilman (1986) which stated that hypotonia was prominent in instances of damage to the lateral (hemispheric) zones of the cerebellum. It is only speculated that an underlying hypotonia could be the cause for reduced MPD in all the subgroups of cerebellar lesions. This requires to be substantiated with evidences from physiological studies before generalizing the results.

A poor range of MPD implies deficit in respiratory support as well as vocal fold vibration. The subjects with left cerebellar (left superior paravermal, left anteroinferior), superior vermis and right cerebellar (right superior paravermal, right posterosuperior and right anterosuperior) lesions all showed poor MPD suggesting that the neural correlates for coordinated activity of respiratory and phonatory skills are equally implied in the left superior paravermal, left anteroinferior, superior vermis, right superior paravermal, right posterosuperior and right anterosuperior areas of the cerebellum.

2. Maximum Fricative Duration and the s/z Ratio

This task is generally employed as a clinical means to infer on the adequacy of laryngeal musculature in disordered individuals. Boone (1977) proposed the use of a measure of voiceless / voiced sustained production ratio for the sounds /s/ and *Izl*. Production of voiceless fricative /s/ requires the coordination of respiratory and supralaryngeal structures, while the production of voiced fricative ***Izl*** requires the coordination of laryngeal musculature along with respiratory and supralaryngeal structures (Keller, Vigneux and Laframboise, 1991). The measure of s / z ratio by itself cannot be used to distinguish a deficit in respiratory support from that of vocal fold vibration. Boone (1977) suggested that the clinical evaluation of vocal fold function can be conducted using the measure of maximum phonation duration for production of voiced *Izl* sound contrasted with a sustained expiration without phonation for voiceless sound *Is/*. The ratio of voiceless / voiced sound (s / z) will be approximately one (unity) for speakers with normal phonatory functions but larger than unity for individuals with laryngeal dysfunction (i.e vocal fold thickening, polyps or nodules). Table 13 and 14 gives

the confidence intervals (CI), Mean (sec) and SD for /s/, *Izl* measures and s/z ratio respectively.

Table 13: *Mean (sec) and SD for control and experimental subjects for /s/ and /z/ measures (in secs) and confidence intervals for normal control subjects. [(*) indicates values outside the confidence intervals].*

Group	N	N	LSP	LAI	LAI	SV	SV	RSP	RPS	RAS
	Female	Male	Female	Female	Male	Female	Male	Female	Male	Male
					/s/					
Mean (sec)	19.80	19.66	* 15.03	•16.11	*15.13	•17.07	•14.28	•16.39	20.01	19.49
SD	9.44	8.18	10.12	5.12	3.11	5.89	7.21	11.23	12.41	5.42
CI	19.00 to 21.61	19.00 to 21.31								
					/z/					
Mean (sec)	20.25	19.51	•13.12	•12.23	•12.44	•9.13	•10.16	•15.52	20.41	19.81
SD	10.29	6.85	12.42	4.84	6.45	8.96	L 4.53	9.87	8.62	8.12
CI	19.54 to 21.97	I8.8U0 22.20								

Table 14: *The Mean and SD for control and experimental groups for s/z ratio. Confidence intervals (CI) for normal controls is also given [(*) indicates values outside the confidence intervals].*

Measure	N	N	LSP	LAI	LAI	SV	SV	LRSP	RPS	RAS
s/z ratio	Female	Male	Female	Female	Male	Female	Male	Female	Male	Male
Mean	0.98	1.01	*1.15	*1.32	*1.22	*1.88	*1.48	*1.06	0.98	0.98
SD	7.04	4.08	8.96	6.12	4.97	6.72	6.87	10.47	7.33	6.47
CI	0.96 to 0.99	0.96 to 1.05								

The results in Table 13 indicate that the duration of /s/ and *Izl* are reduced in all the experimental subgroups, with the exception of right posterosuperior (RPS) and right anterosuperior (RAS) subjects. The duration of /s/ and *Izl* for right posterosuperior and right anterosuperior groups are comparable to that of normal controls. As a task, sustained production of fricative sounds requires good regulation of the muscle forces that produce the aerodynamic conditions of turbulence, it may be presumed that the regulation of these muscle forces were

affected in the left superior paravermal (LSP), left anteroinferior (LAI), superior vermis (SV) and right superior paravermal (RSP) lesions. Delay in generation of muscular forces as a result of hypotonia is a common feature reported in ataxic dysarthria due to nonfocal lesions, further affecting the stability of forces developed by the tongue muscles required for the production of sustained fricatives (Kent and Netsell, 1975). The trend observed in the experimental subjects possibly suggests that tongue muscle force dysfunction may not be a common feature of ataxic dysarthria. It may be affected only in those with lesions in certain areas of the cerebellum like left superior paravermal, left anteroinferior, superior vermis and right superior paravermal lesions. Deficits in tactile feedback have also been reported in ataxic dysarthria by Bowman (1971). It may be too early to presume a deficit in tactile feedback from the tongue muscles as an underlying cause for reduced fricative duration for /s/ and */zl*, in left superior paravermal, left anteroinferior, superior vermis and right superior paravermal lesions. The duration of fricatives (/s/ & /z/) were comparable to normal control group for subjects with lesions in right posterosuperior and right anterosuperior lesion. It may be speculated that these areas of the cerebellum are possibly not involved in the control of sustained production of fricatives. To confirm, these findings need to be supported with physiological evidence as well as data from larger sample.

The results for s / z ratio in Table 14 indicate that s / z ratio of the left superior paravermal, left anteroinferior, superior vermis and right superior paravermal areas in the cerebellum are increased compared to that of normal controls. It may be speculated that the neural mechanisms involved in the

sustained production of fricatives are more implicated in these cerebellar areas. The right posterosuperior as well as right anterosuperior areas in the cerebellum does not seem to be involved in the task of sustained production of fricatives as the s/z ratio is comparable to that of normal controls.

3. Voice Onset Time (VOT)

The measure of VOT was obtained for syllable /ka/ in the word /kAlAm/ and for syllable /gA/ in the word /gAlAm/. The subjects were required to read the minimal pairs /kAlAm/ and /gAlAm/, which were embedded in carrier phrase, five times each at a normal rate and prosody. The duration of voiced / voiceless VOT (VL / VD) were obtained from the samples after spectrographic analysis in the CSL-4400. Voice onset time measures for voiceless sounds are presented in *Section a* and the voice onset time measures for voiced sounds are presented in *Section b.*

Section a

(a) Duration of Voice Onset Time for voiceless sounds

The mean, SD and confidence interval for duration of voiceless VOT (VL) for the normal control subjects and experimental groups are given in Table 15.

Table 15: *Mean VOT and SD for /kA/ for normal control subjects (N) and experimental groups and confidence interval for normal control subjects for duration of voiceless VOT [VL=Duration of voiceless VOT}*

	Mean (ms)	SD	Confidence interval
N	+20.36	11.80	+19.44 to+23.65
LSP	*+32.73	14.18	-
LAI	*+3l.50	17.69	-
SV	*+4l.66	2323	-
RSP	*+39.52	21.56	-
RPS	*+37.6l	16.52	-
RAS	*+32.93	1340	-

The results in Table 15 indicate that duration of voiceless VOT (VL) is increased for all the experimental groups compared to normal controls. In Malayalam, the voiceless stops use the short voicing lag (+) and the voiced stops use the voicing lead (-) as opposed to voiceless stops in English using the long lag (+) and voiced stops using the short lag (+) categories in English.

All the experimental groups showed increased VOT's for voiceless category. In case of unaspirated voiceless stops, no time-critical sequence of events has to be performed by normal speakers since the short lag VOT results from the fact that the released airstream after the velar closure needs some time to induce laryngeal vibration. In case of the experimental groups in this study, the prolonged VOT for the voiceless stops may be attributed to articulatory slowness of the release. Kent and Netsell (1975) opined that hypotonia associated with cerebellar lesions leads to delay in generation of muscular forces, reduced rate of muscular contraction and reduced range of movements. These may be reflected in speech as prolongations and slowness of movement. The increased VOT in all the experimental groups may be attributed to a generalized hypotonia seen in subjects

All the experimental groups showed increased variability compared to normal control subjects for voiceless VOT. The increased variability may be attributed to differences in timing of articulatory release across repetitions. Increased variability in speech is a commonly reported feature in ataxic dysarthria due to nonfocal lesions (Kent and Netsell, 1975; Kent et al., 2000). The results obtained in this study indicate that increased variability is a characteristic feature of cerebellar subjects with lesions restricted to different sites of the cerebellum.

Section b

(a) Duration of Voice Onset Time for voiced sounds

The mean, SD and confidence interval for duration of voiced VOT (VD) for the normal control and experimental subjects are given in Table 16.

Table 16: *Mean and SD for /ga/ for normal control subjects and experimental groups and confidence interval for normal control subjects for duration of voiced VOT. [VD=Duration of voiced VOT]*

	Mean (ms)	SD	Confidence interval
N	-73.64	13.12	-69.12 to-77.31
LSP	+-78.17	15.52	-
LAI	*-80.32	1441	-
SV	*-84.10	2123	-
RSP	*-8766	24.01	-
RPS	-74.33	18.61	-
RAS	*-79.98	14.03	-

The duration of VOT for voiced (VD) is increased for all the experimental groups except for subjects with right posterosuperior lesion (Table 16). VOTs for voiced stop consonants is an index for the production of cross-laryngeal airstream while the place of articulation is still in the occlusion phase (i.e. laryngeal vibrations have to be initiated prior to the release of the burst). This airstream can only be generated by enlarging the intra-oral space behind the place of

with cerebellar lesion irrespective of the lesion site. It may be speculated that the neural correlates underlying the production of VOT for voiceless unaspirated stops are distributed in the left superior paravermal, left anteroinferior, superior vermis, right superior paravermal, right posterosuperior and right anterosuperior regions of the cerebellum.

According to the speculation by Boutsen and Christman (2002), the right cerebellar hemisphere is involved in the coordination of supralaryngeal and laryngeal movements as is required for voice onset time, due to its contralateral connection with the left cerebral hemisphere. Also, the 'Differential cue lateralisation hypothesis' by van Lancker and Sidtis (1992) contends that temporal aspects of speech are lateralized to the left cerebral hemisphere (and hence right cerebellar hemisphere due to contralateral connection). In the present study, it can be seen that VOT for voiceless stops is increased in all the experimental groups compared to normal controls. It may be that in addition to the right cerebellar hemisphere, the left cerebellar hemisphere as well as superior vermis regions are involved in the control of voice onset time. These differences in findings may be attributed to differences in languages studied (and hence differences in VOT categories). The results obtained by Boutsen and Christman (2002) are based on findings of English speaking ataxic dysarthric subjects. In English, VOT's for voiceless stops represent long lag category whereas in Malayalam, it represents the short lag category. Moreover, differences in aspiration of stops (aspirated stops in English as opposed to unaspirated stops in Malayalam), could be another factor attributing to the differences in findings.

articulation, for example, by an active jaw and tongue lowering gesture. It may be presumed that neural structures required for this supralaryngeal coordination as is required for the production of VOT's for voiced stop consonants are mediated in the left superior paravermal, left anteroinferior, superior vermis, right superior paravermal and right anterosuperior areas of the cerebellum. The right posterosuperior region of the cerebellum does not seemed to be involved in controlling the mechanisms involved in the production of VOT's for voiced stop consonants as VOT's for voiced stop consonant is comparable to normal control subjects.

According to the speculation by Boutsen and Christman (2002), the right cerebellar hemisphere is involved in the coordination of supralaryngeal movements as is required for voice onset time, due to its contralateral connection with the left cerebral hemisphere. The 'Differential cue lateralisation hypothesis' by van Lancker and Sidtis (1992) also contends that temporal aspects of speech are lateralized to the left cerebral hemisphere (and hence right cerebellar due to contralateral connection). The results obtained for duration of voiced VOT category indicates that left cerebellar hemisphere (left superior paravermal and left anteroinferior) as well as superior vermis also seemed to be implicated in the control of voice onset time. The results in this study indicate that not all right cerebellar regions may be involved in the control of mechanisms underlying the production of VOT's for voiced stop consonants, as it is found to be increased only in subjects with right superior paravermal and right anterosuperior regions, whereas it is comparable to normal control subjects in subjects with right posterosuperior lesions.

Variability is more in subjects with superior vermis, right superior paravermal, right posterosuperior and right anterosuperior lesions. This indicates instability in temporal control of VOT's for voiced consonants in these experimental groups.

Summary

The VOT for voiceless as well as voiced sounds are increased in all the experimental groups. As an exception, the VOT for voiced sound in subjects with right posterosuperior lesion is comparable to normal control subjects. It may be speculated that the neural correlates underlying the production of VOT for voiceless unaspirated stops are distributed in the left superior paravermal, left anteroinferior, superior vermis, right superior paravermal, right posterosuperior and right anterosuperior regions of the cerebellum. The increased VOT for voiceless sounds in these groups can be attributed to articulatory slowness due to the delay in time taken by the released airstream after velar closure, to induce laryngeal vibration. The neural structures required for this supralaryngeal coordination as is required for the production of VOT's for voiced stop consonants seem to be mediated by the left superior paravermal, left anteroinferior, superior vermis, right superior paravermal and right anterosuperior areas of the cerebellum. The right posterosuperior region of the cerebellum does not seem to be involved in controlling the mechanisms responsible for the production of VOT's for voiced stop consonants as it is comparable to normal control subjects.

B. Spectral measures

1. *Frequency, Amplitude and Noise related measures of vowels*

Phonatory dysfunction is reported as one of the most frequently observed abnormalities in ataxic dysarthria (Duffy, 1995; Hertrich et al., 1998; Kent et al., 2000). These observations are mostly based on studies including subjects with diffuse lesions in the cerebellum. Acoustic studies to quantify the phonatory dysfunction in lesions restricted to the cerebellum are scanty and include only few parameters. The phonation samples of vowels /a/, *I'xl* and /u/ were analyzed on the Multi - Dimensional Voice Program (MDVP) software of Computerized Speech Lab (4400). There is scope to measure 38 parameters using the MDVP software, but based on the smoothing factor and parameters that measure similar aspects, only ten parameters were included in the study. This is also as per the selection paradigm used in the acoustic analysis of ataxic speech by Hertrich et al., (1998) and Kent et al., (2000).

a] Fundamental frequency related parameters

(i) Average Fundamental frequency (Hz)

(ii) Phonatory frequency range (semitones)

b] Frequency perturbation parameters

(i) Jitter percentage (%)

(ii) Smoothed pitch period perturbation quotient (SPPQ) (%)

(iii)Variation in FO (vFO) (%)

c] Amplitude perturbation parameters

(i) Shimmer percentage (%)

(ii) Smoothed amplitude perturbation quotient (SAPQ) (%)

(iii)Variation in amplitude (vAm) (%)

d] Noise **related parameters**

(i) Noise to Harmonic ratio (NHR)

(ii) Soft Phonation Index (SPI)

The data obtained for the experimental groups were compared with the confidence intervals obtained for normal control group. The results are presented in Table 17 to Table 26.

a] Fundamental frequency related parameters:

(i) Fundamental frequency (FO) Hz

Fundamental frequency (FO) for normal controls and experimental subjects are given in Table 17.

Fundamental frequency (FO) measures of left (left superior paravermal, left anteroinferior) and right (right superior paravermal, right posterosuperior and right anterosuperior) cerebellar groups is comparable to normal control subjects for all the three vowels. FO is reduced in subjects with superior vermis lesion, for vowel /a/ and /u/ in female subject and for vowels /i/ and /u/ in male subject, compared to normal control subjects (Table 17).

Table 17: *Mean (Hz) and SD fornormals (N) and experimental groups and confidence intervals (CI) for normals for fundamental frequency (FO) in Hz. [(*) indicate values outside the confidence interval]*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (Hz) (SD)	116.92 (18.32)	119.12 (25.10)	117.31 (26.12)	112.66 (19.20)	116.00 (14.12)
CI	CI	114.63 to 199.21	-	-	-	-
/i/	Mean (Hz) (SD)	117.53 (14.03)	117.03 (22.91)	•111.66 (22.83)	116.61 (17.13)	117.03 (15.75)
	CI	114.75 to 120.30	-	-	-	-
/u/	Mean (Hz) (SD)	117.04 (15.43)	118.24 (24.01)	•110.33 (24.33)	115.33 (23.42)	115.24 (13.46)
	CI	113.77 to 120.31	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (Hz) (SD)	198.04 (21.31)	201.00 (24.21)	•183.50 (19.21)	197.33 (23.01)	195.66 (26.31)
	CI	185.21 to 209.29	-	-	-	-
IV	Mean (Hz) (SD)	196.32 (19.58)	198.00 (19.62)	181.23 (22.64)	196.86 (24.27)	194.44 (21.02)
	CI	176.74 to 215.90	-	-	-	-
/u/	Mean (Hz) (SD)	198.14 (18.06)	197.00 (21.87)	•184.33 (23.81)	197.66 (21.22)	194.33 (25.17)
	CI	187.33 to 210.75	-	-	-	-

FO is reportedly a highly inconsistent feature in ataxic dysarthria with some studies reporting lower FO (Brown et al., 1970; Chenery et al; 1990) and others a higher FO (Joanette & Dudley, 1980). Ackermann and Ziegler (1994) reasoned that higher FO is the general trend seen in subjects with ataxic dysarthria. In this study none of the experimental groups showed a higher FO compared to normal control subjects. The FO in right cerebellar group (right superior paravermal, right posterosuperior and right anterosuperior) and left cerebellar group (left superior paravermal, left anteroinferior) is comparable to that of normal control subjects, but is lower in subjects with superior vermis lesion. Studies have speculated the possible role of cerebellar vermis in aspects related to speech in general (Mills and Weisenburg, 1914; Holmes, 1917, 1922; Chiu et al.,

1996). The findings of the study seem to point to a possible role of the superior cerebellar vermis in controlling FO.

The findings of this study does not confirm with the general impression of a higher FO in ataxic dysarthria as seen in diffuse nonfocal lesions (Joanette and Dudley, 1980). It is too early to presume a trend based on cerebro - cerebellar interaction. The results indicate the possible role of the superior vermis in controlling FO, and not the right (right superior paravermal, right posterosuperior and right anterosuperior) and left (left superior paravermal, left anteroinferior) cerebellar regions.

(ii) Phonatory frequency range (PFR) (semitones)

Phonatory frequency range signifies the difference between the highest and lowest fundamental frequency and helps to infer the flexibility of the vocal system. Table 18 gives the Mean and SD for PFR for normal and experimental groups and the confidence intervals for normals.

The PFR for left (left superior paravermal, left antero inferior), superior vermis and right superior paravermal groups are higher than the normal control subjects (Table 18), signifying that the fine control of laryngeal musculature is affected in these groups. There is an unresolved controversy still continuing regarding whether it is the laryngeal muscles, or changes in subglottal pressure that controls FO (Ohala, 1978; 1990; Ladd, 1984).

Table 18: *Mean (semitones) and SD for normals and experimental groups and confidence intervals (CI) for normals for phonatory frequency range (PFR). [(*) indicates values outside the confidence interval.*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (Semitones) (SD)	4.95 (3.13)	•7.08 (4.61)	*16.27 (6.41)	5.12 (3.63)	5.44 (4.31)
CI	CI	3.98 to 5.92	-	-	-	-
/i/	Mean (Semitones) (SD)	3.19 (2.12)	•7.94 (5.01)	•15.51 (7.02)	3.63 (2.64)	4.01 (3.07)
	CI	2.29 to 4.09	-	-	-	-
/u/	Mean (Semitones) (SD)	2.82 (2.01)	•11.94 (4.33)	•14.46 (6.55)	2.94 (2.44)	3.02 (4.42)
	CI	2.54 to 3.09	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (Semitones) (SD)	3.53 (2.13)	•6.17 (3.15)	•13.47 (5.14)	•16.73 (4.15)	•5.40 (2.14)
	CI	2.31 to 4.75	-	-	-	-
/i/	Mean (Semitones) (SD)	3.02 (2.54)	•6.85 (2.98)	•15.97 (6.02)	•14.58 (5.12)	•5.99 (2.01)
	CI	2.61 to 3.42	-	-	-	-
Id	Mean (Semitones) (SD)	3.11 (2.27)	•9.11 (3.78)	•15.20 (5.87)	•11.04 (4.89)	•5.72 (2.54)
	CI	2.38 to 3.84	-	-	-	-

The increased FO range is generally attributed to inefficiency in the control of subglottal pressure (Collier, 1974), tracheal pull (Maeda, 1976) and inefficient vibration of the vocal folds or aspects related to laryngeal FO control (Strik and Boves, 1992). The fine control of laryngeal stiffness, stability and control of subglottal pressure required for maintenance of steady phonation may be differentially affected in subjects with cerebellar lesions restricted to different loci. Phonatory dysmetria is a well documented phenomenon in ataxic dysarthria due to nonfocal lesions (Cannito and Marquardt, 1997). Subjects with cerebellar lesions may be unable to gauge the range of vibratory movements of the vocal folds that are required for maintenance of steady FO due to phonatory dysmetria and hence there could be variations in FO. It may be speculated that the increased

phonatory frequency range in left superior paravermal, left anteroinferior, superior vermis and right superior paravermal groups may be because of phonatory dysmetria. It is also noticeable that FO is affected only in subjects with superior vermis lesions, whereas PFR is affected in left (left superior paravermal, left anteroinferior), superior vermis and right superior paravermal groups. FO is controlled mainly by laryngeal muscles (stiffness and vibratory pattern of vocal folds), whereas FO range is determined by vibration of vocal folds as well as changes in subglottal pressure. Thus it may be presumed that more number of cerebellar regions are involved in the control of coordinated activity of laryngeal and supralaryngeal regions as is required for FO range.

b] Frequency perturbation parameters

(i) Jitter percentage *(%)*

Jitter percentage indicates cycle to cycle variation in pitch and is a short term measure of FO. Higher values in Jitter percentage indicate irregular vocal fold vibration. Table 19 gives the Mean and SD for Jitter percentage for normal and experimental groups, along with confidence intervals for normals.

Jitter percentage is increased for subjects with left (left superior paravermal, left anteroinferior), superior vermis (SV) and right superior paravermal (RSP) lesions compared to normal control group (Table 19). It may be speculated that increased Jitter percentage in these lesions may be due to irregular vibration of the vocal folds. Increased jitter is generally caused due to irregular neuromuscular excitation at the level of cricothyroid muscles and vocal folds (Baer, 1980).

T a b l e 19: *Mean (%) and SD for normals and experimental groups and confidence interval (CI) for normals for Jitter percentage (%). [(*) indicates values outside the confidence interval].*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (%) (SD)	0.68 (0.34)	1.41* (0.61)	1.38* (0.87)	0.70 (0.64)	0.76 (0.51)
CI	CI	0.55 to 0.81	-	-	-	-
/i/	Mean (%) (SD)	0.76 (0.37)	1.42* (0.74)	1.39 (0.79)	0.84 (0.53)	0.79 (0.62)
	CI	0.60 to 0.91	-	-	-	-
/u/	Mean (%) (SD)	0.65 (0.28)	1.11* (0.70)	1.49* (0.98)	0.73 (0.71)	0.69 (0.58)
	CI	0.54 to 0.77	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%) (SD)	0.90 (0.32)	1.02* (0.76)	1.62* (0.98)	1.18* (0.81)	1.71* (0.52)
	CI	0.75 to 1.00	-	-	-	-
/i/	Mean (%) (SD)	1.08 (0.47)	1.63* (0.84)	1.77* (0.87)	1.51* (0.93)	1.70* (0.61)
	CI	0.77 to 1.40	-	-	-	-
/u/	Mean (%) (SD)	0.97 (0.39)	1.31* (0.98)	1.79* (0.91)	1.38* (0.74)	1.54* (0.58)
	CI	0.70 to 1.23	-	-	-	-

Changes in stiffness of the vocal fold, mass of vibrating structures and changes in subglottal air pressure are cited as possible reasons for increased jitter (Isshiki, 1964; Lieberman and Blumstein, 1988). Abnormal neuromuscular excitation could have lead to changes in vocal fold stiffness and subglottal air pressure in subjects with lesions in left superior paravermal, left anteroinferior, superior vermis and right superior paravermal regions of the cerebellum, only. However, this cannot be generalized as it requires substantive data from physiological analysis.

Ackermann and Ziegler (1994) reported increased jitter and pitch fluctuations during sustained vowel phonation in subjects with cerebellar atrophy (restricted to the cerebellum), suggesting reduced stability of vocal fold oscillations and thus impaired phonatory functions. Such a trend is observed only in left superior paravermal, left anteroinferior, superior vermis and right superior paravermal groups in this study. Interestingly however, subjects with right posterosuperior (RPS) and right anterosuperior (RAS) groups did not show any difference from that of normal control subjects. This may imply that right posterosuperior and right anterosuperior regions of the cerebellum are not involved in phonatory function as reflected by the jitter measures. It can also be seen that the jitter is increased in like cognate pairs of left superior paravermal as well as right superior paravermal regions of the cerebellum, whereas it is comparable to normal controls in right posterosuperior and right anterosuperior regions of the cerebellum. It is also noticeable that the same cerebellar regions (left superior paravermal, left anteroinferior, superior vermis and right superior paravermal) may be involved in controlling aspects related to phonatory frequency range and short term perturbation measure like jitter. It may be that anatomical correlates underlying the control of FO variations (frequency range and jitter) are localized within left superior paravermal, left anteroinferior, superior vermis and right superior paravermal regions of the cerebellum.

(ii) Smoothedpitch perturbation quotient (SPPQ) *(%)*

SPPQ indicates long term cycle to cycle variation in pitch (over 55 cycles). SPPQ is the relative evaluation of the short or long term variability of pitch period within the analyzed voice sample at a defined smoothing factor (default of 55

periods used). That is, it averages the variability of pitch periods across 55 periods. The smoothing factor reduces the sensitivity of the SPPQ to pitch extraction errors and hence it is considered to be a more reliable perturbation measure. Table 20 gives the Mean and SD for SPPQ for normal and experimental groups along with confidence intervals for normals.

Table 20: *Mean (%) and SDfor normals and experimental groups and confidence interval (CI) for normals for Smoothed pitch perturbation quotient (%) [(*) indicates that the Mean values are outside the confidence interval]*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (%) (SD)	0.73 (0.23)	*5.13 (2.14)	•12.66 (5.02)	0.81 (3.41)	0.74 (4.35)
CI	CI	0.62 to 0.84	-	-	-	-
/i/	Mean (%) (SD)	0.60 (0.31)	•6.32 (3.01)	•11.80 (4.98)	0.63 (2.33)	0.61 (4..33)
	CI	0.54 to 0.66	-	-	-	-
lul	Mean (%) (SD)	0.62 (0.34)	•6.31 (3.71)	•11.65 (6.78)	0.62 (3.92)	0.63 (4.33)
	CI	0.58 to 0.66	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%) (SD)	0.79 (0.31)	•4.58 (2.82)	•7.41 (4.31)	•9.86 (3.24)	•1.72 (5.12)
	CI	0.52 to 1.05	-	-	-	-
/i/	Mean (%) (SD)	0.87 (0.28)	•5.61 (5.01)	•7..35 (3.87)	•9.84 (4.47)	•2.22 (6.82)
	CI	0.59 to 1.14	-	-	-	-
lul	Mean (%) (SD)	0.91 (0.37)	•6.01 (2.61)	•4.64 (2.24)	•8.82 (3.51)	•3.23 (4.56)
	CI	0.62 to 1.20	-	-	-	-

The results for SPPQ are similar to the results seen for Jitter percentage. SPPQ is increased for left superior paravermal, left anteroinferior, superior vermis and right superior paravermal groups compared to normal controls (Table 20). In other words it may be inferred that left superior paravermal, left anteroinferior,

superior vermis and right superior paravermal groups demonstrated increased FO variations even after a smoothing factor is applied. The smoothing factor helps to smooth out most of the variations in FO, except for major variations in FO. Hence it may be speculated that pitch variations due to irregular vocal fold vibrations or irregular changes in subglottal pressure (Isshiki, 1964; Lieberman and Blumstein, 1988) are more obvious in left superior paravermal, left anteroinferior, superior vermis and right superior paravermal regions of the cerebellum and the functional correlates related to FO variations could be specific to these cerebellar regions. SPPQ (%) is comparable to normal control group in subjects with right posterosuperior (RPS) and right anterosuperior (RAS) lesions. These regions do not seem to play an important role in controlling FO variations. It may be inferred that anatomical correlates related to cycle to cycle variations in FO may have bilateral representation in the cerebellum, atleast in the left as well as right superior paravermal regions of the cerebellum. The posterior as well as anterior regions of the superior portion of the right cerebellum may not be involved in controlling FO variations as SPPQ (%) is comparable to that of normal controls.

The results obtained for PFR, Jitter percentage and SPPQ (%) indicate that same anatomical regions (left superior paravermal, left anteroinferior, superior vermis and right superior paravermal) may be involved in controlling aspects related to absolute FO measures (PFR) and short term FO measures (Jitter percentage and variation in fundamental frequency).

(ii) Variation in fundamental frequency (vFO) (%)

Variation in fundamental frequency (vFO %) is calculated as the relative standard deviation of the period-to-period calculated fundamental frequency (standard deviation of FO / FO). It reflects the very long term variations of FO for the analyzed sample. Table 21 gives the Mean and SD for vFO (%) for normal and experimental groups and the confidence intervals for normals.

Table 21: *Mean (%) and SD for normals and experimental groups and confidence interval (CI) for normals for variation in fundamental frequency (%). [(*) indicates that the Mean values are outside the confidence interval]*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (%) (SD)	1.23 (0.98)	*6.26** (2.41)	14.15* (5.62)	2.54* (3.42)	1.45* (2.01)
CI	CI	1.10 to 1.36	-	-	-	-
/i/	Mean (%) (SD)	1.25 (1.01)	6.54* (2.53)	13.69* (4.31)	2.08* (2.87)	1.53* (1.98)
	CI	1.10 to 1.41	-	-	-	-
/u/	Mean (%) (SD)	1.44 (0.84)	5.20* (2.01)	12.72* (5.02)	1.91* (1.97)	1.73* (2.77)
	CI	1.27 to 1.62	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%) (SD)	1.29 (0.89)	7.55* (3.02)	13.03* (3.43)	26.35* (9.02)	3.39* (1.12)
	CI	1.06 to 1.51	-	-	-	-
/i/	Mean (%) (SD)	1.35 (0.98)	6.51* (2.41)	12..36* (4.11)	21.99* (9.47)	3.58* (1.03)
	CI	1.17 to 1.54	-	-	-	-
/u/	Mean (%) (SD)	1.50 (0.93)	5.63* (2.11)	12.27* (4.58)	18.61* (10.13)	4.58* (2.12)
	CI	1.27 to 1.74	-	-	-	-

vFO (%) is increased in the left (left superior paravermal, left anteroinferior), superior vermis as well as the right cerebellar groups (right superior paravermal, right posterosuperior, right anterosuperior) compared to normal controls (Table 21). Thus all the cerebellar regions included in this study

seem to be involved in controlling aspects related to vFO (%). It may be presumed that anatomical correlates underlying long term variations in FO may be localized in more number of cerebellar regions compared to localization of neural correlates related to absolute measures of FO and short term variations of FO. vFO (%) is increased in subjects with right as well as left superior paravermal lesions. Interestingly, vFO (%) is increased in right posterosuperior and right anterosuperior lesions unlike for PFR or short term frequency perturbation measures (Jitter percentage and SPPQ). This indicates that right posterosuperior and right anterosuperior regions may only be involved in controlling long term frequency perturbation measures.

Increased vFO (%) is a characteristic finding reported in ataxic dysarthric subjects with nonfocal lesions (Zwirner et al., 1991; Ackermann and Ziegler, 1994; Kent et al., 1997; Hertrich et al., 1998; Kent et al., 2000). Boutsen, Duffy and Dimassi (2004) reported vocal tremor (quasi-periodic long term fluctuations) in majority of subjects with extracerebellar pathology and a single subject with atrophy confined to the cerebellum. Ackermann and Ziegler (1994) explained the long term frequency modulations in ataxic dysarthric subjects as due to isometric contraction of the internal laryngeal muscles. The results of the present study indicate that long term phonatory instability may be a common feature in ataxic dysarthric subjects with lesion either in the left and right cerebellar regions and superior verm is and isometric contraction of internal laryngeal muscles during sustained phonation may be a common feature of dysarthria, irrespective of the site of lesion in the cerebellum.

Summary

The results suggest definite trends with respect to functional control of various parameters (absolute, short and long term measures of FO) obtained from sustained phonation of vowels /a/,/i/, and /u/. Neural correlates underlying the production of FO seem to be localized only in the superior vermis region of the cerebellum, whereas those underlying phonatory frequency range, Jitter percentage and Smoothed amplitude perturbation quotient are localized in more regions of the cerebellum (left superior paravermal, left anteroinferior, superior vermis and right superior paravermal). Long term measure of frequency perturbation (vFO %) is increased in all the experimental groups compared to normal controls.

Neural correlates for precise coordinated movements as is required for steady FO (phonatory frequency range and short term perturbations) seem to be localized only in the left superior paravermal, left anteroinferior, superior vermis and right superior paravermal regions of the cerebellum. Aspects controlling long term frequency perturbation (vFO %) seem to be localized in more regions of the cerebellum. These differences may be due to the differences in the subsystems controlling these parameters. FO control mainly involves laryngeal control whereas variations in FO (short and long term) involves laryngeal control as well as changes in subglottal pressure. It is seen that left (left superior paravermal, left anteroinferior), right superior paravermal as well as superior vermis regions of the cerebellum are involved when coordination between different subsystems are required (FO variations), whereas absolute FO which principally involves laryngeal control seem to be represented only in the superior vermis region of the

cerebellum. AH cerebellar regions irrespective of lesions (right, left as well as superior vermis) may be involved in neural control of long term frequency related measures. There seems to be bilateral representation of neural correlates for the absolute frequency variations (PFR) as well as short and long term measures, as these measures were found to be increased in both right and left superior paravermal regions. The right posterosuperior as well as right anterosuperior seem to be involved only in controlling the long term frequency perturbation measures.

A disruption in the cerebellar control system leads to adjustment in the gain of proprioceptive loops mediated through extracerebellar structures which inturn leads to pitch fluctuations and this is a well documented phenomenon in ataxic dysarthria due to nonfocal lesions (Mackay & Murphy, 1979; Ackermann and Ziegler, 1994). This finding may hold good for subjects with lesions restricted to the cerebellum also, as these subjects also showed increased long term pitch fluctuations. However, the findings need to be substantiated with larger sample size and from physiological studies.

c] Amplitude perturbation parameters

(i) Shimmer percentage *(%)*

Shimmer percentage indicates short term cycle to cycle amplitude perturbation. Table 22 gives the Mean and SD for Shimmer percentage for normal and experimental groups and the confidence intervals for normals.

Table 22: *Mean (%) and SD for normals and experimental groups and confidence interval (CI) for normals for Shimmer percentage (%). [(*) indicates that the Mean values are outside the confidence interval]*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (%) (SD)	3.08 (1.83)	•6.11 (3.1.2)	*10.32 (4.12)	3.36 (1.51)	3.23 (2.56)
Cl	*Cl*	2.20 to 3.40	-	-	-	-
/i/	Mean(%) (SD)	2.51 (0.91)	*5.93 (2.32)	*10.58 (3.02)	2.76 (4.96)	2.64 (3.04)
	Cl	2.22 to 2.81	-	-	-	-
/u/	Mean (%) (SD)	2.47 (0.74)	*7.02 (3.41)	*10.14 (4.74)	2.58 (5.44)	2.66 (2.98)
	Cl	2.23 to 2.71	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%) (SD)	3.13 (0.93)	***5.08** (2.10)	*9.72 (3.12)	*11..9O (3.82)	*5.15 (5.71)
	Cl	2.58 to 3.68	-	-	-	-
/i/	Mean (%) (SD)	2.63 0.81	*6.14 (3.11)	•7.60 (2.34)	* 10.30 (3.41)	•4.06 (2.07)
	Cl	2.06 to 3.20	-	-	-	-
/u/	Mean (%) (SD)	2.98 0.71	*5.54 (3.78)	* 10.62 (4.27)	*9.79 (5.81)	•5.18 (3.47)
	Cl	2.49 to 3.48	-	-	-	

Shimmer percentage is higher in subjects with left (left superior paravermal, left anteroinferior), superior vermis (SV) and right superior paravermal (RSP) lesion compared to normal control subjects (Table 22). These findings are similar to that observed for phonatory frequency range as well as short term frequency perturbations. Increased Shimmer percentage could be attributed to the inability of the subjects to maintain a constant intensity in phonation due to changes in vocal fold tension or stiffness, changes in subglottal air pressure and mass of vibrating structures (Isshiki, 1964; Lieberman and Blumstein, 1988). The subglottic pressure depends on the volume of airflow and the degree of adduction of vocal folds. Respiratory insufficiency or dysregulation

are often reported in subjects with ataxic dysarthria due to nonfocal cerebellar lesions (Tatsumi, et al., 1979; Murdoch et al., 1991; Cisneros & Braun, 1995; Deger, Ziegler & Wessel, 1999; Kent et al., 2000). It may be presumed that airflow volume and vocal fold adduction are inadequate in subjects with left superior paravermal, left anteroinferior, superior vermis and right superior paravermal lesions. Increased Shimmer percentage was reported as a general feature in ataxic dysarthria in subjects with diffuse lesions by Kent et al., (2000). The results in Table 22 indicate that neural correlates controlling short term intensity variations may be localized only in left superior paravermal, left anteroinferior, superior vermis and right superior paravermal regions of the cerebellum as increased Shimmer percentage could be noticed only in subjects with these lesions. Neural correlates associated with short term intensity variations also seem to have bilateral representation as is evident for jitter, as both the right and left superior paravermal areas also show increased jitter. The right posterosuperior and right anterosuperior regions are not involved in controlling aspects related to shimmer as the values are comparable to normal controls in subjects with lesions in these cerebellar regions.

(ii) Smoothed amplitude perturbation quotient (SAPQ) *(%)*

Smoothed amplitude perturbation quotient (SAPQ %) is defined as the relative evaluation of the short or long term variability of the peak-to-peak amplitude within the analyzed sample at a defined smoothing factor (default of 55 periods used). It averages the variability of peak amplitude across 55 periods. The smoothing factor reduces the sensitivity of the SAPQ to amplitude extraction

errors. Table 23 gives the Mean and SD for SAPQ for normal and experimental groups, including the confidence intervals for normals.

Table 23: *Mean (%) and SDfor normals and experimental groups and confidence interval (CI) for normals for Smoothed amplitude perturbation quotient (%). [(*) indicates that the Mean values are outside the confidence interval]*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (%) (SD)	4.26 (2.10)	• 12.54 (4.25)	•8.49 (3.54)	4.55 (4.02)	4.42 (2.01)
CI	CI	3.89 to 4.62	-	-	-	-
/i/	Mean (%) (SD)	3.95 (1.98)	•5.06 (2.10)	•5.28 (2.43)	4.02 (1.87)	4.31 (1.93)
	CI	3.47 to 4.43	-	-	-	-
/u/	Mean (%) (SD)	4.23 (2.23)	•5.61 (1.87)	•5.33 (2.52)	4.52 (1.76)	4.68 (1.83)
	CI	3.71 to 4.76	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%) (SD)	4.28 (2.01)	•5.55 (3.45)	•7.58 (4.01)	•9.91 (5.27)	5.02 (2.71)
	CI	2.27 to 6.29	-	-	-	-
/i/	Mean (%) (SD)	3.73 (1.44)	•5.21 (2.14)	•5.47 (1.91)	•6.94 (4.53)	3.68 (3.02)
	CI	3.01 to 4.44	-	-	-	-
/u/	Mean (%) (SD)	3.90 (1.31)	4.52 (2.76)	•7.30 (3.44)	•8.42 (5.24)	•5.14 (2.25)
	CI	3.23 to 4.56	-	-	-	-

SAPQ is increased in the left (left superior paravermal, left anteroinferior) and superior verm is group (Table 23). SAPQ is comparable to normal control subjects in all the groups with right hemispheric lesions except for increased for vowel *I'll* for right superior paravermal subjects. Dysfunctions at the level of vocal fold vibrations or irregular changes in subglottic pressure could be the reason for increased amplitude perturbation. The amplitude perturbations are evident despite the use of smoothing factor. The smoothing factor (55 cycles in this study)

smoothens most of the variations in amplitude and hence amplitude perturbations are observed only if the intensity variation are very conspicuous. The results in Table 23 indicate that shorterm amplitude perturbations are more in subjects with left (left superior paravermal, left anteroinferior) and superior vermis lesions. In contrast to the findings obtained for short (Jitter percentage and SPPQ) and long term frequency variations (vFO %) and for short term amplitude perturbation (Shimmer percentage), the right superior paravermal region is not implicated in short term variations in amplitude, when a smoothing factor is applied. This indicates that short term amplitude variations are not very conspicuous in subjects with right superior paravermal lesions. In contrast to the findings for short term amplitude perturbations, the right superior paravermal region is involved in the control of short term pitch fluctuations (Jitter percentage and Smoothed pitch perturbation quotient). The neural correlates for control of short term pitch perturbations are not present in right posterosuperior and right anterosuperior lesions.

(Hi) Variation in amplitude (vAm) (%)

Variation in amplitude (vAm %) indicates long term peak to peak variations in amplitude. Increased vAm (%) indicates reduced ability to maintain sound pressure level and and reduced ability to regulate subglottal pressure by proper adduction of vocal folds. Table 24 gives the Mean and SD for variation in amplitude for normal and experimental groups and confidence intervals for normals.

Table 24: *Mean (%) and SDfor normals and experimental groups and confidence interval (CI) for normals for variation in amplitude (%). [(*) indicates that the Mean values are outside the confidence interval]*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (%) (SD)	12.42 (3.41)	*26.51 (11.36)	*39.93 (14.31)	13.01 (4.56)	12.71 (3.45)
CI	CI	11.56 to 13.28	-	-	-	-
/i/	Mean (%) (SD)	10.66 (4.57)	*18.01 (12.42)	*26.40 (13.04)	•12.81 (3.07)	•13.16 (2.98)
	CI	9.49 to 11.83	-	-	-	-
/u/	Mean (%) (SD)	12.10 (5.02)	*24.01 (9.47)	*26.75 (9.83)	•15.42 (5.14)	13.01 (4.01)
	CI	10.84 to 13.35	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%) (SD)	12.08 (3.58)	*39.49 (11.31)	*21.52 (8.74)	•34.78 (8.90)	•15.86 (7.52)
	CI	10.75 to 13.41	-	-	-	-
/i/	Mean (%) (SD)	13.51 (1.02)	•23.69 (14.04)	*18.31 (7.98)	•21.14 (10.12)	13.25 (7.22)
	CI	11.90 to 15.81	-	-	-	-
/u/	Mean (%) (SD).	13.83 (1.03)	* 18.25 (9.83)	•24.32 (6.04)	•26.19 (9.13)	14.50 (9.11)
	CI	12.40 to 15.55	-	-	-	-

vAm (%) is increased in subjects with left (left superior paravermal, left anteroinferior), superior vermis and right posterosuperior lesions compared to normal control subjects (Table 24). It could be that coordination of vocal fold adductions and volume of airflow which inturn controls subglottal pressure is affected in these experimental groups. vAm (%) is increased for vowels /i/ and /u/ in right posterosuperior and for vowel /i/ in right anterosuperior lesion. Increased vAm (%) observed in subjects with right posterosuperior lesions is unlike the findings seen in measures related to short term measures of frequency and amplitude and also unlike that observed for long term measures of frequency variation (vFO). Neural correlates for long term measures of amplitude

perturbation does not seem to implicate the right superior paravermal region and right anterosuperior region as vAm (%) in this group is comparable to that of normal group. vAm (%) is increased in the left superior paravermal region of the cerebellum. The right posterosuperior region of the cerebellum alone seems to be implicated in long term amplitude perturbation.

Several studies have reported increased long-term variability of amplitude (vAm %) in ataxic dysarthria due to nonfocal lesions (Kent et al., 1997; Hertrich, et al., 1998; Kent et al., 2000). The results in Table 24 indicate that vAm (%) is increased only in subjects with left superior paravermal, left anteroinferior, superior vermis and right posterosuperior lesions. The results for vAm (%) for lesions restricted to cerebellum indicate that right superior paravermal as well as right anterosuperior regions may not be involved in controlling aspects related to vAm(%).

Summary

Short (Shimmer percentage and SAPQ) and long term (vAm %) amplitude perturbation measures are increased in subjects with left (left superior paravermal, left anterosuperior) and superior vermis lesions compared to normal controls. This indicates that the subjects in these experimental groups are not able to maintain steady intensity in phonation for short and long durations. In addition to these groups, Shimmer percentage is also increased in subjects with right superior paravermal lesion and vAm (%) is also increased in subjects with right posterosuperior lesion. The right anterosuperior region of the cerebellum may not have neural correlates related to amplitude control as short as well as long term

measures of amplitude perturbation in this group are comparable to that of normal controls. Short as well as long term measures of amplitude perturbation is increased in subjects with left superior paravermal lesions whereas short term measure of Shimmer percentage alone is increased in subjects with right superior paravermal lesion. Short term amplitude perturbation measures in subjects with right anterosuperior and right posterosuperior lesions are comparable to normal control subjects. Long term amplitude perturbation measure (vAm) is increased only in subjects with right posterosuperior lesion.

Table 25: Summary of frequency and amplitude related parameters

(*) *indicates parameters that are deviant from normal controls*

Fundamental frequency related parameters						
	LSP	LAI	SV	RSP	RPS	RAS
FO (Hz)			*			
PFR	*	*	*			
Frequency perturbation related parameters						
	LSP	LAI	SV	RSP	RPS	RAS
Jitt(%)	*	*	*			
SPPQ (%)	*	*	*			
vFO (%)	*	*	*		*	*
Amplitude perturbation related parameters						
Shim (%)	*	*		*		
SAPQ (%)	*	*	*			
vAm (%)	*	*	*			

FO is reduced in subjects with superior vermis lesions and comparable to normal control subjects in all other experimental groups. It can be seen that absolute FO measure of phonatory frequency range (PFR), short and long term amplitude perturbation and Shimmer percentage is increased in the left (left superior paravermal, left anteroinferior), superior vermis and right superior paravermal regions. This leads to the assumption that control of short and long

term measures of frequency and amplitude perturbation may arise from dysfunctions in the same neural correlates of left superior paravermal, left anteroinferior and superior vermis regions of the cerebellum. The right superior paravermal region seems to be involved in controlling aspects related to short and long term measures of frequency. Only short term amplitude perturbation (Shimmer percentage) is increased in subjects with right superior paravermal lesions. The left superior paravermal region of the cerebellum controls neural correlates related to short and long term FO and amplitude variations. The right posterosuperior region of the cerebellum seems to be involved in controlling only long term FO and amplitude measures. Right anterosuperior region is involved only in the control of long term FO perturbation (vFO).

These findings imply definite trends that can be seen with respect to localization of neural correlates of frequency and amplitude perturbation parameters in different regions of the cerebellum.

d] Noise related parameters

(i) Noise ***to harmonic ratio (NHR)***

NHR is a ratio of the in-harmonic energy in the range 1500-4500Hz to the harmonic spectral energy (70 - 4500Hz). Table 26 gives the Mean and SD for NHR for normal and experimental groups and confidence intervals for normals.

Table 26: *Mean (%) and SD for normals and experimental groups and confidence interval (CI) for normals for noise to harmonic ratio. [(*) indicates that the Mean values are outside the confidence interval]*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (%) (SD)	0.14 (1.18)	*0.16 (3.09)	*0.21 (5.10)	•0.18 (4.14)	0.14 (2.17)
	CI	0.13 to 0.15	-	-	-	-
/i/	Mean(%) (SD)	0.12 (2.27)	0.13 (3.62)	•0.31 (4.18)	•0.15 (5.19)	0.124 (3.83)
	CI	0.11 to 0.13	-	-	-	-
/u/	Mean (%) (SD)	0.13 (1.09)	•0.15 (3.92)	*0.18 (2.64)	•0.15 (3.56)	•0.10 (4.91)
	CI	0.12 to 0.14	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%) (SD)	0.13 (1.98)	0.14 (4.73)	•0.24 (6.42)	•0.22 (5.18)	•0.17 (3.96)
	CI	0.12 to 0.15	-	-	-	-
/i/	Mean(%) (SD)	0.11 (1.08)	0.11 (3.97)	•0.22 (5.72)	•0.22 (4.61)	•0.18 (3.82)
	CI	0.08 to 0.14	-	-	-	-
/u/	Mean (%) (SD)	0.14 (2.07)	0.16 (3.70)	•0.18 (4.66)	•0.24 (3.03)	*0.18 (3.68)
	CI	0.12 to 0.16	0.07	0.12	0.13	0.10

NHR is increased in most of the experimental groups compared to normal control subjects. Exceptions are present in female subject with left anteroinferior lesion and subject with right anterosuperior lesion (Table 26), where the measures of NHR for these subjects are comparable to normal control subjects. The increase in NHR in subjects with left superior paravermal, left anteroinferior (male), superior verm is, right superior paravermal and right posterosuperior lesions mean that harmonic energy in the speech range (70 - 4500 Hz) is reduced in these experimental groups due to aperiodic vocal fold vibratory patterns. It could also mean that in-harmonic energy in the range of 1500 Hz to 4500 Hz is increased in these subjects compared to harmonic energy. This increase in noise (in-harmonic

energy) may be due to inadequate vocal fold adductions and escape of excess air through the glottis during phonation resulting in frication noise. This noise is reflected as higher noise level in the spectrum (Hillenbrand, 1987; Krom, 1993). Superior paravermal regions in the left as well as the right cerebellar regions and right posterosuperior regions seem to be involved in controlling movements of the vocal folds which reflect in the production of harmonics in the speech spectrum. The right anterosuperior regions of the cerebellum are not involved in controlling movements of the vocal folds that reflect in the production of harmonics in the speech spectrum.

(ii) Soft Phonation Index (SPI)

Soft phonation index is a ratio of the lower-frequency (70-1600Hz) to the higher frequency (1600-4500Hz) harmonic energy. Table 27 gives the Mean and SD for Soft Phonation Index for normal and experimental groups and the confidence intervals for normals.

SPI is increased in subjects with left (left superior paravermal), superior vermis and right cerebellar (right superior paravermal and right posterosuperior) lesions as can be seen from Table 27. Increased SPI indicates increase in lower frequency harmonic energy (i.e. noise) or loss of harmonic energy in the high frequency range (as is required for speech). Increased SPI indicates inadequate vocal fold adduction leading to excess air leakage through the glottis, producing inharmonic energy in the spectrum. SPI for vowels /a/ and /i/ are comparable to normal control subjects in female subject with left anteroinferior lesion and subject with right anterosuperior lesion.

Table 2 *7: Mean (%) and SD for normals and experimental groups and confidence interval (CI) for normals for soft phonation index (SPI). [(*) indicates that the Mean values are outside the confidence interval]*

Groups		N	LAI	SV	RPS	RAS
Gender		*Male*	*Male*	*Male*	*Male*	*Male*
/a/	Mean (%) (SD)	14.13 (4.12)	•23.11 (8.56)	*36.66 (12.16)	*20.03 (9.87)	15.01 (5.33)
CI	CI	12.30 to 15.96	-	-	-	-
/i/	Mean (%) (SD)	14.05 3.54	14.91 (4.92)	*28.12 (11.01)	•26.04 (12.26)	17.21 (7.01)
	CI	10.51 to 17.59	-	-	-	-
/u/	Mean (%) (SD)	15.45 5.02	•36.28 (15.14)	•31.14 (9.87)	•24.94 (10.51)	•32.76 (10.87)
	CI	13.39 to 17.51	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%) (SD)	12.58 5.58	14.62 (5.44)	*52.50 (19.21)	•34.23 (9.42)	•31.33 (13.43)
	CI	9.95 to 15.20	-	-	-	-
/i/	Mean (%) (SD)	12.42 4.12	13.61 (6.89)	*36.50 (13.47)	•29.66 (11.23)	•24.33 (11.57)
	CI	9.45 to 15.39	-	-	-	-
/u/	Mean (%) (SD)	13,49 4.08	* 16.03 (7.81)	•44.60 (10.13)	•40.90 (14.01)	•29.66 (10.58)
	CI	10.25 to 16.72	-	-	-	-

This indicates that low frequency harmonic energy is reduced compared to high frequency harmonic energy in these subjects leading to the presumption that vocal fold adduction is adequate in these subjects. Hertrich et al., (1998) reported remarkably low SPI in their subjects with atrophy confined to the cerebellum. The results of this study do not agree with the findings of Hertrich et al., (1998). In the present study, a differential effect can be seen with SPI increased only in subjects with left superior paravermal, superior vermis, right superior paravermal and right posterosuperior lesions. Subjects with left superior paravermal as well as right superior paravermal lesions show increased NHR compared to normal controls. NHR is increased in subjects with right postero superior lesions whereas it is comparable to normal controls in subjects with right anterosuperior lesions.

Summary

NHR and SPI are increased in subjects with left superior paravermal, left anteroinferior (male subject), superior vermis, right superior paravermal and right posterosuperior lesions. This indicates that inharmonic energy (noise) is increased in these subjects compared to normal control subjects. This may be attributed to inadequate vocal fold adduction which inturn leads to excessive air leakage through the glottis. NHR and SPI for subjects with left anteroinferior lesion (male) and subject with right anterosuperior lesion is comparable to normal controls. The right posterosuperior lesion is implicated only in the control of long term frequency and amplitude perturbations. Increased NHR and SPI in subjects with right posterosuperior lesions indicate that this region is involved in controlling vocal fold vibrations as well as subglottal pressure variations required for steady phonation. The right anterosuperior region of the cerebellum may not be involved in controlling these factors as NHR and SPI is comparable to normals.

Summary of all spectral parameters

Fundamental frequency related parameters (FO & PFR) were differently affected for different lesions associated with cerebellum. Left (left superior paravermal and left anteroinferior) and right (right superior paravermal, right posterosuperior, subject with right anterosuperior) cerebellar lesions were comparable to normal control subjects for FO, whereas subjects with superior vermis lesion had reduced FO. Phonatory frequency range was increased in left (left superior paravermal, left anteroinferior), superior vermis (SV) and right superior paravermal lesions, whereas it was reduced in subjects with right posterosuperior and right anterosuperior lesions.

Frequency and amplitude perturbation parameters also showed some degree of differential representation in the cerebellum. Frequency perturbation parameters of Jitter and smoothed pitch perturbation quotient were increased in all subjects with cerebellar lesions except for right posterosuperior and right anterosuperior lesions. Differential representation with respect to variation in fundamental frequency could not be observed, as all the cerebellar lesions had increased vFO (%). Amplitude perturbation parameters also showed some degree of differential representation. This was reflected as increased shimmer percentage in subjects representing all lesions except right posterosuperior and subject with right anterosuperior lesion. For smoothed amplitude perturbation quotient, in addition to right posterosuperior and subject with right anterosuperior lesions, right superior paravermal lesions were also comparable to normals. Variation in amplitude showed a different trend as lesions associated with right superior paravermal and right anterosuperior alone were comparable to normals.

Subjects with left anteroinferior and right anterosuperior lesions presented similar values of NHR and SPI as that of normal control subjects. All other experimental groups (left superior paravermal, superior vermis, right superior paravermal and right posterosuperior) had increased NHR and SPI compared to normal control subjects. The subjects with left (left superior paravermal and left anteroinferior lesions), superior vermis and right superior paravermal lesions had increased short term frequency perturbation measures (Jitt, SPPQ), whereas subjects with right posterosuperior as well as right anterosuperior lesions were comparable to normal control subjects. All the groups (left, superior vermis, right)

had increased long term frequency perturbation (vFO %). The subjects with left (left superior paravermal and left anteroinferior), superior vermis and right superior paravermal lesions had increased short term amplitude perturbation measures (Shim & SAPQ) indicative of the fact that neural correlates underlying short term amplitude perturbations may be localized to these regions. Shimmer percentage was comparable to normal controls in subjects with right posterosuperior as well as right anterosuperior lesions. All the right cerebellar groups (right superior paravermal, right posterosuperior and right anterosuperior) showed increased short term amplitude perturbation when a smoothing factor was applied (i.e. increased SAPQ). Long term amplitude perturbation (vAm %) is increased in subjects with left (left superior paravermal and left anteroinferior), superior vermis and right superior paravermal lesions. The findings for vAm (%), are similar to the findings for Shimmer percentage.

II. Articulatory tasks

A. Perceptual measures

I. Word repetition task

A total of 86 stimuli words were used to test 11 vowels, 1 diphthong, 32 consonants and four consonant clusters. Details are provided in Method section and Appendix 3. Articulation analysis was done only for the experimental groups. Two judges transcribed the data of the subjects using IPA narrow transcription method and the errors were analysed in terms of substitution, omission, distortion and addition. Attempt was also made to analyse phonological processes as per Stoel-Gammon and Dunn (1985) and Lowe (1986, 1994). The processes that were looked into under Syllable-structure processes included:

- Unstressed - Syllable Deletion
- Diminutization
- Epenthesis
- Final-Consonant Deletion
- Initial-Consonant Deletion
- Cluster reduction

The substitution processes were analyzed in terms of:

- Stopping
- Deaffrication
- Velar Fronting
- Backing
- Depalatalization
- Liquid gliding
- Vocalization

Assimilation processes were looked into in terms of:

- Labial Assimilation
- Velar Assimilation
- Nasal Assimilation
- Alveolar Assimilation
- Prevocalic voicing
- Postvocalic Devoicing

Percent agreement between the first and second judge, second and third judge and first and third judge was calculated using the formula:

$$\% \text{ agreement} = \frac{\text{Total number of sounds in agreement}}{\text{Total number of sounds}}$$

Where, Total number of sounds = Total number of agreements + total number of disagreements.

Percent agreement for error analysis between the first and second judge was 95.34%, second and third judge was 89.53 % and between first and third judge was 94.18%. The same three judges re-analysed the samples of 5 experimental subjects after 3 months and responses were recorded and the percent agreement for error analysis between the first and second judge was 91.86%, second and third judge was 88.37% and first and third judge was 91.86%.

A seven point severity rating scale was adopted for perceptual judgement of sample of narration. The scale was as follows:

1 = normal speech
2 = mild
3 = mild to moderate
4 = moderate
5 = moderate to severe
6 = severe
7 = Profound

Ratings of severity of dysarthria were done by three judges. Severity was rated as mild in all the experimental groups except subject ST (left anteroinferior lesion) and RN (right anterosuperior lesion). Severity was rated as mild to moderate in subjects ST and RN.

1 (a) Vowel error patterns

Among subjects with left superior paravermal lesion, no articulation errors were observed except for subject NB who showed breathiness for short vowel /A/. Subjects with left anteroinferior lesions did not show any vowel errors. In subjects with superior vermis lesion, subject MK showed prolongation of/u/ and /o/ in the medial position [/u/ as /u:/ and /o/ as /o:/]. Subject BT, with lesion in the superior vermis showed breathiness associated with all vowels as well as diphthongs.

Vowel errors were absent in right superior paravermal, right posterosuperior and right anterosuperior subjects. Vowels /A/ and /o/ were associated with breathiness for subject ST, with right superior paravermal lesion. Less number of vowel errors may be attributed to the mild severity of dysarthria in most of the experimental subjects.

/ *(b) Consonant error patterns*

With regard to consonants, the nasal sounds were not affected in any of the place of articulation (bilabial, velar, palatal, dental, alveolar, retroflex). Stops were not affected in any of the place of articulation (bilabial, velar, dental and retroflex), for any of the subjects in the experimental group.

Subject TJ, with left superior paravermal lesion, showed derhotacization of alveolar flap *Ixl* and alveolar trill /R/ in the initial as well as medial position. Also, there is fronting of retroflex lateral approximant *l\l* for this subject. Subjects with left anteroinferior lesion did not show any errors for consonants. The alveolar flap *Ixl* is derhotacized for subject MK with lesion in the superior vermis. For subject BT with superior vermis lesion, there is derhotacization of /r/ and fronting of /i/. Subject SP with right superior paravermal lesion showed derhotacization of /r/ and /R/. Subject ST with right superior paravermal lesion showed fronting of /i/. Subject RN with right anterosuperior lesions show fronting of retroflex fricative /s/ and retroflex lateral approximant /i/..

The spatial errors were observed in terms of substitution (S), omission (O), distortion (D) and addition (A) and phonological processes. The results show that

substitution, omission and addition errors were not present in any of the dysarthric groups. Also, fronting was the only phonological process that could be identified in the analysis of the samples of experimental subjects. However, it is noticeable that it is seen only in few subjects (TJ, BT, MK, ST & RN) in the experimental group. These subjects had lesions in the left superior paravermal, superior vermis, left anteroinferior and right anterosuperior regions of the cerebellum, respectively. The few articulatory errors may be due to the mild degree of severity of dysarthria in most of the experimental subjects.

Although the errors associated with distortion were very less, based on the complexity of utterance, a pattern was evident from the data. Errors were minimal in all dysarthric groups. Also, there were very few vowel errors when compared to consonantal errors. The consonantal errors were mostly restricted to rhotacization of alveolar flap *hi* and alveolar trill /R/ and fronting of retroflex lateral approximant *l\j*. The reason that errors were associated with only these consonants may have to do with the difficulty in production of these sounds in Malayalam language (Syamala Kumari, 1972). In the production of the voiced alveolar flap /r/, the tongue touches the alveolar ridge for a single tap. The rest of the tongue is low and during the tap it is laterally contracted. The soft palate is raised and the vocal cord vibrates. During the production of the alveolar trill /R/ in Malayalam, the tongue makes rapid vibrations against the alveolar ridge. The soft palate is raised to prevent the escape of air through the nasal cavity and there is vibration of the vocal cords. For the production of/i/ in Malayalam, the tongue tip is curled back and it reaches towards the palate making a partial closure. The air is let out through the sides as well as over the tongue, with slight friction. The soft palate is

raised and the vocal cords vibrate. Considering the fact that slow articulatory movements are a characteristic feature of ataxic dysarthria (Kent and Netsell, 1975., Kent and Rosenbek, 1982), and the presumed difficulty in production of/r/, /R/ and /i/ in Malayalam language could have contributed to increased frequency of errors on these consonants than others.

Subjects with ataxic dysarthria associated with diffuse or multifocal lesions demonstrated more errors in the noninitial position of words and substitution errors were more than distortion errors (Odell et al., 1991). In this study, there were very few errors observed in the dysarthric subjects and when present, they were mostly in the medial positions. Zyski and Weisiger (1987) and Zeplin and Kent (1996) observed vowel distortions and imprecise consonants in words of ataxic dysarthric subjects due to diffuse or multifocal lesions. It is noticeable that vowel distortions and consonantal errors were very few and only associated with few of the subjects with lesions restricted to the cerebellum. Out of twenty phonological processes that were looked for in the transcribed data, fronting is the only process that could be identified. The very few vowel and consonant errors in the experimental groups may be because of the mild severity of dysarthria of the experimental subjects. It would have been interesting to see if the trend remained the same with more severe forms of dysarthria. The results obtained for the word repetition task cannot be generalized due to the small sample size.

B. Measures of DDK

Temporal and intensity measures were extracted for AMR and SMR tasks. The diadochokinetic (DDK) task involved rapid repetition of the syllables in an alternating manner for AMR task for the syllables /pA/, /tA/ and /kA/ and a sequential manner for the SMR task for the syllable sequence /pAtAkA/. The intention of including an AMR and SMR task was to analyze the temporal measures of diadochokinetic task with increasing task complexity. According to Tjaden and Watling (2003), the task of SMR is more complex than the AMR task, as SMR involves rapid repetition of syllable sequence /pAtAkA/ whereas AMR involves rapid repetition of the same syllable. Measures of total duration ('td'), syllable duration ('sd'), closure duration ('cd') ('td'='sd'+'cd') were obtained for the stimuli in AMR as well as SMR tasks. Section (a), (b) and (c) represents the temporal measures for AMR and SMR respectively. Section (d) and (e) represents the amplitude measures for AMR and SMR tasks respectively.

(a.1) Total duration ('td'), syllable duration ('sd') and closure duration ('cd') in AMR task

Total duration ('td'), syllable duration ('sd') and closure duration ('cd') were obtained for ten consecutive syllables from the stimuli obtained by fast repetition of the syllables */pA/*, /tA/ and /kA/. Total duration ('td') was operationally defined as the sum of syllable duration ('sd') and closure duration ('cd') for the syllable ('td'= 'sd'+ 'cd'). Syllable duration was operationally defined as the temporal interval between the vertical spike corresponding to burst release and the offset of glottal pulsing for the following vowel. Closure duration was operationally defined as the interval between the offset of glottal pulsing for a

vowel and the burst release for a consonant. For voiceless stop consonants, the closure duration is silent; because the vocal tract is occluded and the vocal folds are not vibrating (voicing energy is absent) ((as per the criteria by Ozawa et al., 2001).

The findings for 'td\ 'sd' and 'cd' for AMR task for normal control group (N) and experimental groups for the syllables /p/v/, /W and *l\uJ* are presented in Table 28 and Table 29 respectively.

Table 28: *Mean, SD and confidence intervals (CI) for normal control subjects (N) for AMR for total duration ('td'), syllable duration ('sd') and closure duration ('cd') for syllables /pA/./tA/ and /kA/..*

		/pA/			**/tA/**			/kA/		
N		Mean (ms)	SD	CI	Mean (ms)	SD	CI	Mean (ms)	SD	CI
	'td'	136.03	10.01	128.45 to 144.37	141.00	12.44	136.53 to 148.32	131.26	16.84	123.25 to 138.56
	'sd'	73.75	16.12	67.21 to 79.22	79.56	15.03	74.10 to 84.33	70.66	12.44	53.22 to 88.10
	'cd'	62.28	15.25	57.21 to 68.31	61.44	16.36	55.03 to 66.49	60.60	14.21	56.34 to 64.23
	'sd': 'cd'	*1.2:1*	-	-	*1.3:1*	-	-	*1.2:1*	-	

Table 29: *Mean and SD for total duration ('td'), syllable duration ('sd') and closure duration ('cd')for the experimental groups for syllables /pA/, /tA/ and /kA/ in AMR task (*) indicates values that are outside confidence interval*

		/pA/		/tA/		/kA/	
Experimental group	parameter	Mean (ms)	SD	Mean (ms)	SD	Mean (ms)	SD
LSP	'td'	129.55	18.02	140.81	17.91	129.32	16.34
	'sd'	72.22	24.21	79.77	22.02	72.66	19.04
	'cd'	57.33	21.23	61.04	19.41	56.66	24.21
	'sd':'cd'	1.26:1	-	1.31:1	-	1.3:1	-
LAI	'td'	133.83	14.03	138.37	22.45	132.51	15.68
	'sd'	68.83	18.44	82.33	20.32	72.51	24.11
	'cd'	65.00	23.53	56.00	18.20	60.00	17.64
	'sd':'cd'	1.06:1	-	1.47:1	-	1.21:1	-
SV	'td'	134.72	28.31	139.93	25.38	140.24	30.24
	'sd'	78.86	26.43	77.73	32.36	76.08	34.24
	'cd'	57.86	17.53	62.20	36.11	64.16	27.13
	'sd':'cd'	1.41:1	-	1.25:1	-	1.34:1	-
RSP	'td'	*154.32	18.47	*163.00	16.87	*174.97	22.78
	'sd'	*87.66	27.31	*86.00	21.05	*84.97	32.04
	'cd'	66.66	25.02	*77.00	23.33	*90.00	28.41
	'sd':'cd'	1.32:1	-	1.12:1	-	0.94:1	-
RPS	'td'	133.88	15.72	140.33	20.52	131.11	22.23
	'sd'	*89.22	19.41	77.00	21.31	66.78	24.33
	'cd'	*44.66	17.44	63.33	17.83	64.03	18.56
	'sd':'cd'	2:1	-	1.22:1	-	1.04:1	-
RAS	'td'	*165.24	21.03	*179.46	24.01	*158.56	15.42
	'sd'	*89.23	16.24	*92.34	18.42	81.04	16.71
	'cd'	*76.01	14.34	*87.12	17.71	*77.52	20.13
	'sd':'cd'	1.17:1	-	1.06:1	-	1.05:1	-

Total duration ('td') is comparable to normal controls for all the experimental groups (left superior paravermal, left anteroinferior, superior vermis, right posterosuperior), except in subjects with right superior paravermal and right anterosuperior lesions. These two groups showed increased 'td' for syllables /pA/, /tA/ and /kA/. Total duration for the syllable /pa/ for subjects with right posterosuperior lesions is comparable to normal controls despite increased syllable duration and closure duration ('sd' and 'cd' outside confidence interval)

(Table 29). This is because 'sd' is increased compared to normals whereas 'cd' is reduced compared to normals. The results for total duration ('td') for syllables /pA/, /tA/ and /k/\ are given for right superior paravermal, right posterosuperior and right anterosuperior groups in Figure 13, Figure 14 and Figure 15 respectively.

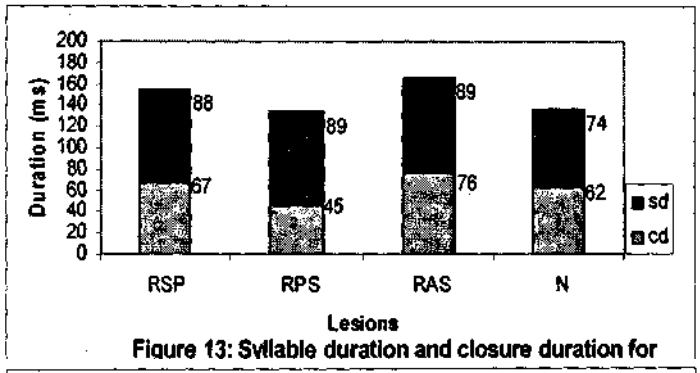

Figure 13: Syllable duration and closure duration for

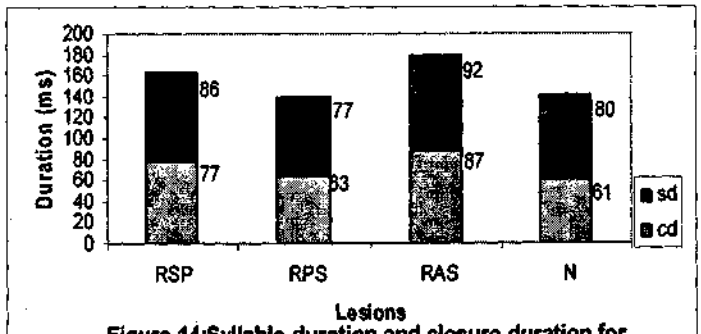

Figure 14:Syllable duration and closure duration for /ta/ for AMR:Right superior paravermal, right posterosuperior, right anterosuperior and normal controls

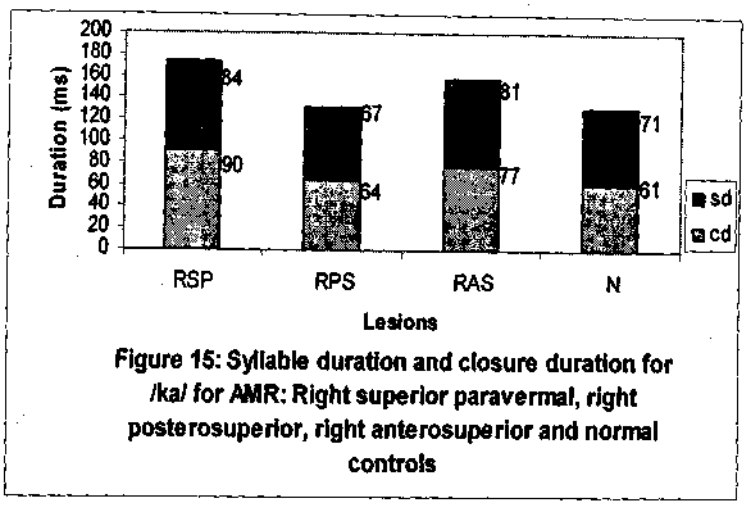

Figure 15: Syllable duration and closure duration for /ka/ for AMR: Right superior paravermal, right posterosuperior, right anterosuperior and normal controls

It can be seen that total duration is increased in subjects with right superior paravermal and right posterosuperior lesions compared to normal controls (Figure 13 to Figure 15). The increased 'td' in right superior paravermal and right anterosuperior is due to increased 'sd' and increased 'cd' (Table 29). These results agree with the findings of Kent et al., (2000) who attributed increased DDK durations in ataxic dysarthric subjects to increase in syllable duration as well as closure duration.

Boutsen and Christman (2002) speculated that durational parameters in speech is controlled by the right cerebellar hemisphere because of its contralateral connection with the left cereberal hemisphere and the proposed role of the left cerebral hemisphere in controlling durational aspects of speech. The results as seen in Table 29 raises interesting questions as to whether the right superior paravermal and right anterosuperior regions of the cerebellum are involved in maintaining syllable durations. However, not all right cerebellar regions seem to be involved in controlling total duration of syllable. It is observed that the total

duration of syllables are not increased in subjects with right posterosuperior lesions.

(b.I) sd:cd ratio in AMR

The trend seen for ratio of syllable duration to closure duration (sdxd ratio) for all the experimental groups (left superior paravermal, left anteroinferior, superior vermis, right superior paravermal, right posterosuperior, right anterosuperior) are comparable to that of normal controls for the syllables */pA/,* /tA/ and /k/v/. In both normal controls and experimental groups, syllable duration is more than closure duration implying that the execution phase ('sd') is more than the 'preparatory phase' ('cd'). Syllable duration and closure duration are almost equal for syllable /kA/ in subjects with right superior paravermal lesion. These findings are contrary to the observation of Ozawa et al., (2001) who opined that 'cd' contributes more than 'sd' to increased 'td' in ataxic dysarthric subjects with nonfocal lesions.

According to Kent et al., (1998), speech diadochokinetic task involves the coordination of several motor systems such as peripheral nerve innervations and different neural regulatory loops. That is, the syllables */p/\/,* /tA/ and/kA/ can reveal different manifestations based on the extent of involvement of the peripheral nerve innervations and different neural regulatory loops in the production of these syllables. Kent et al., (1997) reported that faster repetition of syllable /kA/ was more affected in subjects with nonfocal cerebellar lesions. The findings in this study for subjects with lesions restricted to different cerebellar regions do not reveal any such trend. Ozawa et al., (2001) reported that the tongue related sounds

(/tA/ and /kA/) were more prolonged than lip related sound *(/pA/)* in subjects with degenerative cerebellar disease. Definite trends with respect to tongue and lip related sounds are not seen in subjects with lesions restricted to the cerebellum.

(c.l) DDK rate in AMR

DDK rate for AMR task was operationally defined as the number of syllables in the fast repetition of the syllables */pA/, /tA/* and /kA/ that is uttered in a second. Total time taken to utter each of the syllables was measured from the waveform display in CSL - 4400. The total number of syllables were counted from the waveform display. DDK rate was calculated by the number of syllables uttered in a second.

The results of AMR rate for normal control subjects and experimental groups are given in Table 30.

Table 30: *AMR rate (syll / s) for normal control group (N) and experimental groups and confidence intervals (CI) for normal controls for syllables /pA/, /tA/ and /kA/. [(*) indicates values outside confidence interval]*

Group	**/PA/**			**/tA/**			/kA/		
	Mean (syll/sec)	SD	CI	Mean (syll/sec)	SD	CI	Mean (syll/sec)	SD	CI
N	6.62	5.67	5.57 to 7.13	6.38	7.13	5.66 to 7.01	5.80	6.41	5.11 to 6.41
LSP	5.68	4.72	-	5.73	15.61	-	5.14	9.46	-
LAI	5.84	8.51	-	5.77	13.44	-	5.50	11.13	-
SV	5.73	14.45	-	5.69	9.03	-	5.48	12.24	-
RSP	*4.02	9.43	-	•4.38	8.72	-	*3.97	10.58	-
RPS	5.59	16.13	-	5.72	14.58	-	5.20	13.06	-
RAS	**•4.41**	8.20	-	*4.33	10.23	-	*3.92	9.42	-

From Table 30, it is seen that AMR rate is reduced for all three syllables in right superior paravermal as well as right anterosuperior groups, whereas all other

groups show values comparable to that of normal control group. Variability in repetition rate is considered a crucial feature to aid in the diagnosis of ataxic dysarthria due to nonfocal lesions (Brown et al., 1970; Chenery et al., 1990; Murdoch and Theodoras, 1998). The results, as seen in Table 30 indicate that in comparison to normal controls, variability is increased for syllable /p/\ in subjects with lesion in the superior vermis and right posterosuperior regions, increased for syllable /tA/ in subjects with left superior paravermal, left anteroinferior, right posterosuperior and right anterosuperior lesions.

Variability for syllable /kA/ is increased in all experimental groups except for the groups with left superior paravermal and right anterosuperior lesions. The increased variability in some of the experimental groups may indicate reduced motor stability in these groups.

Analysis was also done for stimuli obtained from SMR task to obtain total duration, syllable duration and closure duration. Syllable rate (syll/s) was also calculated for the SMR task. AMR task involves fast repetition of the same syllable whereas SMR task involves fast repetition of a sequence of syllables. Hence SMR task is more difficult than AMR task (Forrest, Weismer & Turner, 1989; Tjaden and Watling, 2003).

The results obtained for SMR task is given in (a.2), (b.2) and (c.2).

(a.2) Total duration ('td), syllable duration ('sd') and closure duration fed') in SMRtask

The total duration ('td), syllable duration ('sd') and closure duration ('cd') were analysed for three syllables for /pA/, /tA/ & /kA/ each in three consecutive syllable sequences of /pAtAkA/ in the SMR task. The findings for 'td', 'sd' and 'cd' for SMR task for normal control group (N) and experimental groups are presented in Table 31 and Table 32 respectively.

' Table 31: *Mean, SD and confidence interval (CI) for normal control subjects (N) for SMR for total duration ('td'), syllable duration (sd) and closure duration (cd) for /pA/ /tA/ and /kA/*

		/pA/			/tA/			/kA/		
N		Mean (ms)	SD	CI	Mean (ms)	SD	CI	Mean (ms)	SD	CI
	'td'	160.95	14.82	153.12 to 167.81	156.13	23.04	149.04 to 163.61	160.27	21.71	154.17 to 167.22
	'sd'	88.21	18.02	82.01 to 93.51	92.01	21.03	85.03 to 98.11	94.08	15.44	87.43 to 101.21
	'cd'	72.74	17.02	67.33 to 78.01	64.12	16.43	58.13 to 71.12	66.19	19.44.	59.34 to 72.41
	sdxd	*1.21:1*	-	-	*1.43:1*	-	-	*1.42:1*	-	-

Table 32: *Mean and SD for total duration ('td'), syllable duration ('sd') and closure duration ('cd') for the experimental subjects for /pA/, /tA/ and /kA/ in SMR task. [(*) indicates values outside confidence interval*

		/PA/		/tA/		/kA/	
Lesion	parameter	Mean (ms)	SD	Mean (ms)	SD	Mean (ms)	SD
LSP	'td'	*202.86	19.73	•182.96	26.15	•198.16	15.08
	'sd'	* 100.20	28.21	•111.16	34.42	•119.96	36.51
	'cd'	* 102.66	23.33	•71.80	19.41	•78.20	18.53
	sd:cd	0.98:1	-	1.50:1	-	1.53:1	-
LAI	'td'	•201.10	21.62	•184.70	21.78	•212.10	16.82
	'sd'	•108.10	24.73	•115.40	30.85	•126.60	33.23
	'cd'	*93.00	17.04	69.30	25.52	•85.50	28.04
	sdxd	1.20:1	-	1.76:1	-	1.50:1	-
SV	'td'	•221.14	28.14	•222.34	32.38	*222.72	31.71
	'sd'	•119.60	34.63	•132.32	38.90	•134.52	24.20
	'cd'	•101.54	31.52	•90.02	23.57	•88.20	42.17
	sd:cd	1.20:1	-	1.50:1	-	1.53:1	-
RSP	'td'	•260.32	24.14	•271.03	19.74	•280.32	26.79
	'sd'	•136.66	28.03	•147.00	24.41	•148.66	18.44
	'cd'	•123.66	17.81	•124.03	34.33	•131.66	33.04
	sd:cd	1.10:1	-	1.20:1	-	1.13:1	-
RPS	'td'	•216.36	26.91	•229.66	29.62	•238.36	27.93
	'sd'	•124.33	32.01	•135.33	40.32	•143.06	34.23
	'cd'	•92.03	36.21	•94.33	34.33	•95.30	19.51
	sdxd	1.35:1	-	1.43:1	-	1.50:1	-
RAS	'td'	•243.14	19.73	•255.50	25.80	•289.50	23.58
	'sd'	•141.00	34.40	•130.50	21.51	•138.50	22.12
	'cd'	•102.00	26.73	•125.00	32.64	•151.00	18.60
	sdxd	1.40:1	-	1.04:1	-	0.92:1	-

Total duration is increased compared to normal controls in all the experimental groups (left superior paravermal, left anteroinferior, superior vermis, right superior paravermal, right posterosuperior & right anterosuperior) (Table 32). In all the experimental groups, increase in 'td' is due to increase in 'sd' as well as 'cd'. In subjects with left anteroinferior lesions, increased 'sd' alone ('cd' comparable to normal control subjects) contributed to increased 'td' for syllable /tA/. The results in this study as can be seen from Table 32 indicate that even the

left (left superior paravermal, left anteroinferior) and superior vermis regions of the cerebellum may be involved in controlling durational aspects of speech.

The left (left superior paravermal, left anteroinferior), superior vermis and right posterosuperior regions of the cerebellum do not seem to be involved in durational aspects related to AMR task. Differential representation within the cerebellum is not evident for SMR task, as 'td' is increased in all the experimental groups. This may have to do with the complexity of the DDK task involved, as the sequential motion rate requires fast production of the sequence /pAtAkA/, whereas the alternating motion rate requires the alternation of the same syllable only (Forrest et al., 1989; Tjaden and Watling, 2003). This leads to the observation that more complex tasks (like SMR) seem to be represented in more regions of the cerebellum than simpler tasks like AMR.

(b.2) sd:cd ***ratio in SMR task***

Syllable duration is increased compared to closure duration for all three syllables for normal control group in the SMR task (Table 32). All the experimental groups also show similar trend to that of normal controls with increased syllable duration compared to closure duration in SMR task (Table 32). This may imply that the 'execution phase' (i.e. 'sd') is more than the 'preparatory phase' (i.e. 'cd'). Syllable duration is comparable to closure duration for syllable /pA/ in subjects with left superior paravermal lesions.

(c.2.) DDK rate in SMR task

DDK rate for SMR task was operationally defined as the number of syllables in the fast repetition of the syllable sequence /pAtAkA/ that is uttered in a second. Total time taken to utter the syllable sequences was measured from the waveform display in CSL - 4400. The total number of syllables were counted from the waveform display. DDK rate was calculated by the number of syllables uttered in a second. Table 33 represents the SMR rate for the syllable sequence /pataka/.

Table 33: *SMR rate for normal control group (N) and experimental subjects for syllable sequence /pA tAkA/ and confidence interval for normal control subjects.*

Group	Rate (syll/sec)	SD	CI
N	5.54	14.89	4.97 to 6.03
LSP	*4.4l	18.20	-
LAI	*4.33	16.83	-
SV	*3.95	23.12	-
RSP	*3.05	24.89	-
RAS	•3.12	15.32	-
RPS	*3.89	21.67	-

Results in Table 33 reveal that SMR rate is reduced for all the experimental groups compared to normal controls. The variability is increased in all the experimental groups (particularly for subjects with left superior paravermal, superior vermis, right superior paravermal and right posterosuperior lesions), compared to normal controls. The findings for SMR rate are different from that for AMR task. AMR rate is reduced only in subjects with right superior paravermal and right anterosuperior lesions, whereas SMR rate is reduced in all the experimental groups. The results in this study for AMR rate and SMR rate as given in Table 30 and Table 33 respectively indicate that SMR rate appear to be a

general indicator of dysarthria associated with all lesions restricted to the cerebellum, while AMR rate appear to be sensitive to dysarthria associated with specific lesions in the cerebellum.

Studies have suggested that SMR task is more difficult in terms of utterance complexity than AMR task. The multiple articulatory configurations for /pAtAkA/ or SMR task is more complex than the AMR tasks, which require only a single articulatory configuration (Forrest et al., 1989; Tjaden and Watling, 2003). The difference in DDK rate in AMR and SMR tasks may be due to differences in task complexity. Given a trade-off between movement speed and movement accuracy, all the experimental groups may have used a slower rate in the SMR task to assure accurate syllable productions, as per task requirement to produce syllables both rapidly and precisely.

d) Intensity measures

Reduced amplitude maxima in DDK tasks (Kent et al., 2000), and variability in amplitude maxima and amplitude minima across a syllable train is reported to be a characteristic feature of ataxic dysarthria (Kent et al., 1999; Kent et al., 2000). Intensity variations in DDK repetitions (AMR and SMR) have been examined less thoroughly than temporal characteristics of DDK in ataxic dysarthric subjects.

The measure of minimum and maximum intensity measures for the syllables /pA/, /tA/, /kA/ (AMR) and syllable sequence */pAtAtkA/* (SMR) were obtained by placing the cursors on the trough of the energy contour for a syllable

(for minimum intensity) and then on the corresponding peak (for maximum intensity) for that syllable (as per Kent et al., 1999). These measures were obtained for 10 consecutive syllables for the AMR task and for three consecutive syllable sequences in the SMR task. Energy minima (minimum intensity) and energy maxima (maximum intensity) were averaged for each speaker.

d.1) Minimum and maximum intensity for AMR

The minimum intensity, maximum intensity and intensity range for AMR task in AMR task for normal control group (N) and experimental groups are given in Table 34 and Table 35 respectively.

Table 34: *Mean, SD and confidence interval (Cl)for normal control subjects (N) for minimum and maximum intensity for /pA/,/t A/ and /kA/ for AMR*

		/pA/			/tA/			/kA/		
N		Mean (dB)	SD	CI	Mean (dB)	SD	CI	Mean (dB)	SD	CI
	Min IO	48.28	14.42	45.21 to 52.03	46.13	18.23	44.11 to 51.07	48.27	19.67	44.56 to 52.83
	Max 10	70.23	18.35	67.01 to 74.82	73.94	15.02	69.81 to 77.56	72.83	13.14	68.02 to 76.01
	10 range	21.95	15.69	18.15 to 23.76	27.81	16.71	18.03 to 25.97	24.56	16.23	19.13 to 27.89

Table 35: *Mean and SDfor minimum and maximum intensity (dB) and intensity range (10 range) for the experimental groups for /pA/, /tA/ and /kA/ for AMR*

	/pA/		/tA/		/kA/	
Lesion	Mean (dB)	SD	Mean (dB)	SD	Mean (dB)	SD
Minimum intensity (dB)						
LSP	51.49	24.33	*54.95	26.04	*53.10	34.24
LAI	*53.15	22.12	*54.30	18.02	52.03	36.41
SV	*54.28	27.03	*53.28	24.24	*53.82	34.11
RSP	50.61	20.02	*52.28	18.41	51.63	22.33
RPS	48.99	21.52	50.55	24.44	49.05	18.03
RAS	49.03	18.01	48.25	16.33	51.87	21.53
Maximum intensity (dB)						
LSP	*62.93	15.22	*64.80	21.24	*60.31	20.13
LAI	•60.88	16.41	*62.22	14.32	*66.21	24.12
SV	*62.65	18.21	*64.36	21.03	*63.65	28.40
RSP	*66.28	21.12	*65.62	19.42	*66.22	24.44
RPS	69.75	18.13	*68.73	23.14	68.47	26.52
RAS	* 64.80	16.43	*67.93	15.33	*66.20	20.01
10 range for AMR						
LSP	* 11.44	24.33	*9.85	26.04	•7.21	34.24
LAI	*7.73	22.12	* 7.92	18.02	*13.58	36.41
SV	*8.37	27.03	*11.08	24.24	*9.83^	34.11
RSP	*15.67	20.02	* 13.34	18.41	* 14.59	22.33
RPS	20.76	21.52	18.18	24.44	19.42	18.03
RAS	* 15.75	18.01	19.68	16.33	* 14.33	21.53

Intensity range is reduced in subjects with left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right anterosuperior) cerebellar lesions. 10 range in subjects with posterosuperior lesions is comparable to normal control subjects (Table 35 and Figure 16 to Figure 24). 10 range of /tA/ for subjects with right anterosuperior lesion is comparable to that of normal control subjects (Table 35).The bars in the Figure 16 to Figure 24 indicate intensity range (minimum and maximum intensity) for experimental groups and the upper and lower lines indicate maximum and minimum intensity for normal controls.

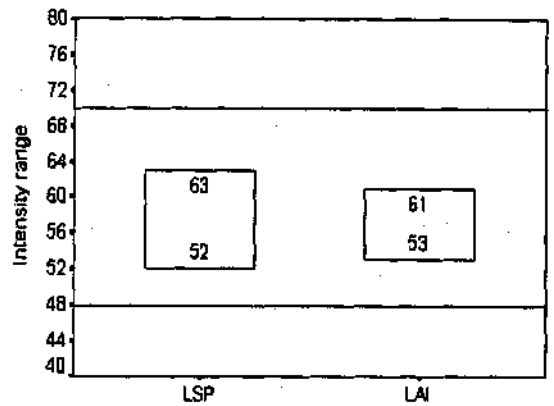

Figure 16: Intensity range for syllable /pa/ in AMR task: Left superior paravermal, left anteroinferior and normal controls

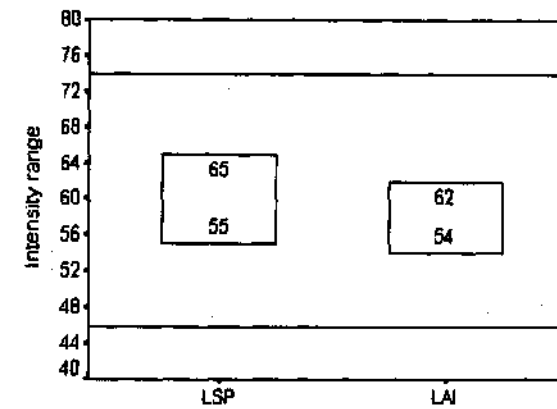

Figure 17: Intensity range for syllable /ta/ in AMR task: Left superior paravermal, left anteroinferior and normals

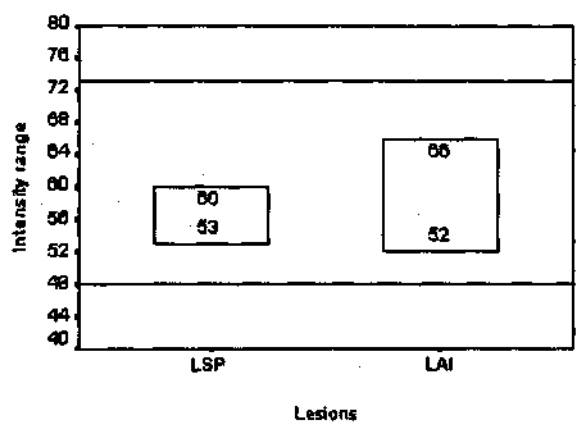

Figure 18: Intensity range for syllable /ka/ in AMR task: Left superior paravermal, left anteroinferior and normals

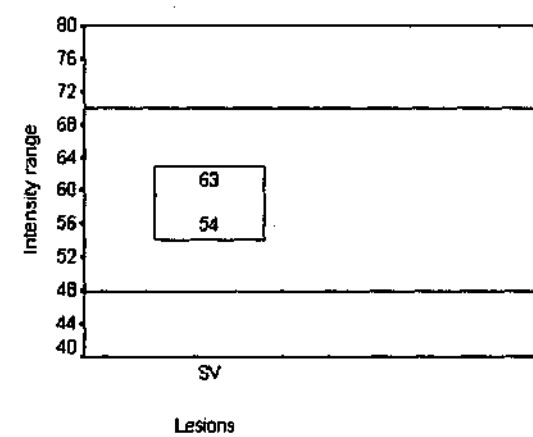

Figure 19: Intensity range for syllable /pa/ in AMR task: Superior vermis and normal controls

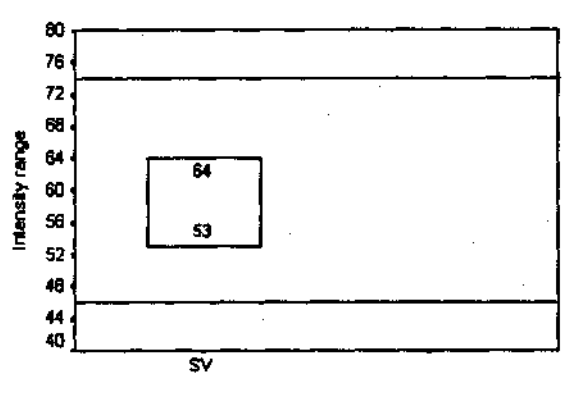

Figure 20: Intensity range for syllable /ta/ in AMR task: Superior vermis and normal control subjects

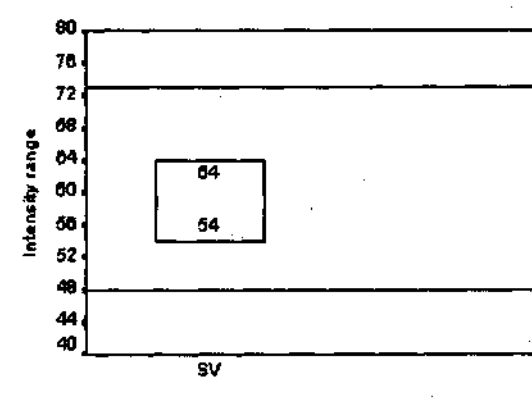

Figure 21: Intensity range for syllable /ka/ in AMR task: Superior vermis and normal controls

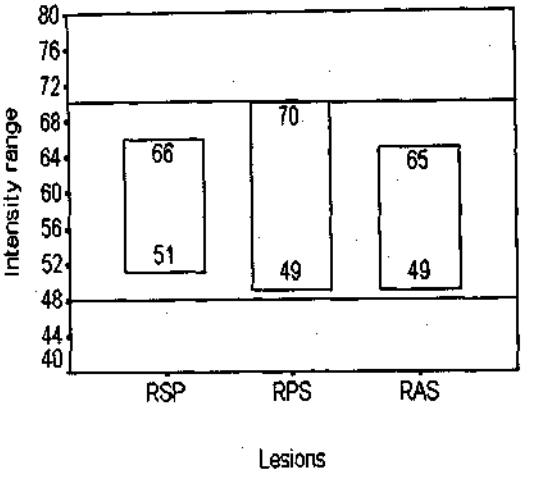

Figure 22: Intensity range for /pa/ in AMR: Right superior para vermal, right posterosuperior, right anterosuperior & normals

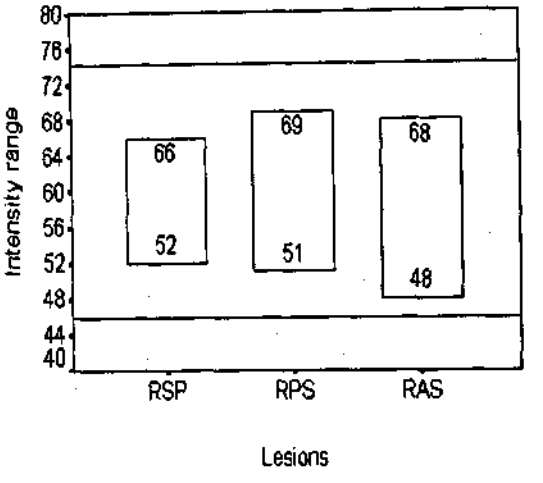

Figure 23: Intensity range for /ta/ in AMR: Right superior para vermal, right posterosuperior, right anterosuperior & normals

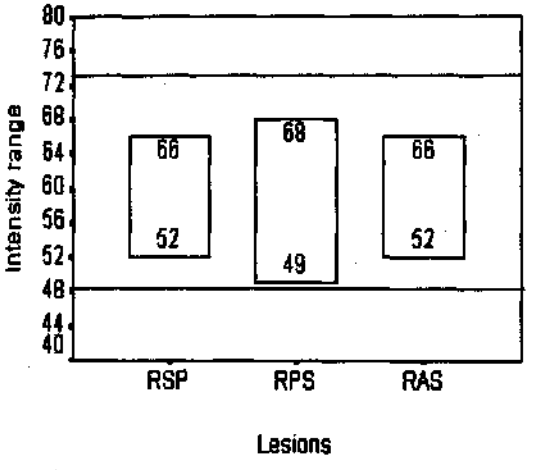

Figure 24: Intensity range for /ka/ in AMR: Right superior para vermal, right posterosuperior, right anterosuperior and normals

Reduced energy maxima and variable vocal intensity in DDK tasks is reported in individuals with dysarthria (Murdoch et al., 1991). Reduced 10 range for /tA/ and /kA/ in subjects with left superior paravermal lesions, for /pA/ and /to/ in left anteroinferior lesions, for /pA/, */tA/* and /tA/ in subjects with superior vermis lesions and for /tA/ in subjects with right superior paravermal lesions is due to higher energy minima and lower energy maxima. The reduced range for /pA/ in subjects with left superior paravermal lesions, /k^/ in subjects with left anteroinferior lesions, /pA/ and /k^/ in subjects with right superior paravermal and right anterosuperior lesions are because of reduced energy maxima (Table 35).

Respiratory insufficiency or dysregulation are often reported in subjects with ataxic dysarthria due to nonfocal cerebellar lesions (Tatsumi, et al., 1979; Murdoch et al., 1991; Cisneros & Braun, 1995; Deger et al., 1999; Kent et al., 2000). According to Brown et al., (1970); Chenery et al., (1990) and Murdoch and Theodoras (1998) and Kent et al., (2000), variability in loudness in DDK task is considered a crucial feature in the diagnosis of ataxic dysarthria due to nonfocal lesions. The reduced 10 range due to an increase in energy minima or a decrease in energy maxima or both in subjects with left superior paravermal, left anteroinferior, superior vermis, right superior paravermal and right anterosuperior lesions could be indicative of insufficient respiratory support for maintenance of steady AMR. It may be too early to presume an underlying respiratory cause for these findings as further studies are required in subjects with focal cerebellar lesions.

Variability is high for energy minima of syllable /pA/ in subjects with superior vermis lesion, for syllable /kA. in subjects with left superior paravermal, left anteroinferior and superior vermis lesions, compared to normal control subjects. Kent et al., (2000) opined that increased variability of energy minima could be due to poor coordination of voicing and articulation. It may be presumed that coordination of voicing and articulation is affected in subjects with left superior paravermal, left anteroinferior and superior vermis lesions. Increased variability of energy minima is a characteristic feature of ataxic dysarthria due to nonfocal lesions (Kent et al., 1999; Kent et al., 2000). The results in Table 35 indicate that increased variability of energy minima is a characteristic feature of subjects with left superior paravermal, left anteroinferior and superior vermis lesions, only. Increased variability for energy maxima is associated only with syllable /kA/ and can be seen in all experimental groups (Table 35). Increased variability of energy maxima reflects respiratory instability or dyscoordination. Hence it may be inferred that respiratory dyscoordination is a characteristic feature of subjects with left (left superior paravermal, left anteroinferior), superior vermis and right cerebellar (right superior paravermal, right posterosuperior and right anterosuperior) lesions. The results in Table 35 indicate that tongue back sound (syllable /kA/) shows more variability than tongue blade or tongue tip sounds for energy maxima.

a.2) Minimum and maximum intensity for SMR

The findings for minimum and maximum intensity and intensity range for SMR task for normal control group (N) and experimental groups are presented in Table 36 and Table 37 respectively.

Table 36: *Mean, SD and confidence interval (C) for normal control subjects (N) for SMRfor minimum and maximum intensity (dB) and intensity range for /pA/, /tA/ and /kA/.*

		/pA/			/tA/			/kA/		
N		Mean (dB)	SD	CI	Mean (dB)	SD	CI	Mean (dB)	SD	CI
	MinIO	49.83	18.41	46.41 to 53.12	48.28	22.05	45.98 to 51.07	49.27	24.33	46.03 to 52.33
	Max 10	69.91	17.06	67.03 to 73.57	70.82	21.14	69.81 to 77.56	69.83	19.23	67.93 to 73.23
	10 range	20.08	14.28	17.56 to 23.42	22.54	18.54	19.98 to 26.68	20.56	16.89	17.04 to 24.01

Table 37: *Mean and SD for minimum and maximum intensity (dB) and intensity range (10 range) for the experimental groups for /p/\/, /tA/ and /KA/ for SMR*

	/pA/		/tA/		/kA/	
Lesion	Mean (dB)	SD	Mean (dB)	SD	Mean (dB)	SD
Minimum intensity (dB)						
LSP	52.63	28.12	*53.12	32.33	*54.75	36.51
LAI	*54.51	32.42	*54.64	22.44	*53.59	38.67
SV	*54.48	31.02	*52.89	29.16	*54.08	34.37
RSP	50.72	19.14	50.04	26.22	51.52	24.41
RPS	48.31	22.43	48.87	28.75	49.99	26.69
RAS	49.51	18.24	48.73	25.15	51.10	30.21
Maximum intensity (dB)						
LSP	•61.52	24.33	*62.76	22.01	*62.55	28.47
LAI	*62.69	19.21	*63.92	22.34	*65.39	26.71
SV	*64.73	23.11	*65.45	27.34	*64.90	32.76
RSP	68.91	23.52	*64.69	21.78	*66.68	26.65
RPS	67.78	20.42	*67.83	26.35	69.73	29.85
RAS	*64.45	22.72	69.93	19.11	68.14	21.46
10 range forSMR						
LSP	*8.89	21.04	*9.64	23.50	*7.80	26.51
LAI	*8.18	19.33	*9.28	16.87	*11.80	28.36
SV	•10.25	18.62	* 12.56	20.19	* 10.82	22.11
RSP	18.19	22.86	* 14.65	16.73	*15.16	19.72
RPS	19.47	23.51	* 18.96	22.98	19.74	17.11
RAS	* 14.94	19.52	19.77	18.62	17.98	23.06

Subjects with left (left superior paravermal, left anteroinferior), superior vermis and right superior paravermal lesions show reduced I0 range for /pʌ/, /tʌ/ and /kʌ/ (Table 37 and Figure 25 to Figure 33).

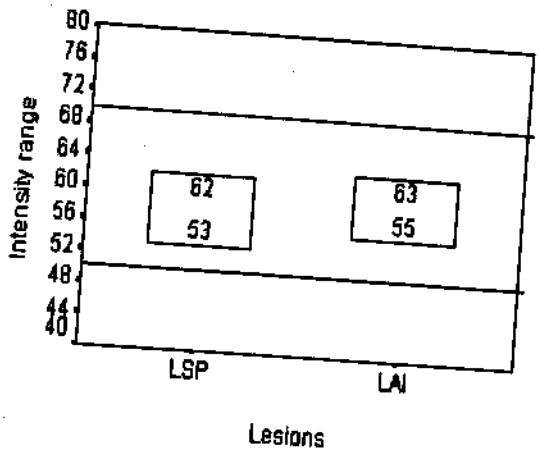

Figure 25: Intensity range for syllable /pa/ in SMR: Left superior paravermal, left anteroinferior and normals

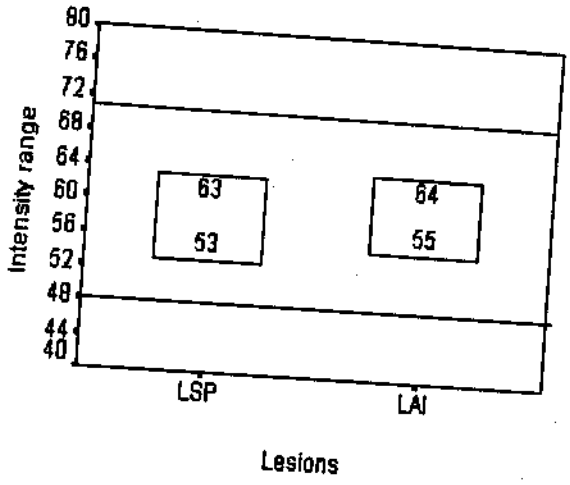

Figure 26: Intensity range for syllable /ta/ in SMR: Left superior paravermal, left anteroinferior and normals

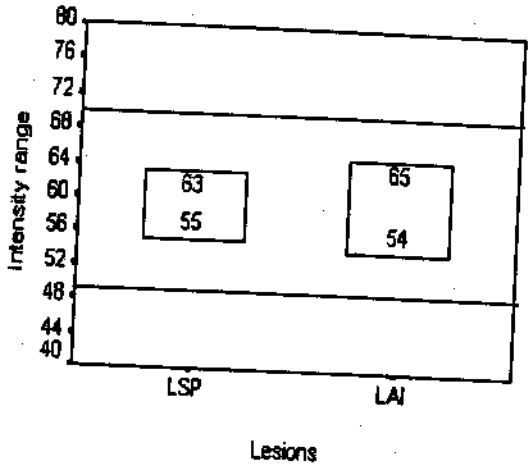

Figure 27: Intensity range for syllable /ka/ in SMR: Left superior paravermal, left anteroinferior and normals

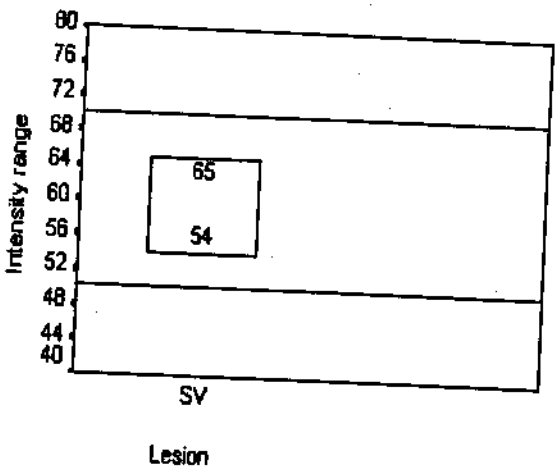

Figure 28: Intensity range for syllable /pa/ in SMR: Superior vermis and normal controls

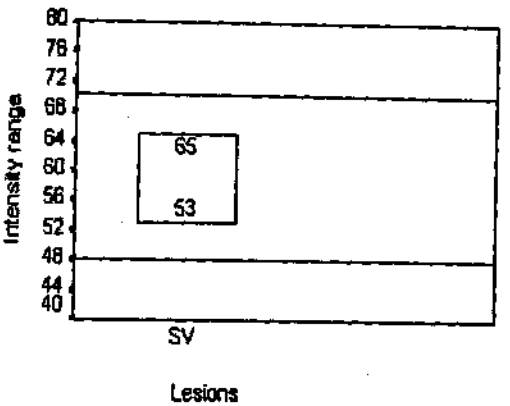

Figure 29: Intensity range for syllable /ta/ in SMR: Superior vermis and normals

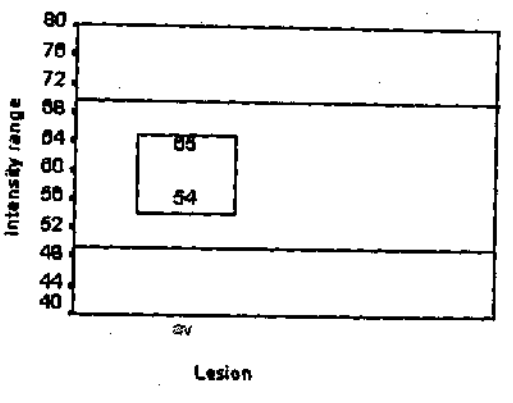

Figure 30: Intensity range for /ta/ in SMR: Superior vermis and normal controls

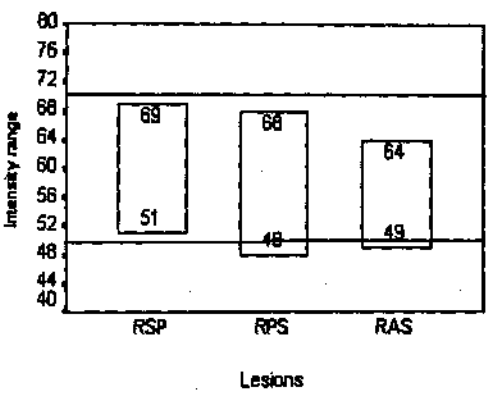

Figure 31: Intensity range for syllable /pa/ in SMR: Right superior paravermal, right posterisuperior, right anterosuperior and normals

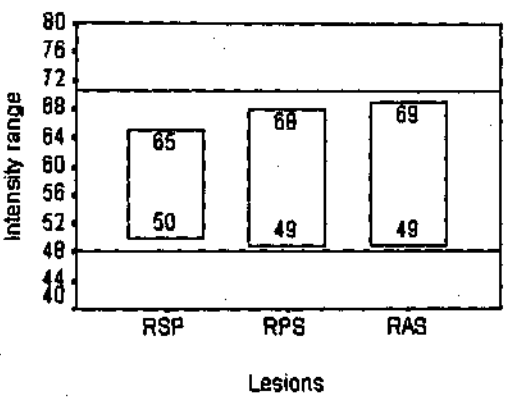

Figure 32: Intensity range for /ta/ in SMR: Right superior paravermal, right anterosuperior and normal controls

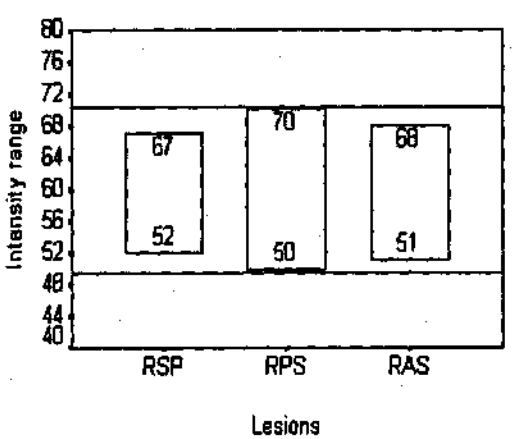

Figure 33: Intensity range for syllable /ka/ in SMR: Right superior paravermal, right anterosuperior and normals

Reduced 10 range in subjects with left superior paravermal, left anteroinferior and superior vermis lesions is due to increase in minimum intensity and decrease in maximum intensity for all the three syllables. Reduced 10 range for syllable /pa/ in subjects with left superior paravermal lesions is due to decrease in maximum intensity alone. The reduced intensity range in these groups may be indicative of respiratory insufficiency. 10 range for syllable /pA/ in subjects with right superior paravermal lesion, for syllables /pV and /*kA/* in subjects with right posterosuperior lesion, for /tA/ and /k/\/ in subjects with right anterosuperior lesion is comparable to normal controls (Table 37). The reduction in 10 range for syllable /pA/ in subject with right antero superior lesion, for syllable /t*A/* and /*kA/* in subjects with right superior paravermal and for syllable /W in right posterosuperior lesion is due to reduction in energy maxima. It may be inferred that right posterosuperior and right anterosuperior regions of the cerebellum are not involved in controlling aspects of intensity related to fast repetition of syllable sequence as in SMR task and that respiratory insufficiency may be a factor in

Summary

Total duration of syllable is comparable to normal controls in subjects with left (left superior paravermal, left anteroinferior), superior vermis and right posterosuperior lesions, for AMR task. It is increased compared to normal controls in subjects with right superior paravermal and right anterosuperior lesions. Increased syllable duration as well as increased closure duration contributed to increase in total duration in these experimental groups. No lesion specific findings are observed for SMR task, as total duration is increased in all the experimental groups. Akin to the findings obtained for AMR, increase in total duration is due to increase in syllable duration as well as closure duration.

In both AMR and SMR tasks, syllable duration contributes more to total duration than closure duration. Thus it may be presumed that irrespective of task complexity (AMR or SMR), time required for execution of the syllable ('sd') per se is more than the time prior to actual execution or preparatory phase of the syllable ('cd'). AMR rate is comparable to normal control subjects in some experimental subjects (left superior paravermal, left anteroinferior, superior vermis, right posterosuperior), whereas it is reduced in subjects with right superior paravermal and right anterosuperior lesions. SMR rate is reduced in all the experimental groups.

The findings that emerged for total duration for AMR and SMR task indicates that some speech task specific (AMR and SMR) findings can be found in subjects with lesions in different cerebellar loci. The findings indicate that simpler tasks like AMR may have less regions of representation (right superior

paravermal, right anterosuperior) in the cerebellum compared to neural correlates underlying complex task like SMR which seem to be represented in more regions of the cerebellum (left superior paravermal, left anteroinferior, superior vermis, right superior paravermal, right posterosuperior, right anterosuperior). The findings obtained for AMR task are similar to the 'Differential cue lateralization hypothesis' by van Lancker and Sidtis (1992) and study by Boutsen and Christman (2002). According to these studies spectral aspects of speech are lateralized to right cerebrum and temporal aspects of speech are lateralized to left cerebrum. Due to contralateral cerebro-cerebellar connections, spectral aspects are controlled by the left cerebellum and temporal aspects are controlled by the right cerebellum, respectively. Increased total duration is seen only for lesions associated with right cerebellar regions (right superior paravermal and right anterosuperior) for AMR task. It may be presumed that right posterosuperior region is involved in functional aspects related to AMR. The findings obtained for SMR cannot be explained on the premise of the 'Differential cue lateralization hypothesis' and study by Boutsen and Christman (2002). Durational aspects related to SMR task seem to be represented throughout the cerebellum as total duration is increased in all the experimental groups.

Intensity range is reduced in subjects with left (left superior paravermal, left anteroinferior), superior vermis, right superior paravermal and right anterosuperior lesions compared to normal controls in AMR task. It is comparable to normal controls in subjects with right posterosuperior lesions. Reduced intensity range seems to be because of increase in energy minima and decrease in energy maxima or due to decrease in energy maxima only. In the SMR task

intensity range is reduced in the left (left superior paravermal, left anteroinferior), superior vermis and right superior paravermal lesions compared to normals. The right posterosuperior and right anterosuperior lesions are not implicated in aspects related to intensity control in a task like SMR. The reduced intensity range in the SMR task in subjects with left superior paravermal, left anteroinferior and superior vermis lesions is due to decrease in energy maxima and increase in energy minima. Reduced intensity range for SMR task in subjects with right superior paravermal lesions is due to decrease in energy maxima only. In the AMR and SMR task, intensity related parameters (spectral aspect) seem to be lateralized not only to left cerebellar regions (left superior paravermal, left anteroinferior) but also to superior vermis and right superior paravermal regions. The results obtained is partly comparable to increased variation in amplitude (vAm %) in subjects with left superior paravermal, left anteroinferior, superior vermis and right posterosuperior lesions for sample of phonation. It is noticeable that right posterosuperior lesions are not involved in aspects of intensity related to DDK tasks as also observed in the phonatory task (Category I).

It is evident that some task specific findings emerge with respect to temporal and spectral (intensity related) aspects of DDK task (AMR and SMR). Differential localization for aspects related to temporal control of DDK is evident only for AMR task and not for SMR task. Differential localization can also be observed with respect to aspects related to intensity control in DDK tasks, for both AMR and SMR tasks.

C. Duration of short and long vowels

This task involved the production of a minimal pair /kAlAm/ and */kA:lAm/*, varying in the length of the vowel (short vowel /A/ and long vowel /A:/) in the initial syllable. Each of these words were embedded in a carrier phrase and produced with normal rate and prosody, five times each. The sentences were produced in a random order. The duration of the vowels (short and long) were measured. Prolonged vowel duration is a well reported characteristic feature in ataxic dysarthria due to nonfocal lesions (Kent et al., 1979; Ackermann and Hertrich, 1994; Ackermann et al., 1999). None of the earlier studies have analyzed vowel duration patterns in subjects with lesions restricted to the cerebellum. Hence this task was involved in the study. The parameters with respect to short vowel are discussed in section l(a) and those related to long vowel are discussed in section 1 (b).

1. (a). Duration of short vowel

The Mean and SD for duration of short vowel for normal and experimental subjects and the confidence intervals for normal controls are given in Table 38.

Table 38: Mean and SDfor duration of short vowel for normal control subjects (N) and experimental subjects and confidence intervals for normal controls. [(*) indicate values outside confidence interval]

	Mean (ms)	SD	Confidence interval
N	97.32	15.81	92.76 to 104.08
LSP	*111.78	18.69	-
LAI	* 119.83	21.45	-
SV	* 125.00	15.61	-
RSP	*131.76	25.73	-
RPS	* 123.52	19.76	-
RAS	* 134.94	13.89	-

Duration of short vowel is increased in all the experimental subjects compared to normal control subjects (Table 38). Slow speaking rate and prolonged speech segments are reported as salient features of ataxic dysarthria associated with diffuse or multifocal lesions (Kent et al., 1979; Ackermann and Hertrich, 1993). Also, Kent and Netsell, (1975); Hirose et al., (1978) and Ackermann and Hertrich (1993, 1994) stated that increased movement times with variable articulatory velocities, high variability of movement durations, and abnormal temporal patterns of muscular force development could be the reasons for the slow rate in ataxic dysarthria due to diffuse or multifocal lesions. Prolonged vowels in ataxic speech have often been noted in acoustic studies on cerebellar dysarthria due to multifocal lesions (Kent et al., 1979; Kent & Rosenbek, 1982; Gandour and Dardarananda, 1984; Ackermann and Hertrich, 1993; Ackermann et al., 1995). The results in Table 38 indicate that duration of short vowel is increased in subjects with lesions restricted to the cerebellum, also.

1 (b) Duration of long vowel

The Mean and SD for duration of long vowel for normal and experimental subjects and the confidence intervals for normal controls are given in Table 39.

Table 39: Mean and SD for duration of long vowel for normal control subjects (N) and experimental subjects and confidence ntervals for normal controls. [(*) indicate values outside confidence interval]

	Mean	SD	Confidence interval
N	212.83	13.87	204.66 to 221.89
LSP	*227.06	18.76	-
LAI	•243.34	14.55	-
SV	*236.84	23.78	-
RSP	•241.33	17.08	-
RPS	*246.67	21.83	-
RAS	*232.71	17.44	-

The duration of long vowel is increased in all the experimental groups (Table 39). Duration of tense vowels are reported to be longer in subjects with ataxic dysarthia due to diffuse or multifocal lesions (Kent et al., 1979; Kent & Rosenbek, 1982). Prolonged acoustic segments and vowel durations, as a result of slow speech have often been noted in acoustic studies on cerebellar dysarthria due to multifocal lesions (Kent et al., 1979; Ackermann and Hertrich, 1993). The results in Table 39 indicate that duration of the long vowel is increased in subjects with lesions restricted to the cerebellum also, irrespective of the site of lesion.

The results indicate that duration of short as well as long vowels are increased in all the experimental groups compared to normal controls. These results agree with the findings of increased duration of short and long vowels in subjects with nonfocal lesions. The results suggest that neural correlates underlying the control of vowel duration (for both short and long vowels) seem to be represented in the left (left superior paravermal, left anteroinferior), superior vermis and right cerebellar (right superior paravermal, right posterosuperior, right anterosuperior) regions of the cerebellum.

C. Prosodic measures

Slow speech and reduced speech rate is a characteristic feature associated with subjects with ataxic dysarthria (Kent et al., 1975; Ackermann and Hertrich, 1994; Kent et al., 1997; Kent et al., 2000). Speech rate in a reading and narration sample was calculated for normal control subjects and experimental groups.

1 (a) Reading task

Speech rate was calculated for passage reading. The total numbers of syllables in the passage were noted. The total duration of the speech sample was obtained by measuring the total time taken for reading the passage, from the waveform obtained. The duration from the beginning to the end of the sample was measured. Speech rate (syll / s) in reading was obtained by the formula:

$$\text{Speech rate (syll / s)} = \frac{\text{Total number of syllables in the passage}}{\text{Total time taken for the sample (s)}}$$

Table 40: *Speech rate for normal control group and experimental groups for reading and confidence intervals (CI) for normal control subjects. [(*) indicate values outside confidence interval]*

Group	Rate (syll / s)	CI
N	4.65	4.17 to 4.82
LSP	*3.98	-
LAI	*4.03	-
SV	*3.99	-
RSP	*3.87	-
RPS	4.40	-
RAS	*3.93	-

Speech rate in reading is reduced for all the groups, except for subjects with right posterosuperior lesion (Table 40). Speech rate in reading is comparable to normal controls for subjects with right posterosuperior lesions.

1 (b) Narration task

Speech rate was calculated from the initial 45 s sample of of narration task. This duration was selected as this was the minimum sample duration for which all the experimental subjects spoke without inserting a very long pause in speech. The whole narrative sample could not be included as there were longer pauses in between when the experimental subjects formulated sentences. The total number of syllables in the passage was noted. Speech rate (syll / s) for narrative sample was obtained by the formula:

$$\text{Speech rate (syll / s)} = \frac{\text{Total number of syllables in narration}}{\text{Total time taken for the narrative sample (s)}}$$

Table 41: *Speech rate for normal control group and experimental groups for narration and confidence intervals (CI) for normal control subjects. [(*) indicate values outside confidence interval]*

Group	Rate (syll / s)	CI
N	4.79	4.37 to 5.02
LSP	*4.02	
LAI	*4.26	-
SV	*4.15	-
RSP	*3.79	-
RAS	*4.20	-
RPS	4.83	-

Speech rate is reduced in subjects with left superior paravermal, left anteroinferior, superior vermis, right superior paravermal and right anterosuperior lesions, compared to normal control subjects (Table 41). Speech rate is comparable to normal control subjects in subjects with right posterosuperior lesions.

The results in Table 40 and Table 41 indicate that reduced speech rate is a characteristic feature in subjects with lesions in left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal and right anterosuperior) cerebellar regions for both reading and narration task. The results indicate that slow speech was not a characteristic feature of subjects with right posterosuperior lesions, as evident from reading and narration task.

IV. Perceptual analysis

Perceptual analysis was carried out for 38 dimensions used in the Mayoclinic study by Darley et al., (1975). Seventy five second sample of narration was used for the perceptual analysis. Three judges (post graduate students in speech - language pathology) independently rated the speech samples of the subjects in both the groups on each of the 38 dimensions using a 7 - point rating scale. All three judges were experienced in perceptual ratings of dysarthric speech in a clinical setting.

The scale is as follows:

1 = normal speech
2 = mild
3 = mild to moderate
4 = moderate
5 = moderate to severe
6 = severe
7 = Profound

The samples of normal and experimental subjects were randomized and rerecorded. Judges were kept blind to the purpose of the study. All three judges rated the 75 s speech samples of subjects for each of the 38 dimensions based on **a**

7-point rating scale. The various perceptual parameters are categorized under the major headings of vocal quality (laryngeal and velopharyngeal), articulation, respiration, prosody, intelligibility, bizarreness and severity. The parameters with the (*) mark indicates parameters that were rated as absent ($\propto$ = zero variance) by all the three judges. These parameters were not observed in the speech of subjects. The interjudge reliability measures for the rest of the parameters are given in parentheses. There were twenty one dimensions which were rated as deviant and these included: pitch breaks, excess loudness variation, loudness decay, alternating loudness, hoarse (wet voice), breathy voice (transient), strained strangled voice, voice stoppages, hypernasality, hyponasality, nasal emission, forced inspiration - expiration, audible inspiration, grunt, increased rate in segments, increased overall rate, reduced stress, variable rate, inappropriate silences, short rushes of speech and repeated phonemes.

Item by item reliability was obtained for all the dimensions rated in the perceptual task. Reliability was calculated for all three judges as a whole for each dimension. The three judges rated all the dimensions for a second time for ten samples of subjects after a period of .3 months. Item by item reliability measures were calculated for these ratings also. All the dimensions showed good interjudge reliability ($\propto$ > 0.7500) and intrajudge reliability ($\propto$ > 0.8376). Among the dimensions that were rated, atleast two out of three judges rated similarly for each dimension, for each of the subjects. Since the interjudge reliability scores [alpha coefficient ($\propto$)] is high for all the dimensions, this rating alone (the rating that was similar between two out three judges) is considered for graphical representation

Figure 34 to Figure 46. No deviations in dimensions were observed for normal control subjects.

1) Vocal quality - laryngeal

- Voice tremor (0.9280), harsh voice (0.7773), breathy voice (continuous) (0.8462), strained strangled voice (*), hoarse (wet voice) (*), breathy voice (transient) (*), voice stoppages (*)

2) Phonatory - prosodic features

- Pitch -pitch level (0.7774), monopitch (0.9808), pitch breaks (*)
- Loudness - monoloudness (0.8962), loudness (overall) (0.9808), excess loudness variation (*), Loudness decay (*), alternating loudness (*)

3) Vocal quality - velopharyngeal

- Hypernasality (*), hyponasality (*), nasal emission (*)

4) Articulation

- Distorted vowels (0.8903), prolonged phonemes (0.9656), imprecise consonants (0.9706), irregular articulatory breakdown (*), repeated phonemes (*)

5) Respiration

- Forced inspiration - expiration (*), audible inspiration (*), grunt (*)

6) **Prosody**

- *Stress* - Excess and equal stress (0.9484), reduced stress (*)
- *Rate* - General rate (0.8440), prolonged intervals (0.8855), increased rate in segments (*), increased overall rate (*), variable rate (*), short rushes of speech (*), inappropriate silences (*)
- *Phrasing* - short phrases (0.9209)

7) Intelligibility, bizarreness and severity

- Ratings of intelligibility (0.7765), bizarreness (0.8750) and severity (1.000) were also obtained from the samples.

The most deviant speech dimensions were rated as vocal quality, phonatory-prosodic dimensions, articulation and prosody. The deviant speech dimensions (a) for vocal quality included voice tremor, harsh voice and breathy voice, (b) for phonatory - prosodic dimensions included pitch level and monopitch, monoloudness and overall loudness, (c) for articulation included distorted vowels, prolonged phonemes, imprecise consonants and (d) for prosody included excess and equal stress, general rate, prolonged intervals and short phrases.

Ataxic dysarthria due to nonfocal cerebellar lesions are related to a disturbance in the neural mechanisms that underlie the coordination, temporal regulation, and automatic control of respiratory, phonatory and articulatory movements for speech (Duffy, 1995; Ziegler & Wessel, 1996; Kent et al., 1997; Kent et al., 1998; Kent & Kent, 2000; Kent et al,, 2000; Kent et al., 2001). The results obtained for lesions restricted to different cerebellar areas, indicate that deficits are noticeable in speech dimensions related to vocal quality (laryngeal), articulation and prosody. Deviant dimensions related to respiratory feature are not seen as a characteristic feature of dysarthria associated with focal cerebellar lesions. This finding is in contrast to reports of reduced respiratory support for speech, forced inspiratory and expiratory sighs and audible inspiration in subjects with nonfocal cerebellar lesions (Kluin et al., 1988; Chenery et al., 1990).

Velopharyngeal involvement is not a common feature in ataxic dysarthria associated with nonfocal lesions (Duffy, 1995). The results indicate that vocal dimensions related to velopharyngeal aspects is not a characteristic feature of subjects with lesions in the left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right posterosuperior and right anterosuperior) cerebellar lesions, also. Brown et al., (1970) and Duffy (1995) reflected that characteristics of ataxic dysarthria due to nonfocal lesions are more apparent in articulation and prosody. The findings in this study for subjects with lesions in left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right posterosuperior and right anterosuperior) cerebellar lesions indicate that in addition to articulatory and prosodic dimensions, phonatory aspects are also deviant.

1) Vocal quality (laryngeal)

The rating that is same between atleast two of the three judges for each dimension (harsh voice, breathy voice and voice tremor) is represented graphically in Figure 34, 35 and 36 respectively.

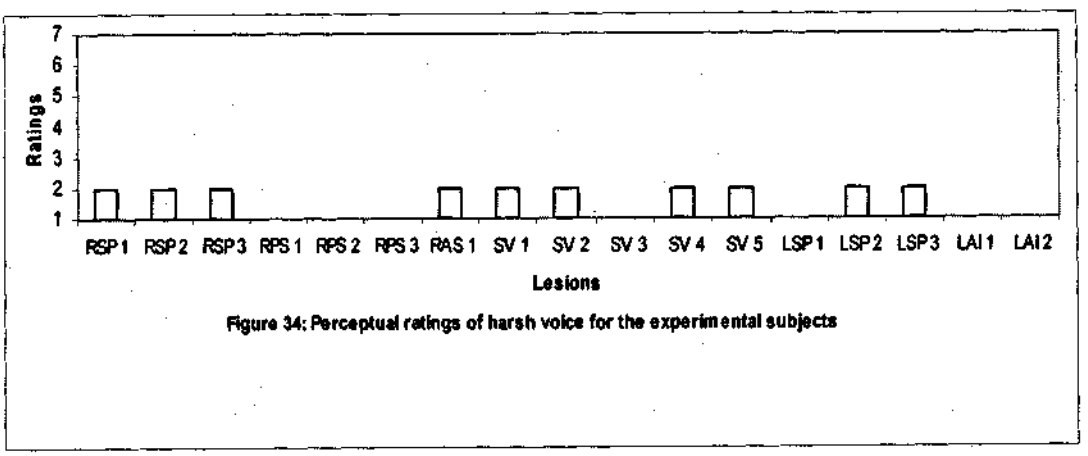

Figure 34: Perceptual ratings of harsh voice for the experimental subjects

been variously related to measures of vocal jitter (Wendahl, 1966; Takahashi & Koike, 1975; Eskenazi, Childers & Hicks, 1990), shimmer (Kitajima & Gould, 1976) and noise level (Lively & Emanuel, 1970; Wolfe, Cornell & Palmer, 1991; Yumoto, Sasaki & Okamura, 1984). Harshness in subjects with right superior paravermal, superior vermis and left superior paravermal lesion is associated with increased jitter, shimmer, NHR and SPI as seen in the Table 19, Table 22, Table 26 and Table 27 respectively in section B (spectral measures) on phonatory measures. Harshness is not associated with any of these acoustic correlates in subject with right anterosuperior lesion. Voice tremor is present only in subjects with superior vermis lesions (Figure 36). Jitter, shimmer and variation in F0 is also increased in subjects with superior vermis lesions. It is noticeable that parameters of breathy voice (continuous) and voice tremor are not characteristic features of ataxic dysarthria due to nonfocal lesions (Darley et al., 1975).

The results indicate that breathy voice is associated with subjects with superior vermis and left superior paravermal lesions alone and voice tremor is present only in subjects with superior vermis lesion. Increased breathiness in these groups is also associated with increased Jitter percentage, Shimmer percentage, NHR and SPI, as seen in Table 19, Table 22, Table 26 and Table 27 respectively in section B (spectral measures) on phonatory measures. Although the presence of harsh, breathy voice and voice tremor are usually attributed to phonatory dysfunction, it may also be due to dysfunction at other levels of speech production (respiratory system).

2) Phonatory - prosodic parameters

2 (a) Pitch

Pitch level: Variations in pitch level is absent in subjects with right cerebellar (right superior paravermal, right posterosuperior and right anterosuperior) and left cerebellar (left superior paravermal, left anteroinferior) lesions. It is mild in subjects with superior vermis lesions (Figure 37). Change in pitch level is not a characteristic feature in ataxic dysarthria due to nonfocal lesions (Darley et al., 1975). The results as in Figure 37 indicate that variations in pitch level are associated with superior vermis lesions alone. The results also agree with the finding of reduced FO in subjects with superior vermis lesions. FO (Hz) is comparable to normal control subjects in all other experimental groups.

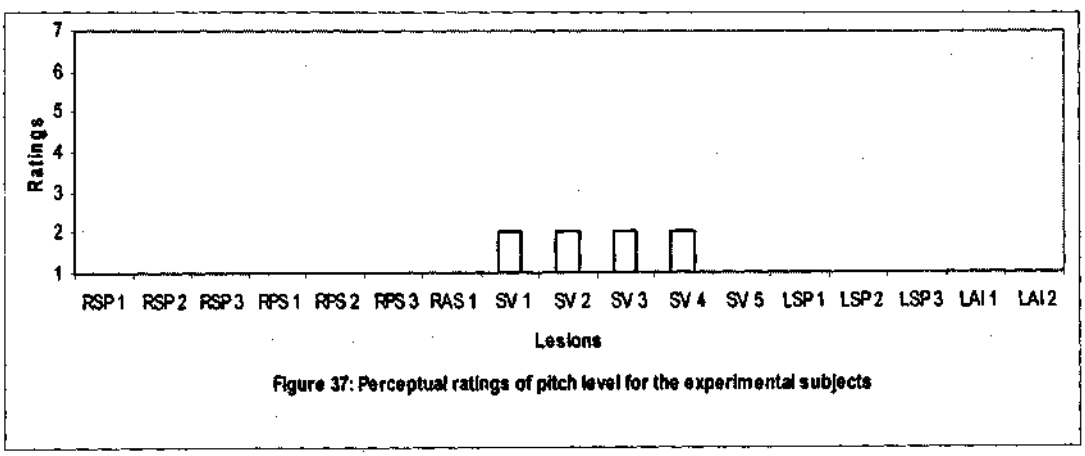

Figure 37: Perceptual ratings of pitch level for the experimental subjects

2 (b) Monopitch: Monopitch is a salient feature in ataxic dysarthria associated with nonfocal lesions (Darley et al., 1969 a, 1969 b; Brown et al., 1970; Chenery et al., 1990). However this feature is not predominant in ataxic dysarthria due to lesions restricted to different areas of the cerebellum, as it is seen only in single subject each with right superior paravermal, right anterosuperior, superior vermis and left superior paravermal lesions (Figure 38). It is absent in subjects with right posterosuperior and left anteroinferior lesions. Variation in fundamental frequency

(vFO) is associated with lesions in left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right posterosuperior, right anterosuperior) cerebellar lesions for task of sustained phonation. In conversational task, variation in FO is absent (i.e. monopitch) in individual subjects with lesion in right (right superior paravermal, right anterosuperior), superior vermis and left superior paravermal lesions.

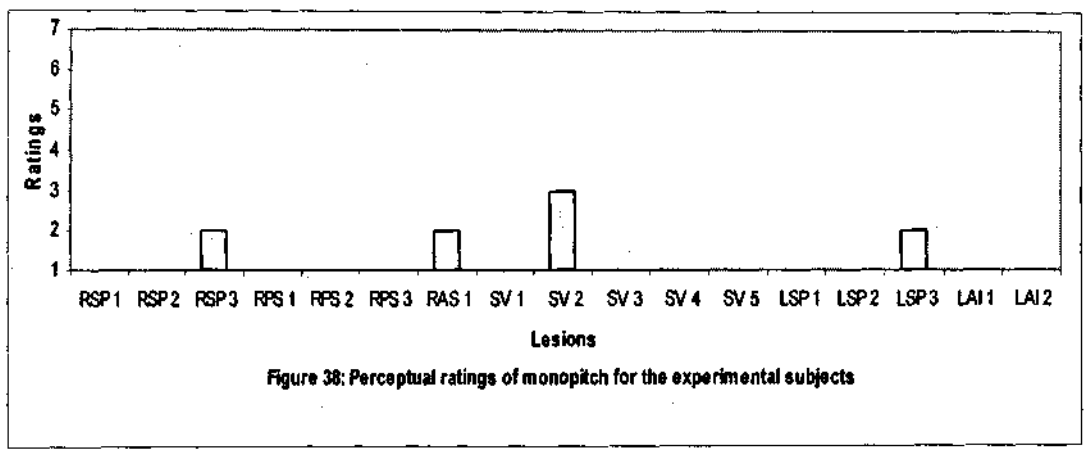

Figure 38: Perceptual ratings of monopitch for the experimental subjects

2 (c). Loudness

Monoloudness is a predominant dimension in ataxic dysarthria due to nonfocal lesions (Darley et al., 1969 a; 1969 b; Brown et al., 1970; Chenery et al., 1990). The presence of this dimension varied across experimental groups with different lesions restricted to the cerebellum. It is not a characteristic feature in subjects with right posterosuperior, right anterosuperior and left anteroinferior lesions. It is seen only in subjects with lesions in right superior paravermal, superior vermis and left superior paravermal regions in the cerebellum (Figure 39). Lesions in these cerebellar regions are associated with increased variation in amplitude (vAm %) in sustained phonation. The absence of variation in loudness in narrative sample in subjects with right superior paravermal, superior vermis and left superior paravermal regions in the cerebellum may be attributed to task based

differences. It may be that variations in loudness are more evident in a task like sustained phonation than in narration.

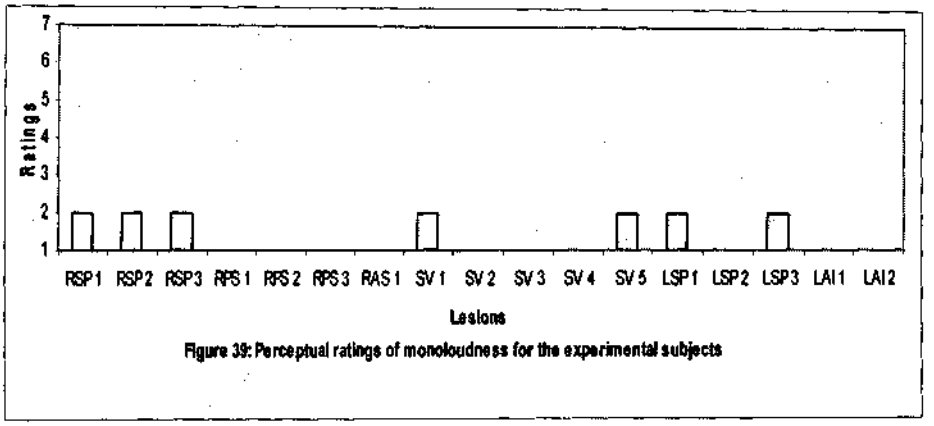

Figure 39: Perceptual ratings of monoloudness for the experimental subjects

According to Darley et al., (1975), change in overall loudness (i.e. voice that is excessively loud or soft) is a characteristic feature of ataxic dysarthria associated with nonfocal lesions. The results in Figure 40 indicate that a change in overall loudness is a characteristic feature only in subjects with lesions in superior vermis and left superior paravermal regions of the cerebellum.

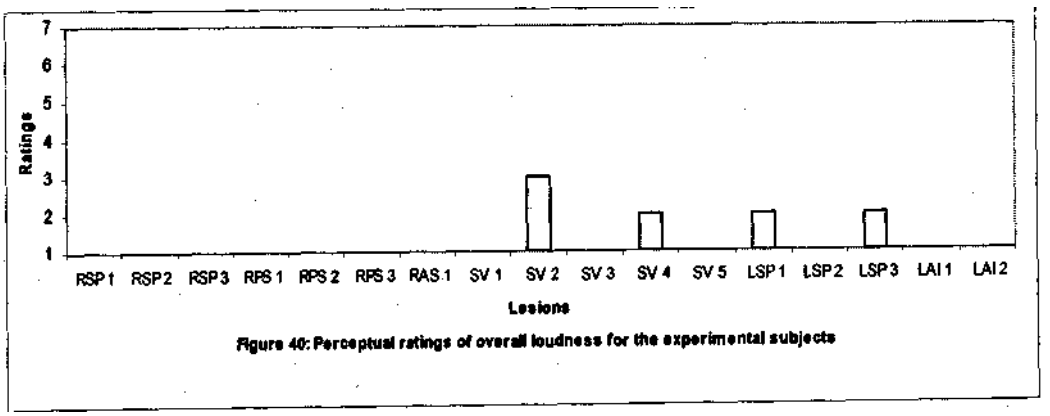

Figure 40: Perceptual ratings of overall loudness for the experimental subjects

3) Articulation

Distorted vowels are not rated as a predominant feature in any of the experimental groups. Distorted vowels as a feature of articulatory error, is seen only in single subject each with right superior paravermal, superior vermis and left superior paravermal lesions (Figure 41). Ackermann et al., (1992) rated irregular articulatory breakdown as a predominant feature in ataxic dysarthria associated with nonfocal lesions. Irregular articulatory breakdown is not a characteristic feature in any of the subjects with lesions in left superior paravermal, left anteroinferior, superior vermis, right superior paravermal, right posterosuperior and right anterosuperior lesions. These findings contradict the reported findings of presence of distorted vowels and irregular articulatory breakdown as the most frequently observed deviant features in ataxic dysarthria due to nonfocal lesions (Darley, et al., 1969). Prolonged phonemes are seen only in subjects with right superior paravermal, superior vermis and left superior paravermal lesions. Prolonged total duration of syllable is seen only in subjects with right superior paravermal and right anterosuperior lesions in the AMR task as seen in Table 29. Total duration of syllable is increased in all the experimental groups for SMR task (Table 32). Moreover the duration of short and long vowels is also increased in these groups (Table 38 and Table 39). The dimension of prolonged phonemes is

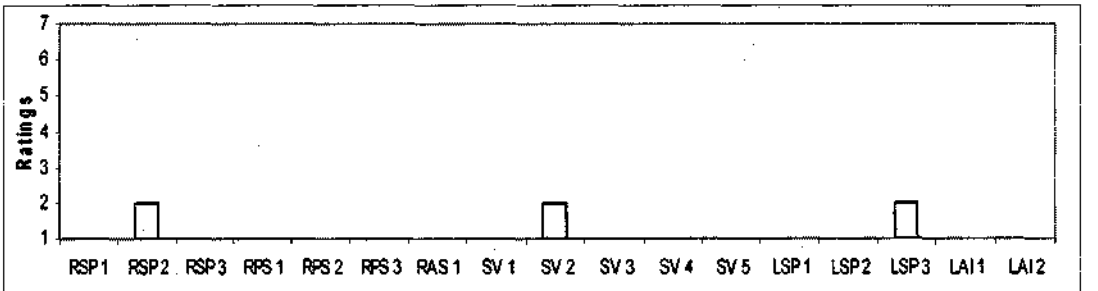

Figure 41: Perceptual ratings of distorted vowels for the experimental subjects

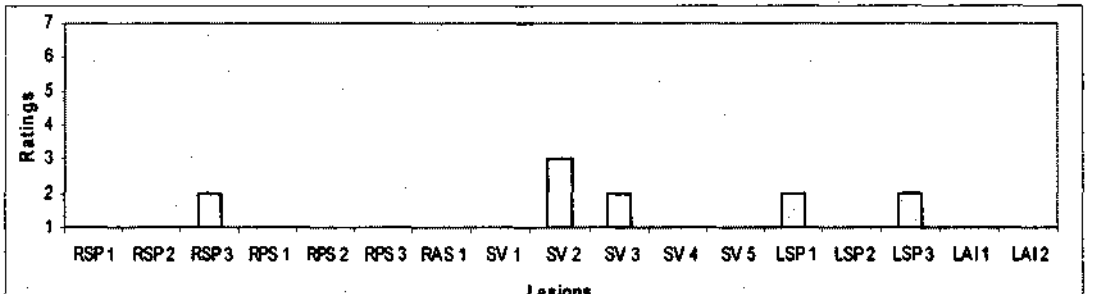

Figure 42: Perceptual ratings of prolonged phonemes for the experimental subjects

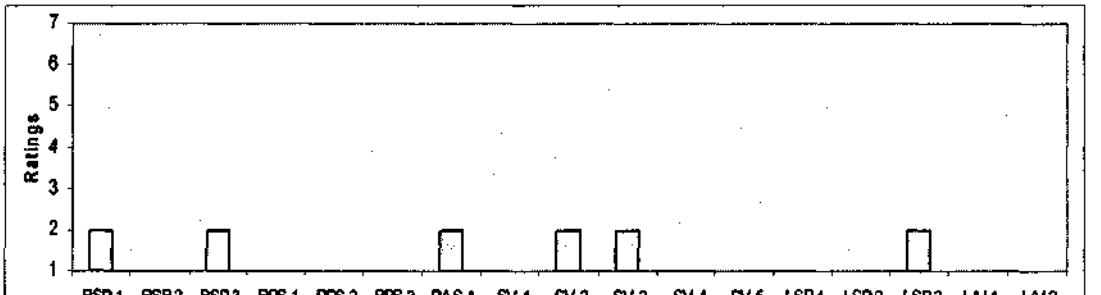

Figure 43: Perceptual ratings of imprecise consonants for the experimental subjects

4) Prosody

4 (a) Stress:

Excess and equal stress is not a characteristic feature in any of the subjects with lesion in right cerebellar (right superior paravermal, right posterosuperior, right anterosuperior) or in subjects with left anteroinferior lesions. It is seen only in one subject each with lesion in the superior vermis lesion and left superior paravermal region. This is in contrast to reports of predominance of excess and equal stress in the speech of ataxic dysarthric subjects with lesions in different cerebellar loci (Brown et al., 1970; Kluin et al., 1988).

4 (b) Rate:

Slow rate is a characteristic feature in ataxic dysarthria due to nonfocal lesions (Darley et al., 1969 a; 1969 b; Brown et al., 1970). Acoustic studies have also reported of slow rate and prolonged intervals in ataxic dysarthria, in the form of reduced velocities, reduced speech movement acceleration and inability to increase speech rate (Kent et al., 1979; Hirose, 1986; Ackermann et al., 1995; Kent et al., 1997). A different pattern emerged for lesions restricted to the cerebellum. Slow rate is a characteristic feature in subjects with lesions in the left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right anterosuperior) cerebellar lesions. Rate is normal in subjects with right posterosuperior lesion (Figure 44). These findings are agreeable with the finding of reduced speech rate in reading and narration in subjects with left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right posterosuperior and right anterosuperior) cerebellar regions as seen in Table 40 and Table 41. It is

noticeable that speech rate in narration is comparable to normal controls in subjects with right posterosuperior and left anteroinferior cerebellar lesions.

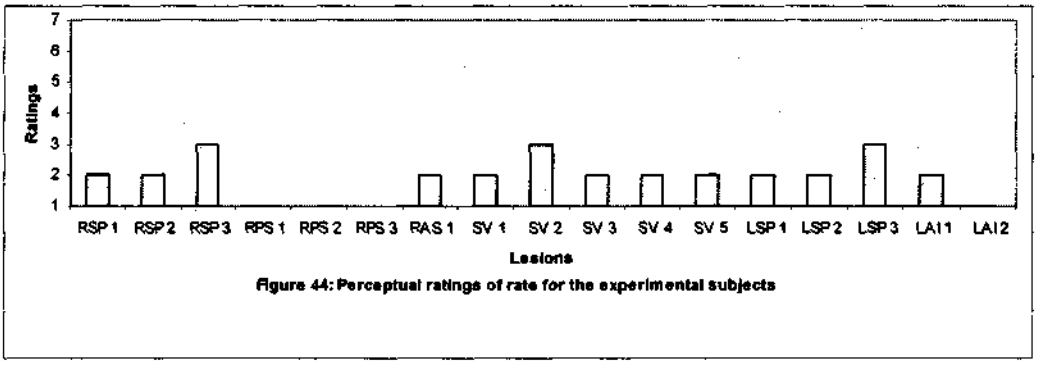

Figure 44: Perceptual ratings of rate for the experimental subjects

Prolongation of interword or intersyllable intervals is a characteristic feature in subjects with right superior paravermal, superior vermis and left superior paravermal lesions (Figure 45). It is not seen in subjects with right posterosuperior, right anterosuperior and left anteroinferior lesions. With respect to DDK task, the intersyllable durations (i.e. closure duration) is prolonged only in subjects with right superior paravermal and right anterosuperior lesions in AMR task as seen in Table 29. Closure duration is increased in all experimental groups for SMR task as seen in Table 32.

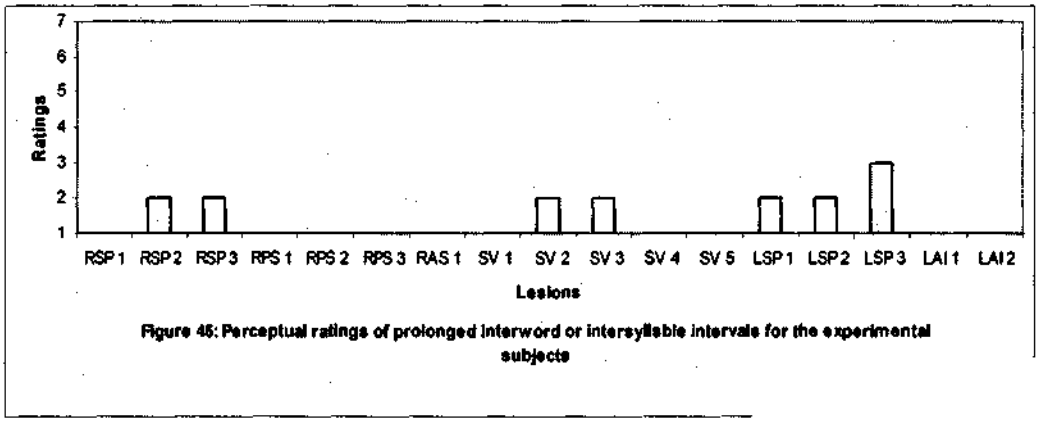

Figure 45: Perceptual ratings of prolonged interword or intersyllable intervals for the experimental subjects

4 (c) Phrasing:

Reduced length of phrases is described as a characteristic feature of ataxic dysarthria associated with nonfocal lesions (Darley et al., 1975). Reduced phrase length is seen only in subjects with lesions in right superior paravermal, right anterosuperior, superior vermis and left superior paravermal lesions (Figure 46). It is normal in subjects with right posterosuperior and left anteroinferior lesions. Respiratory abnormalities associated with speech have been reported in subjects with diffuse cerebellar lesions (Ludlow and Bassich, 1984; Murdoch et al., 1991). Some studies on ataxic dysarthria due to nonfocal cerebellar lesions report irregularities in chest wall movements during speech tasks (Murdoch et al., 1991) whereas other studies report hypotonia in the respiratory and laryngeal musculature (Brown et al., 1970; Darley et al., 1975; Chenery et al., 1990). Reduced phrase length in subjects with lesions in right superior paravermal, right anterosuperior, superior vermis and left superior paravermal lesions may be indicative of reduced respiratory support for speech in these groups. However physiological data is required to confirm this view.

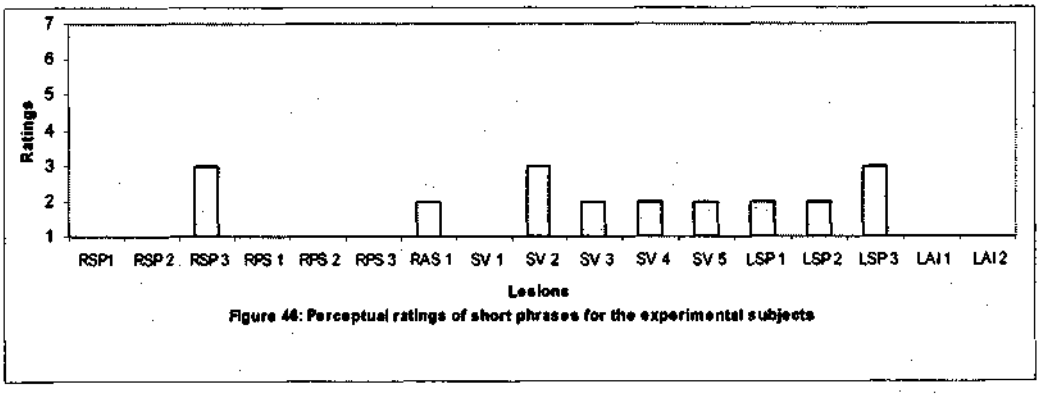

Figure 46: Perceptual ratings of short phrases for the experimental subjects

5. Intelligibility and bizareness

Intelligibility and bizarreness is mild in all subjects with left superior paravermal, superior vermis, right superior paravermal and right posterosuperior lesions. Mild to moderate intelligibility and bizarreness is seen in subject ST with left anteroinferior lesion and in subject with subject RN with right anterosuperior lesion.

6. Severity ofdysarthria

The ratings for severity were the same by all the three judges. Severity is mild in all the experimental subjects except subject ST (left anteroinferior lesion) and RN (right anterosuperior lesion). These subjects (ST and RN) are rated as mild to moderately severe. Murdoch and Theodoros (1998) reported that in slowly progressive lesions like cerebellar tumours, symptoms of cerebellar disease are less severe. The findings of the study agree with the finding of Murdoch and Theodoros (1998) as majority of the experimental subjects had mild severity.

Summary of perceived speech deviations in experimental subjects

Table 42: *Experimental groups in which the following perceptual dimensions of speech are absent*

Vocal quality			
	Right	Superior vermis	Left
Harsh voice	RPS	-	LAI
Breathy voice (continuous)	RSP, RPS,RAS	-	LAI
Voice tremor	RSP, RPS,RAS	-	LSP, LAI
Articulation			
Distorted vowels	RPS, RAS	-	LAI
Prolonged phonemes	RPS, RAS	-	LAI

Prosody			
Excess and equal stress	RSP, RPS,RAS	-	LAI
Rate	RPS	-	-
Prolonged intervals	RPS,RAS	-	LAI
Short phrases	RPS	-	LAI
Monoloudness	RPS, RAS	-	LAI
Overall loudness	RSP, RPS, RAS	-	LAI
Pitch level	RSP, RPS, RAS	-	LSP, LAI
Monopitch	RPS	-	LAI

It can be seen from the Table 42 that most of the deviant perceptual dimensions are absent in subjects with right cerebellar (especially right posterosuperior lesion) and left cerebellar (especially left anteroinferior lesion) lesion. These findings are not in agreement with the reports of Brown (1949, 1959), Amici et al., (1976) and Timmann et al., (1999), who emphasized the role of the cerebellar hemispheres in speech motor control.

However, it is to be noted that most of the studies implicating the role of right cerebellar hemisphere (Ackermann et al., 1992; Cruz-Martinez and Arpa, 1997; Urban et at., 2003) or left cerebellar hemispheric areas (Amici et al., 1976; Lechtenberg and Gilman, 1978; Amarenco et al., 1991) in speech motor control have not mentioned the importance of these regions in speech motor control. Majority of the perceptual dimensions are deviant in subjects with superior vermis lesions. The role of superior cerebellar vermis in speech motor control has been supported by several studies (Holmes, 1917; 1922; Chiu et al., 1996). The role of superior vermis in speech motor control is also contradicted by several studies (Brown (1949; 1959; Amici et al., 1976; Lechtenberg and Gilman, 1978; Ackermann and Ziegler, 1992; Timmann et al., 1999), who emphasized instead the role of the cerebellar hemispheres in speech motor control.

Differential localization is evident for the dimensions related to vocal quality. It is seen that harsh voice is seen in subjects with right superior paravermal, superior vermis and left superior paravermal lesions. Breathy voice is seen in subjects with superior vermis and left superior paravermal lesions. The finding of increased Jitter percentage, Shimmer percentage, NHR and SPI for the task of sustained phonation (Table 19, Table 22, Table 26 and Table 27 respectively) agrees with the findings of harshness and breathy voice in these groups. Voice tremor is seen only in subjects with superior vermis lesions also. Voice tremor associated with increased variation in fundamental frequency is reported in ataxic dysarthric subjects with diffuse lesions (Ackermann & Ziegler, 1994).

Neural correlates underlying dimensions related to articulation also revealed some differential localization in the cerebellum. Distorted vowels and prolonged phonemes are characteristic features of subjects with right superior paravermal, superior vermis and left superior paravermal regions of the cerebellum. In the task of diadochokinesis, prolonged total duration of syllable is seen only in subjects with lesion in right superior paravermal and right anterosuperior lesions in the AMR task (Table 29). In SMR task, total duration of syllable is prolonged in all the experimental groups (Table 32). Duration of short and long vowels is increased in all the experimental subjects (Table 38 and Table 39). Thus task specific findings emerge with respect to dimension of prolonged phonemes in a narration sample versus that in DDK task and also based on

duration of short and long vowels. Thus different patterns seem to emerge based on the speech task involved.

Prosodic dimensions are not equally affected in all the experimental groups. Excess and equal stress which is often quoted as a characteristic feature in ataxic dysarthria due to nonfocal lesions and is not a predominant feature in subjects with lesions in left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right posterosuperior, right anterosuperior) lesions. It is seen only in single subject with superior vermis and left superior paravermal lesion. Rate of speech is slow in subjects with left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal and right anterosuperior) lesions. It is comparable to normal controls in subjects with right posterosuperior lesion. This perceptual finding agrees with the findings of speech rate in narration and reading task (Table 40 and Table 41).

It is noticeable that subjects with right superior paravermal, superior vermis and left superior paravermal lesions show deviations from normal ratings for most of the parameters. According to Darley et al., (1975), there are ten perceptual dimensions that are predominant in subjects with ataxic dysarthria due to nonfocal lesions. These perceptual dimensions are imprecise consonants, excess and equal stress, irregular articulatory breakdown, distorted vowels, harsh voice, prolonged phonemes, prolonged intervals, monopitch, monoloudness and slow rate. Eight out of these ten dimensions are present in subjects with lesions in different cerebellar loci (imprecise consonants and irregular articulatory breakdown absent). The presence of these dimensions varied among different

lesion groups with left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right posterosuperior and right anterosuperior) cerebellar lesions.

These perceptual findings in ataxic dysarthria due to lesions in different cerebellar areas may have a physiologic basis, as studies on ataxic dysarthria have documented slow movements, errors of direction and range of movements, impaired muscular forces in production of rapid movements, and reduced or exaggerated range of movements in respiratory, phonatory and articulatory systems (Duffy, 1995; Kent et al., 2000). These abnormalities may be augmented by the motor symptoms characterizing cerebellar dysfunction, such as hypotonia, broad-based stance and gait, truncal instability; dysmetria, tremor and dysdiadochokinesis (Duffy, 1995; Kent et al., 2000).

Summary of acoustic and perceptual analysis of voice and speech of cerebellar subjects

Different protocols were used for investigation of the following subsystems of speech production mechanism:

A. Protocol for Phonatory tasks
B. Protocol for Articulatory tasks
C. Protocol for Prosodic tasks

Each protocol included tasks which were sensitive to investigate the function of the respective subsystem of speech.

The protocol for assessing phonatory subsystem included:

(a) Maximum phonation duration
(b) Maximum fricative duration and s/z ratio
(c) Voice onset time

The protocol for assessing articulatory mechanism included:

(a) Word repetition task

(b) Diadochokinetic task

The protocol for prosodic subsystem included:

(a) Speech rate in passage reading and narration

The results indicate that not all speech tasks are sensitive to support specific functional control of speech in different cerebellar areas. Maximum phonation duration is increased in all experimental groups compared to normals. Duration of fricatives /s/ and */zl* and s/z ratio is reduced for all experimental groups except in subjects with right posterosuperior and right anterosuperior lesion. Hence this task reveals some differential localization with respect to the mechanisms underlying the neural control of production of /s/ and */zl*. Aspects related to functional localization in the cerebellum also emerge from the results obtained for spectral aspects related to phonatory sample. Fundamental frequency is comparable to normal controls in subjects with left (left superior paravermal, left anteroinferior), and right (right superior paravermal, right posterosuperior, right anterosuperior) cerebellar lesions. FO is reduced only in subjects with superior vermis lesion. Phonatory frequency range and frequency perturbation measures (Jitter percentage, SPPQ %) are increased only in subjects with left (left superior paravermal, left anteroinferior), superior vermis and right superior paravermal lesions. However variation in fundamental frequency (vFO %) is increased in all the experimental groups. Amplitude perturbation parameters also revealed differential localization in the cerebellum for short term and long term measures of intensity. Shimmer percentage, SAPQ % and variation in amplitude (vAm %) are increased only in subjects with left superior paravermal, left

anteroinferior and superior vermis lesions. In addition, Shimmer percentage is increased in subjects with right superior paravermal lesions and vAm (%) is increased in subjects with right posterosuperior lesion. Noise to harmonic ratio is increased in all subjects except female subject with left anteroinferior lesion and subject with right anterosuperior lesion. Soft phonation index is increased only in subjects with left superior paravermal, superior vermis, right superior paravermal and right posterosuperior cerebellar lesions. Voice onset time is increased in all the experimental groups and no lesion specific findings emerged from the findings.

No salient findings can be seen in the results obtained for word repetition task. Results show differential localization within the cerebellum based on AMR and SMR tasks in diadochokinetic task. The total syllable duration is increased only in subjects with right superior paravermal and right anterosuperior lesions in AMR task. It is increased in all the experimental groups for SMR task. Intensity range in AMR task is reduced in subjects with left (left superior paravermal, left anteroinferior), superior vermis and right (right superior paravermal, right anterosuperior) cerebellar lesions and comparable to normal controls in subjects with right posterosuperior lesions. In the SMR task, intensity range is increased in all experimental groups except in subjects with right posterosuperior and right anterosuperior lesions probably indicating that these areas of the cerebellum are not involved in controlling aspects related to control of intensity. Duration of short and long vowels are increased in all experimental groups compared to normal controls.

Rate of speech is reduced in all the experimental groups for narration as well as reading task. Speech rate in subjects with right posterosuperior lesion is comparable to normal control subjects.

Perceptual analysis of narrative sample revealed functional localization of neural correlates underlying dimensions related to vocal quality (breathy voice, harsh voice, voice tremor), loudness (monoloudness, overall loudness), articulation (distorted vowels and prolonged phonemes), prosody (stress and rate of speech, prolonged intervals and phrasing).

The results indicate that tasks of maximum fricative duration, spectral dimensions of sustained phonation, durational aspects related to AMR task, intensity related measures in AMR and SMR task and rate of speech are salient tasks that reveal functional localization for speech in the cerebellum. Perceptual analysis is also a useful method to infer on functional localization in the cerebellum.

SUMMARY & CONCLUSIONS

Dysarthria is a motor speech disorder that results from neurological impairments associated with weakness, slowness, or incoordination of the musculature used to produce speech (Kent, 2000). In dysarthria, the subsystems of speech production mechanism, such as respiratory, phonatory, articulatory, resonatory and prosody are affected to a degree which is dependent on the type of dysarthria. Dysarthria caused due to damage to the cerebellar structure of the brain is called ataxic dysarthria.

Initially, ataxic dysarthria was considered as a homogenous disorder. But the contemporary view is that ataxic dysarthria is not a homogenous disorder. This observation is based on findings which suggest that the areas / loci of the cerebellum have a differential role in speech motor control (Duffy, 1995; Boutsen et al., 1997; Kent et al., 2000). Localisation of functional control in the cerebellum has been discussed with specific reference to the vermal / paravermal, hemispheric, anterior / posterior and superior / inferior regions of the cerebellum (Lechtenberg & Gilman, 1978; Amarenco et al., 1991; Duffy, 1995; Gerwig et al., 2003; Richter et al., 2005). Functional control of the cerebellum is also discussed in relation to its connection with the cerebral hemispheres (cerebro - cerebellar connection). There are many hypotheses proposed in this regard. In a study by Boutsen and Christman (2002), it was suggested that functional control of syllable duration, movement transition, interword intervals, voice onset time, consonantal and vocalic gesture (front - back) was controlled by left cerebral and consequently the right cerebellar areas (because of contralateral connections). They attributed the functional control of phrasal intonation, tonic stress and overall speech rate

that are message driven to the right cerebral (and left cerebellar hemispheres) and functional control of voice onset time and syllable duration to left cerebral (and right cerebellar hemisphere).

Functional localization in the cerebellum has also been looked upon based on the differential performance by ataxic subjects on speech tasks. The differences observed was explained as due to the different degrees of demands that the tasks placed on speech motor control (Kent et al., 2000). Few studies have reported on the task based performance differences in subjects with lesions restricted within the cerebellum (Lechtenberg & Gilman, 1978; Amarenco et al., 1991; Ackermann et al., 1992; Urban et al., 2003). These studies described the characteristics of dysarthria through perceptual measures of subjects with atrophy or nonfocal lesions in the cerebellum.

It is not known clearly whether functional localization based on different speech tasks are quite specific to focal areas of the cerebellum. In particular, it is not known if various parameters of voice and speech are controlled by different sites in the cerebellum. Observation and study of voice and speech dimensions in subjects with lesion in specific sites of the cerebellum, thus would contribute significantly in understanding the functional control of different cerebellar regions in speech motor control.

Increasing evidences are proposed to postulate that there are subgroups in ataxic dysarthria and that acoustic analysis is the most sensitive tool to identify the subgroups. There is a need to address the issue of differential contribution of

cerebellar regions in speech motor control keeping in mind the various facets of evidences proposed to support this notion. The present study probes into voice and speech manifestations in subjects with lesions in different cerebellar sites by measuring the various perceptual and acoustic dimensions in the speech of such subjects.

The study aimed:

- To analyse and differentiate some aspects of voice and speech in selected speech tasks in subjects with ataxic dysarthria due to lesions in various sites of the cerebellum using acoustic and perceptual analysis.
- To compare the results obtained in individuals with ataxic dysarthria against that of normal control group.

It was hypothesized that there would be a difference in voice and speech characteristics of subjects with cerebellar lesions when compared to that of normal control subjects and it was also hypothesized that there would be differences between groups of subjects with lesions in different cerebellar loci [left superior paravermal (LSP), left anteroinferior (LAI), superior vermis (SV), right superior paravermal (RSP), right posterosuperior (RPS) and right anterosuperior (RAS)].

Method:

The study attempted to investigate the voice and speech dimensions in Malayalam speaking ataxic dysarthric subjects with lesions in different cerebellar loci. Only subjects with specified variety of lesions (tumour) were included so as

to maintain homogeneity within the cerebellar groups. Stringent inclusion and exclusion criterias specified in the study restricted the number of subjects and there were limited number of subjects with lesions in different cerebellar loci. The experimental group included seventeen subjects with ataxic dysarthria. This group included subjects with lesions in left superior paravermal (LSP), left anteroinferior (LAI), superior vermis (SV), right superior paravermal (RSP), right posterosuperior (RPS) and right anterosuperior (RAS) regions of the cerebellum. The control group included thirty subjects matched for age and sex of the experimental subjects. A cross sectional standard group comparison research design was used for the study. Both the experimental and control subjects were asked to perform various voice and speech tasks of a test protocol. The speech tasks in this protocol facilitated testing of the function of the following mechanism of speech system: A) Phonatory B) Articulatory and C) Prosody.

Material and recording

A task protocol was compiled to study the dysfunction in the domain of A) Phonation, B) Articulation and C) Prosody. The protocol included tasks which were sensitive to investigate the function of the respective subsystem of speech. There were three tasks under phonatory, three tasks under articulatory and one task under prosodic domain. The subjects in the experimental and control group were instructed to perform the various tasks in the selected protocol. The subjects were tested in individual setup.

Analysis

The voice and speech samples of the subjects were subjected to acoustic and perceptual analysis. Acoustic analysis for the phonatory samples were carried out using Multi Dimensional Voice Profile (MDVP) Program in Computerized Speech Lab 4400 (CSL - 4400), Kay Elemetrics software. Other speech parameters were analysed using spectrogram, pitch contour and energy contour modules in Computerized Speech Lab 4400 (CSL - 4400), Kay Elemetrics software. Perceptual analysis was carried out for 38 dimensions of speech based on the method proposed by Darley et al., (1975). The speech samples were rated on a seven point rating scale by three judges. Inter and intra judge reliability were computed.

The data were compiled and discussed under spectral, temporal and perceptual measures for the various tasks. The data obtained from the samples were tabulated and compared between normals and experimental groups. Within experimental groups, comparisons were made only between right superior paravermal and left superior paravermal lesions and between right posterosuperior and right anterosuperior lesions, as they were corresponding groups.

The data was subjected to appropriate statistical analysis using Statistical Package for Social Sciences (SPSS -10).

The tasks and the measures elicited are depicted in Table 43:

Table 43: *Brief overview of the tasks involved in the study and the measures obtained from these tasks*

	Task	Dimensions measured		
		Spectral	Temporal	Perceptual
1. Pholatory tasks	Maximum phonation duration (MPD) for vowels /a/, *l\l* and *IvJ*	• Fundamental frequency • Frequency perturbation • Amplitude perturbation • Noise related	MPD (s)	-
	Maximum fricative duration for *Isl* and *111*		Maximum fricative duration and s/z ratio	-
	Voice onset time (VOT) in the stimuli words *lkA[/\ml* and /gAlAm/		• VOT for voiceless sound in the stimuli word /kAiAm/ (CVCVC) • VOT for voiced sound in the stimuli word /gA^m/ (CVCVC)	-
II. Articijlat**or**)*rtoisks*	Word repetition task	-	-	• Analysis of phoneme articulation using SODA, phonologica 1 process analysis
	Diadochokinetic task • /pA/, /W and /kV in AMR • /pAtAW in SMR	• Minimum intensity • Maximum intensity • Intensity range	• Syllable duration • Closure duration • Total duration • DDK rate	

	Duration of short and long vowels in the stimuli words /kAlArn/ and /kA:lArn/		• Duration of short vowel in the stimuli word /kAlAm/ (CVCVC) • Duration of long vowel in the stimuli word /kA.-lAm/ (CVVCVC)	
III. Prosodic tasks	• Passage reading • Narration task		Speech rate (syll/s)	

The salient results are as follows:

/. *Phonatory tasks:*

1) Temporal measures

- Maximum phonation duration for vowels was reduced in all the experimental groups compared to normal controls.
- Maximum fricative duration of /s/ and *IzJ* were reduced in all the experimental groups, with the exception of subjects with right posterosuperior (RPS) and right anterosuperior (RAS). The s/z ratio was increased in all the experimental groups when compared to normal controls, except in subjects with right posterosuperior and right anterosuperior lesions (RAS).
- Duration of voice onset time (VOT) for voiceless / voiced sounds were increased in all the experimental groups compared to normal subjects. VOT for voiced sound was comparable to normal subjects for subjects with right posterosuperior lesions (RPS).

2) Spectral measures

- Fundamental frequency (FO) measure was comparable to normal group in subjects with left (left superior paravermal, left anteroinferior) and right (right superior paravermal, right posterosuperior, right anterosuperior) cerebellar groups for all three vowels. FO was reduced in subjects with superior vermis lesion.
- Phonatory frequency range (PFR) in subjects with left [left superior paravermal (LSP), left anteroinferior (LAI)], superior vermis (SV) and right superior paravermal (RSP) lesions were increased compared to normal control subjects. PFR was comparable to normal subjects in subjects with right posterosuperior (RPS) and right anterosuperior lesions (RAS).
- Jitter percentage (Jitt) (%) and Smoothed amplitude perturbation quotient (SPPQ) (%) were increased for the left [left superior paravermal (LSP), left anteroinferior (LAI)], superior vermis (SV) and subjects with right superior paravermal (RSP) lesion compared to normal control group. Jitter percentage was comparable to normal group in subjects with right posterosuperior (RPS) and right anterosuperior lesions (RAS).
- Variation in fundamental frequency (vFO %) measures was increased in all experimental groups compared to normal group
- Shimmer percentage (Shim) (%) was increased in subjects with left [left superior paravermal (LSP), left anteroinferior (LAI)], superior vermis (SV) and subjects with right superior paravermal (RSP) lesion compared to normal control subjects. It was comparable to normal

controls in subjects with right posterosuperior lesions (RPS) and subject with right anterosuperior lesion (RAS).

- SAPQ (%) was increased in subjects with left and superior vermis group. SAPQ (%) was comparable to normal control subjects in subjects with right superior paravermal lesion (RSP) and subjects with right posterosuperior (RPS) and right antero superior lesions (RAS).
- vAm (%) was increased in subjects with left superior paravermal (LSP), left anteroinferbr (LAI), superior vermis (SV) and right posterosuperior (RPS) lesions compared to normal control subjects. vAm (%) was increased for vowels /i/ and /u/ in subjects with right posterosuperior (RPS) and for vowel *I'll* in subjects with right anterosuperior lesion (RAS). vAm (%) was increased only for vowel /a/ in subjects with right superior paravermal (RSP) lesions.
- NHR was increased in all the groups except for female subject with left anteroinferior lesion (LAI) and for subject with right anterosuperior lesion (RAS), compared to normal control subjects
- SPI was increased in subjects with left superior paravermal lesion (LSP), superior vermis (SV) and right cerebellar [right superior paravermal (RSP) & right posterosuperior (RPS)] lesions. SPI for vowels /a/ and *III* were comparable to normal control subjects for female subject with left anteroinferior lesion (LAI) and subject with right anterosuperior lesion (RAS).

II. Articulatory measures

A. Perceptual measures

1. Word repetition task:

- Very few vowel and consonantal errors were observed in few subjects only of the experimental groups. Errors were mostly seen for the consonants /r/, /R/, /i/ and /dz/. No lesion specific trend could be observed based on the findings

B. Temporal measures

1. Diadochokinetic task

AMR for /pA/, /tA/ and /kA/:

- Total duration of the syllable ('td') was comparable to normal controls in all the experimental groups, except for subjects with right superior paravermal (RSP) and right anterosuperior lesions (RAS). These two groups showed increased ('td') for syllables /pA/, /tA/ and /KA/
- Syllable duration ('sd') was more than closure duration in subjects with left superior paravermal (LSP), left anteroinferior (LAI) and superior vermis (SV) lesions, for syllables /p/\/, /tA/ and /k/\/.
- Increased syllable duration ('sd') as well as increased closure duration ('cd') contributed equally for increased total duration ('td') of syllables in subjects with right anterosuperior (RAS) lesion, for all three syllables. In subjects with right superior paravermal (RSP) and right posterosuperior (RPS) lesions, it was observed that 'sd' contributed more to 'td' than 'cd', for syllable */pA/*.

- AMR rate was reduced for all three syllables in subjects with right superior paravermal (RSP) as well as subject with right anterosuperior lesion (RAS), whereas the measures from all other groups were comparable to normal control group.

SMR for /pAtAkA/:

- 'td' was increased in all the experimental groups compared to normal control subjects which was associated with increased 'sd' as well as increased 'cd'.
- SMR rate was reduced in all the experimental groups compared to normal controls.

***2. Duration of** short **and** long vowels*

- Duration of short and long vowel were prolonged in all the experimental groups

***C.** Spectral measures*

a) Minimum and maximum intensity for AMR

- Subjects with left [left superior paravermal (LSP), left anteroinferior (LAI)], superior vermis (SV) and right cerebellar [right superior paravermal (RSP), right anterosuperior (RAS)J lesions showed reduced intensity range compared to normal control subjects for syllables /p/\/, /tA/ and /kA/
- Intensity range was comparable to normal control subjects for */pA/* , /tA/ and /KA/ in subjects with right posterosuperior (RPS) lesions. Intensity

range in subject with right anterosuperior lesion was comparable to normal control subjects for syllable /tA/.

- Reduced intensity range in subjects with left [left superior paravermal (LSP), left anteroinferior (LAI)], superior vermis (SV) and right cerebellar [right superior paravermal (RSP), right anterosuperior (RAS)] lesions were due an increase in minimum intensity and decrease in maximum intensity or because of reduction in maximum intensity alone.

b) Minimum and maximum intensity for SMR

- Subjects with left [left superior paravermal (LSP), left anteroinferior (LAI)], superior vermis (SV) and right superior paravermal (RSP) lesions showed reduced 10 range for /p/\/, /tA/ and /KA/
- 10 range for syllable /pA/ in subjects with right superior paravermal lesions was comparable to normal control subjects. Intensity range for syllables /p/\/ and /KA/ in subjects with right posterosuperior (RPS) lesions and for syllables /tA/ and /KA/ in subject with right anterosuperior (RAS) lesion were comparable to normal control subjects.
- Reduced intensity range in the experimental groups was due to increase in minimum intensity and decrease in maximum intensity or due to reduction in maximum intensity alone.

HI,Prosodic measures

1) Speech rate in reading

- Speech rate in reading was reduced in all the experimental groups, except in subjects with right posterosuperior lesion (RPS), when compared to normal control subjects

2) Speech rate in narration

- Speech rate was reduced in subjects with left superior paravermal (LSP), left anteroinferior (LAI), superior vermis (SV), right superior paravermal (RSP) and right anterosuperior (RAS) lesions compared to normal control subjects except in subjects with right posterosuperior (RPS) lesions.

IV. Perceptual measures

1. Vocal quality (laryngeal)

- Features of harsh voice, breathy voice and voice tremor were absent in subjects with left anteroinferior (LAI) and right posterosuperior (RPS) lesions.
- Voice tremor was present only in subjects with superior vermis (SV) lesions

2. Phonatory - prosodic parameters

- Pitch level was rated as normal for the right cerebellar [right superior paravermal (RSP), right posterosuperior (RPS), right anterosuperior

(RAS)] as well as left cerebellar [left superior paravermal (LSP), left

anteroinferior (LAI)] lesions. It was rated as mild for subjects with superior vermis (SV) lesions.

- Monopitch was not predominant in most of the experimental groups. It was rated as mild in single subjects each with left superior paravermal (LSP), superior vermis (SV), right superior paravermal (RSP) and right anterosuperior (RAS) lesions.
- Monoloudness was absent in subjects with left anteroinferior (LAI), right posterosuperior (RPS) and right anterosuperior (RAS) lesions.
- Overall loudness was affected only in subjects with superior vermis (SV) and left superior paravermal (LSP) lesions.

3. Articulation

- Distorted vowels were not a predominant feature in any of the experimental groups.
- 'Prolonged phonemes' was present to a mild degree in subjects with right superior paravermal (RSP), superior vermis (SV) and left superior paravermal (LSP) lesions.
- Imprecise consonants were not a characteristic feature in subjects with right posterosuperior (RPS) and left anteroinferior (LAI) lesions. This feature was rated as mild in all the other experimental groups.
- Irregular articulatory breakdown was not a characteristic feature in any of the experimental groups

4. Prosody

- Excess and equal stress was not a characteristic feature in any of the subjects with left superior paravermal (LSP), left anteroinferior (LAI), right superior paravermal (RSP), right posterosuperior (RPS) and right anterosuperior (RAS) lesions. It was rated as mild in two subjects with SV lesions and one subject with left superior paravermal lesion.
- Slow rate was not a characteristic feature of subjects with right posterosuperior (RPS) lesion. All the other experimental groups were rated as mild on this parameter.
- Prolonged intervals was seen as a characteristic feature only in subjects with right superior paravermal (RSP), superior vermis (SV) and left superior paravermal (LSP) lesions.
- Short phrases were seen only in subjects with left superior paravermal (LSP), right superior paravermal (RSP) and right anterosuperior (RAS) lesions

Overall perceptual findings

- Majority of the abnormal perceptual features were absent in subjects with right posterosuperior (RPS) and left anteroinferior (LAI) lesions.
- Subjects with superior vermis (SV) lesions were rated as abnormal for all the perceptual parameters related to vocal quality, articulation and prosody.

Summary:

This study pointed to the possibility of a distinct localization for voice and speech measures within the cerebellum as revealed through acoustic and perceptual measures. The results obtained in the study suggest that the tasks of Maximum Phonation Duration, Word repetition, Voice Onset Time measures and short versus long vowel contrasts are not sensitive in identifying discrete neural correlates underlying localization of speech within the cerebellar areas. The performance of subjects with right posterosuperior (RPS) and right anterosuperior (RAS) lesions were comparable to normal control subjects for the task of maximum fricative duration and for short term measures of frequency and amplitude perturbation. The neural correlates related to FO seems to be localized in the superior vermis (SV) region of the cerebellum only. However, long term measures of frequency (vFO %) and amplitude (vAm %) were not sensitive enough to reveal any lesion specific information as vFO (%) and vAm (%) were found to be increased in all cerebellar lesions.

Some specific trend was observed for temporal measures related to articulation (AMR & SMR tasks). In the AMR task, total syllable duration was increased only in subjects with right posterosuperior (RPS) and right anterosuperior (RAS) lesions. Differential neural correlates underlying speech was not evident from the results of SMR task as all experimental groups showed increased total duration. It may be speculated that neural correlates for a complex task like SMR may be distributed throughout the cerebellum, whereas simpler task like AMR may have fewer neural correlates compared to SMR. Different findings emerged for spectral measures related to AMR and SMR tasks. Preliminary

findings indicate that neural correlates for control of intensity in AMR and SMR tasks are not localized in right posterosuperior (RPS) regions.

Some lesion specific patterns were evident from the perceived ratings of vocal quality, articulation and prosody. These parameters were not deviant in subjects with right posterosuperior (RPS) and left anteroinferior (LAI) lesions. This observation is well supported by the fact that subjects with right posterosuperior (RPS) lesions did not show deviation in most of the spectral and temporal parameters related to phonation and articulation. However, subjects with left anteroinferior (LAI) lesion presented some discrepancy between perceptual and acoustic findings. Subjects with superior vermis (SV) lesions showed disturbances in perceptual parameters of vocal quality, articulation and prosody.

There was a definite trend observed with the spectral and temporal measures related to phonation showing the involvement of left cerebellar [left superior paravermal (LSP) and left anteroinferior (LAI)] regions. This may be understood in the light of contralateral cerebro - cerebellar connections between the right cerebrum and left cerebellum and role of right cerebrum in spectral aspects. In addition, superior vermis (SV) was also implicated. It is noticeable that in addition to left superior paravermal (LAP), left anteroinferior (LAI) and superior vermis (SV) lesions, right superior paravermal areas of the cerebellum seemed to be involved in controlling spectral and temporal aspects of speech.

Temporal aspects related to articulation also seem to show some cerebro-cerebellar interaction. Increased total duration for AMR task was seen only in

subjects with right superior paravermal (RSP) and right anterosuperior (RAS) lesions. This may again be due to contralateral cerebro-cerebellar connections and role of left cerebral hemisphere in controlling aspects related to syllable duration. However, right posterosuperior (RPS) region of the cerebellum does not seem to be involved in controlling durational aspects related to AMR task. The findings for SMR do not draw support from contralateral cerebro-cerebellar findings as total duration was increased in all cerebellar regions. This may probably indicate that more complex tasks may have bilateral representation within the cerebellum. Additional evidence is required to support this presumption. Spectral aspects related to DDK (AMR and SMR) seem to show some trends based on possible cerebro - cerebellar interactions, as right cerebellar regions [right posterosuperior (RPS) and right anterosuperior (RAS)] were not implicated, whereas left cerebellar [left superior paravermal (LSP), left anteroinferior (LAI)] and superior vermis (SV) were affected.

The results of this study imply that localization of aspects of voice and speech dimensions within different cerebellar areas is highly likely, since specific patterns emerged for various voice and speech tasks. This study has made an attempt to study the voice and speech dimensions in dysarthric subjects with lesions in various cerebellar areas, using acoustic (temporal and spectral) and perceptual measures. The result of the study points to strong possibility of existence of subgroups in ataxic dysarthria. The findings stand in support of the notion that distinct forms of cerebellar dysarthria may be present, probably explaining inconsistencies in the speech characteristics of ataxic dysarthria. The study also shows that deviant findings are not evident for all tasks and measures,

implying that subjects with cerebellar dysarthria do not have a 'generic dysarthria'. The tasks which showed differential findings for different cerebellar lesions, would help us in characterizing possible subgroups and help in better classification of ataxic dysarthric features. The preliminary results of this study also point to the fact that localization may be a function of different speech tasks. But these conclusions are limited and are assumed in the light of small sample size of experimental subjects in the study.

Recommendations for future research:

The results have to be substantiated with: (a) more number of subjects with cerebellar lesions in specific areas distributed across the cerebellum (b) subjects with cerebellar tumours of different sizes (c) more voice and speech tasks. Collaborative evidences are required using physiological measures along with acoustic and perceptual measures.

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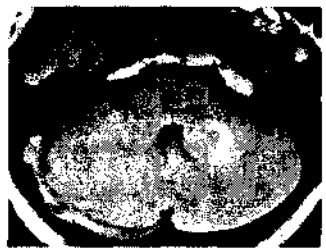
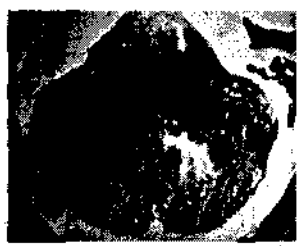
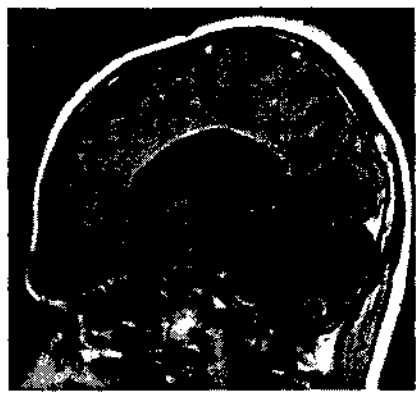
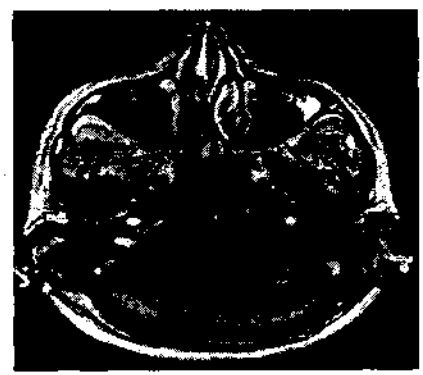
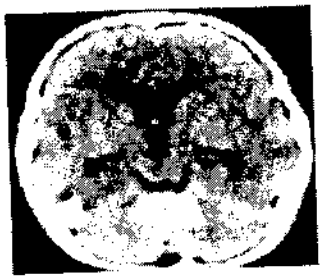
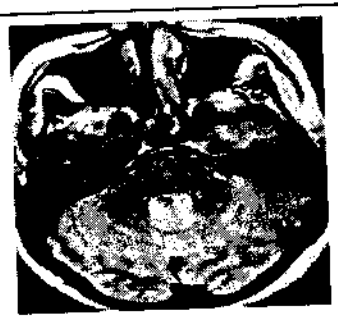
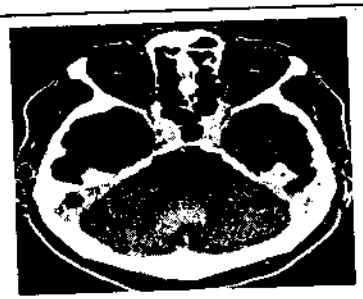
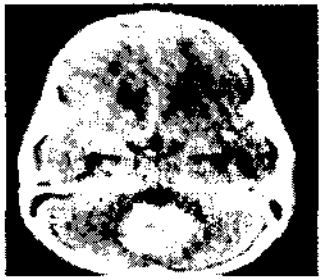
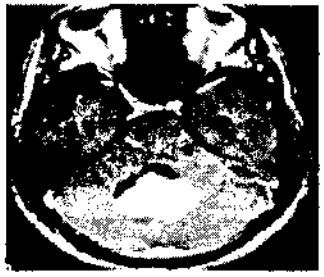
APPENDIX 1

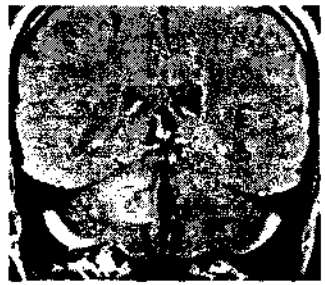
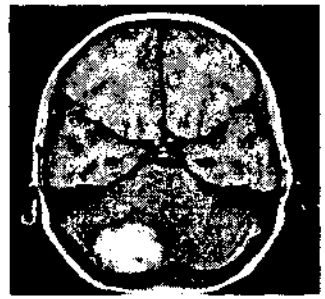
CT or MRI, Pathophysiology and diagnosis of the experimental subjects

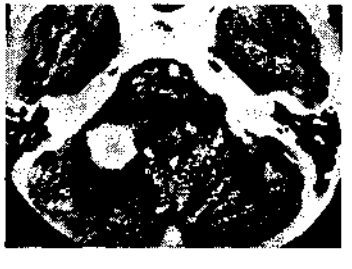
No.	Subject	CT or MRI	Pathophysiology	Diagnosis
1	OK		Cavernous angioma	MRI T1 weighted image with contrast showed well defined heterogeneous enhanced lesion in left superior paravermal region. Lesion size was 2* 2.2 cm.
2	NB		Medulloblastoma	CT scan with contrast shows hyperintense homogenously enhanced lesion in left superior paravermal region. The lesion size was 3*2.5 cm.
3	TJ		Venous angioma	CT scan with contrast shows enhancing vessels in left superior paravermal region. The lesion size was 3*2 cm.

No:	Subject	CT or MRI	Pathophysiology	Diagnosis
4	JA		Tuberculoma.	MRI T1 weighted image with contrast showed ring enhancing lesion in left lateral hemisphere (anteroinferior region). The lesion size was 1.4*1.8cm.
5	ST		Lymphoma.	MRI showed hyperintense lesion in the left lateral cerebellum (anteroinferior region) on T2 weighted images, which was isointense on T1 weighted images and showed intense enchancement with contrast. The lesion size was 1.1*1.2cm.

No.	Subject	CT or MRI	Pathophysiology	Diagnosis
6	MK		Medulloblastoma	CT scan showed isodense space occupying lesion with homogenous contrast enhancement in the superior cerebellar vermis. CT revealed compression of fourth ventricle and mild hydrocephalus. Lesion size was 3*2.5 cm
7	VN		Astrocytoma	MRI: T1 weighted image showed heterogenous contrast enhancing well defined non cystic mass in superior cerebellar vermis. Lesion size was 2*1.4 cm.
8	BT		Adenocarcinoma.	CT scan showed hyperdense mass in cerebellar vermis with homogenous and moderate enhancement with contrast. Lesion size was 2.2*2 cm.

No.	Subject	CT or MRI	Pathophysiology	Diagnosis
9	HR		Pilocytic astrocytoma	CT scan shows hypodense space occupying lesion with contrast enhancement and central necrosis in the cerebellar vermis. Lesion size was 3.1*3.4cm
10	SD		Hemangioblastoma	MRI axial view showing cystic lesion with enhancing nodule in the superior cerebellar vermis. Lesion size was 3.3*3.2 cm.
11	SP		Astrocytoma	MRI - T1 weighted image with contrast showed intensely enhanced lesion in the right superior paravermal region. Lesion size was 1.4*1 cm.

No.	Subject	CT or MRI	Pathophysiology	Diagnosis
12	ST		Angioma	MRI T1 weighted image with contrast shows heterogenous enhanced well defined lesion in right superior paravermal region. Lesion size is 2.2*2 cm.
13	OK2		Pilocytic astrocytoma	CT scan with contrast showed intense enhanced lesion in the right superior paravermal region. Lesion size was 2.5*2cm.
14	N		Hemangioblasto-ma.	MRI: T1 weighted image showed well defined hyperintense lesion with perilesional hypointense peripheral ring in right lateral (posterosuperior) cerebellar hemisphere. Lesion size was 4*3.5 cm.

No:	Subject	CT or MRI	Pathophysiology	Diagnosis
15	JT		Tuberculoma	CT scan with contrast showed ring enhanced lesion with peripheral edema in right lateral (posterosuperior) cerebellar hemisphere. Lesion size was 1.8*1.5 cm.
16	RP		Hemangio-blastoma	MRI T2 weighted image showed hyperintense lesion with surrounding edema in right lateral (posterosuperior) cerebellar hemisphere. Lesion is of low signal intensity in T1W MRI and showed intense enhancement with Gadolinium. Lesion size was 1.8*1.3 cm.
17	RN		Astrocytoma	CT scan with contrast showed homogenously enhanced, round, well defined lesion in right anterosuperior aspect of cerebellar hemisphere, with perilesional edema. Lesion size was 2.4*2.2 cm.

APPENDIX 2

Phonemic inventory ofMalayalam language

Vowels in Malayalam language (Syamala Kumari, 1972)

Vowel	Description
/A/	Short, low, back vowel
/A:/	Long, low, back vowel
/i/	Short, unrounded, high, front vowel
/i:/	Long, unrounded, high, front vowel
lul	Short, high, back, rounded vowel
/u:/	Long, high, back, rounded vowel
/e/	Short, unrounded, mid, front vowel
/e:/	Long, unrounded, mid, front vowel
lol	Short, mid, back, rounded vowel
lo:l	Long, mid, back, rounded vowel
/u/	Long short, high, back, unrounded vowel

Consonants in Malayalam language (Syamala Kumari, 1972)

	Bilabial	Labio dental	Dental	Alveolar	Retroflex	Palatal	Velar	Glottal
Stops	p , p \ b, b^h		t.d, th.dh				k,k" g,g^h	
Nasals	m		n	n	nm	n	ŋ	
Fricatives		v		s	s	l		h
Affricate						tf.ds		
Laterals				1	\			
Flaps				R				
Trill				r				

APPENDIX3

Word list in Malayalam

Vowels

Sl. No.		Target vowel	Initial	Medial	Final
1	M	അ	അലമാറി	കലം	വല
	T	/ʌ/	/alʌmʌ:ri/	/kʌləm/	/vʌlʌ/
	E	a	almirah	pot	net
2	M	ആ	ആമ	കാൽ	
	T	/ʌ:/	/ʌ:mʌ/	/kʌ:l/	
	E	aa	tortoise	leg	
3	M	ഇ	ഇല	കിണർ	മുടി
	T	/i/	/ilʌ/	/kiɳʌr/	/mudi/
	E	i	leaf	well	hair
6	M	ഈ	ഈച്ച	മീൻ	
	T	/i/	/i:ʧʌ/	/mi:ɳ/	
	E	ii	Food fly	fish	
7	M	ഉ	ഉറുമ്പ്	കുതിര	പുഴു
	T	/u/	/urumbe/	/kuTirʌ/	/puɻu/
	E	u	ant	horse	maggot
10	M	ഊ	ഊഞ്ഞാൽ	ചൂല്	
	T	/u:/	/u:ñʌ:l/	/cUlə/	
	E	uu	swing	broom	
11	M	ു			നാല്
	T	ʊ			/na:lə/
	E				four
14	M	എ	എരുമ	പെട്ടി	അവിടെ
	T	/e/	/erumʌ/	/peṯṯi/	/ʌviḍe/
	E	e	buffalo	box	there
15	M	ഏ	ഏണി	പേന	
	T	/e:/	/e:ɳl/	/pe:nʌ/	
	E	ee	ladder	pen	
18	M	ഒ	ഒട്ടകം	കൊടി	
	T	/o/	/oṯṯʌkʌm/	/koTi/	
	E	o	camel	flag	
19	M	ഓ	ഓല	കോഴി	
	T	/o:/	/o:lʌ/	/ko:ḻi/	
	E	oo	Palm leaf	hen	
20	M	ഐ	ഐരാവതം	കൈ	
	T	/ai/	/ʌirʌ:vʌtəm/	/kʌi/	
	E	ai	Airavatam	hand	

Note: M = Malayalam, T = Transliteration, E = Meaning in English

Consonants

Sl. No.		Target consonant	Initial	Medial	Final
1	M	ക	കസേര	ചകിരി	
	T	/k/	/kʌse:rʌ/	/ʧʌkiri/	
	E	k	chair	husk	
2	M	ഗ	ഗണപതി	ബാഗ്	
	T	/g/	/gʌɳʌpʌt/i	/bʌ:gə/	
	E	g	Lord Ganesha	bag	
3	M	ഖ		നഖം	
	T	/kʰ/		/nʌkʰam/	
	E	kh		nail	
4	M	ഘ	ഘടികാരം		
	T	/gʰ/	/gʰʌTikʌ:rʌm/		
	E	gh	Time piece		
5	M	ങ		മാങ്ങ	
	T	/ŋ/		mʌ:ŋʌ	
	E	ng		mango	
6	M	ച	ചെവി	സൂചി	
	T	/ʧ/	/ʧəvi/	/sUʧI/	
	E	ch	ear	needle	
7	M	ജ	ജനൽ	രാജാവ്	
	T	/ʤ/	/ʤənʌl/	/rʌ:ʤʌ:və/	
	E	j	window	king	
8	M	ഞ	ഞണ്ട	മഞ്ഞ	
	T	/ñə/	/ñʌɳɖə/	/mʌñʌ /	
	E	-	crab	mango	
9	M	ട		ആട്	
	T	/ʈ/		/ʌ: ʈə/	
	E	t		goat	
10	M	ഡ		ആഡംബരം	
	T	/ɖ/		/ʌ:ɖʌmbʌrəm/	
	E	d		decoration	
11	M	ണ		മണി	
	T	/ɳ/		/mʌɳI/	
	E	-		bell	
12	M	ത	തവള	വാതിൽ	
	T	/t/	/tʌvʌlʌ/	/vʌ:til/	
	E	tha	frog	door	
13	M	ഥ		രഥം	
	T	/tʰ/		/rʌtʰʌm/	
	E	th		chariot	
14	M	ധ	ധനം		
	T	/dʰ/	/dʰʌnʌm/		
	E	dh	money		

15	M	ദ	ദേവി	പാദസരം	
	T	/d/	/de:vi/	/pʌ:dʌsʌrʌm/	
	E	da	Goddess	anklet	
16	M	ന	നക്ഷത്രം		
	T	/n/	/nʌkśʌtrʌm/		
	E	-	star		
17	M	ഩ		ആന	
	T	/ń/		/ʌ:nʌ/	
	E	ñ		Elephant	
18	M	പ	പശു	അപായം	
	T	/p/	/pʌʃu/	/ʌpʌ:jəm/	
	E	p	cow	danger	
19	M	ഫ	ഫലിതം		
	T	/pʰ/	/pʰʌlitəm/		
	E	f	joke		
20	M	ബ	ബലം		
	T	/b/	/bʌləm/		
	E	b	strength		
21	M	ഭ	ഭരണി	ആഭരണം	
	T	/bʰ/	/bʰʌrʌɳi/	/ʌ: bʰʌrʌɳʌm/	
	E	bh	jar	ornament	
22	M	മ	മഷി	വിമാനം	
	T	/m/	/mʌši/	/vimʌ:nʌm/	
	E	m	ink	aeroplane	
23	M	യ	യന്ത്രം	കയർ	
	T	/j/	/jəndRəm/	/kʌjʌr/	
	E	y	engine	coir	
24	M	ര	രത്നം	മുന്തിരി	
	T	/r/	/rʌtnəm/	/mundiri/	
	E	-	diamond	grapes	
25	M	ല		അലമാറി	
	T	/l/	/lo:kəm/	/ʌlʌmʌ:ri/	
	E	l	world	almirah	
26	M	വ	വിളക്ക്	തവള	
	T	/v/	/vilʌkkə/	/tʌvʌlʌ/	
	E	v	lamp	frog	
27	M	ശ	ശംഖ്	പരമശിവൻ	
	T	/ʃ/	/ʃʌŋkə/	/pʌrʌmʌʃivʌn/	
	E	-	conch	Lord Shiva	
28	M	ഷ		മഷി	
	T	/š/		/mʌši/	
	E	sh		ink	
29	M	സ	സിംഹം	പാദസരം	
	T	/s/	/simhəm/	/pʌ:dʌsʌrʌm/	
	E	s	Lion	anklet	

30	M	ഹ	ഹൽവ	ഗുഹ	
	T	/h/	/hʌluvʌ/	/guhʌ/	
	E	h	Halva	Den	
31	M	ള		കാള	വാൾ
	T	/ɭ/		/kʌ:ɭʌ/	/vʌ:ɭ/
	E	-		Ox	sword
32	M	ഴ		മെഴുകുതിരി	
	T	/ɻ/		/məɻukutiri/	
	E	-		candle	
33	M	റ		താറാവ്	
	T	/r/	/rʌ: ɳl/	/tʌ:rʌ:və/	
	E	R	queen	duck	

Note: M = Malayalam, T = Transliteration, E = Meaning in English

Consonant clusters

Sl. No.		Target consonant	Initial	Medial	Final
1	M	ക്ഷ	ക്ഷീണം	നക്ഷത്രം	
	T	/kš/	/kši: ɳʌm/	/nʌkšʌtrʌm/	
	E	ksh	tired	star	
2	M	ത്ര		വസ്ത്രം	
	T	/tr/		/vʌstrəm/	
	E	tr		dress	
3	M	ഷ്ട		ഇഷ്ടിക	
	T	/št/		/i štikʌ/	
	E	sht		brick	
4	M	ക്ര	ക്രമം	ആക്രമണം	
	T	/kr/	/krʌməm/	/ʌ:kremʌɳʌm/	
	E	kr	order	attack	

Note: M = Malayalam, T = Transliteration, E = Meaning in English

APPENDIX 4

Reading passage in Malayalam

കേരളം

എല്ലാവർക്കും അവരവരുടെ നാടിനോടും ഭാഷയോടും പ്രത്യേക ഇഷ്ടമുണ്ടാവും. അതുപോലെത്തന്നെ എനിക്കും എന്റെ നാടും എന്റെ ഭാഷയും പ്രിയങ്കരമാണ്. ഇവയുടെ ഓർമ്മകൾ പോലും മധുരമുള്ളവയാണ്.അവ നല്കുന്ന സുഖം ഒന്ന് വേറെത്തന്നെയാണ്. ധാരാളം പുഴകളും അരുവികളും കായലുകളും ഗ്രാമങ്ങളും പട്ടണങ്ങളും കൊച്ചു നഗരങ്ങളും തെങ്ങിൻ തോപ്പുകളും വൻവനങ്ങളും നിറഞ്ഞ എന്റെ കൊച്ചു കേരളം സഞ്ചാരികൾക്ക് സ്വർഗ്ഗസമാനമാണ്. ദൈവത്തിന്റെ സ്വന്തം നാടാണ് കേരളം എന്ന ചൊല്ല് കേട്ടിരിക്കുമല്ലോ. ഒരു തരത്തിൽ പറഞ്ഞാൽ അതു വെറും ചൊല്ലല്ല കാര്യം തന്നെയാണ്. അത്രമാത്രം ഭംഗിയുണ്ട് എന്റെ കേരളത്തിന്. ആർക്കും കേരളത്തിന്റെ പ്രകൃതിഭംഗി അപ്പാടെ അവരുടെ വാക്കുകളിലൂടെ നിങ്ങൾക്ക് പറഞ്ഞുതരാനോ എഴുതിപ്പിടിപ്പിച്ചു കാണിക്കുവാനോ പറ്റുകയില്ല. അത് നേരിട്ട് നോക്കിക്കണ്ട് ഒരു സുന്ദരമായ അനുഭൂതിയായി നുണഞ്ഞിറക്കേണ്ടതാണ്.

സസ്യങ്ങളാൽ പച്ച നിറഞ്ഞ കേരളത്തെ മലനാട്, ഇടനാട്, സമതലം എന്ന് മൂന്നായി ഭാഗിക്കാം. പർവ്വതപ്രദേശമായ മലനാട്ടിൽ റബ്ബർ, തേയില, കാപ്പി, ഏലം എന്നിവ കൃഷി ചെയ്യുന്നു. ഇടനാട്ടിൽ ഇഞ്ചി, മഞ്ഞൾ, കുരുമുളക്, മരച്ചീനി മുതലായവയും സമതലത്തിൽ തെങ്ങും നെല്ലുമാണ് പ്രധാന വിളകൾ. ഭാരതത്തിന്റെ പടിഞ്ഞാറൻ തീരത്ത് ആദ്യമായി പാശ്ചാത്യനാടുമായി ബന്ധം സ്ഥാപിച്ചത് ഈ നാട്ടുകാരാണെന്ന് പറയപ്പെടുന്നു. തെങ്ങ് കേരളത്തിൽ എല്ലായിടത്തും കാണാവുന്നതാണ്. കേരളീയർ തെങ്ങിൻ തടി വീട് വെക്കാനും, തേങ്ങ ആഹാരത്തിനും, വെളിച്ചെണ്ണ തലയിൽ തേക്കാനും, ഓല വീട് മേയാനും ഉപയോഗിക്കുന്നു. അവർക്ക് തെങ്ങ് ഒരു കല്പവൃക്ഷം തന്നെയാണ്. കേരളീയരുടെ പ്രധാന തൊഴിൽ കൃഷിയാണ്.ഇപ്പോൾ കേരളത്തിൽ ഓരോ കൊല്ലം ചെല്ലുന്തോറും കൃഷിഭൂമിയും കൃഷിയും കുറഞ്ഞ് കുറഞ്ഞ് വരികയാണ്. ഇന്ന് അത്യാവശ്യ സാധനങ്ങൾക്കെ പോലും കേരളത്തിന് മറ്റു സംസ്ഥാനങ്ങളെ ആശ്രയിക്കേണ്ടി വരുന്നു. നാളത്തെ സ്ഥിതി എന്താകുമെന്ന് പറയാനാവില്ല. ജലസമൃദ്ധിയുടെ നാടെന്ന് പറയപ്പെട്ടിരുന്ന കേരളത്തിന് ഇന്നി കുടിവെള്ളം പോലും പൈസ കൊടുത്ത് വാങ്ങേണ്ടി വരുന്ന സ്ഥിതിയാവില്ലേ എന്ന് സംശയമില്ലാതില്ല.

Transliteration

Reading passage in Malayalam

ʞe:ɾʌlʌm

ellʌ:vʌɽkkum ʌvʌɾʌvʌɾuɽe nʌ:ʈɪno:ɖum bʰʌ:śʌjo:ʈum pɽətje:kʌ iʂʈʌmunɖʌ:vum. ʌtupo:lettʌɳɳə əɳikkum əɳʈə nʌ:ʈum əɳʈə bʰʌ:śʌjum priyʌŋkʌɾʌmʌ:ɳə. ivʌjuʈə o:rmʌkʌl̩ po:lum mʌdʰuɾʌmul̩lʌvʌjʌ:ɳə. ʌvʌ nʌlkunnʌ sukʰəm onnə ve:ɽəttʌnnəjʌ:ɳə. dʰʌ:ɾʌ:l̩ʌm puzhʌkʌl̩um ʌruvikʌl̩um kʌ:jʌlukʌl̩um gɾʌ:mʌŋʌl̩um pʌʈʈʌɳʌŋʌl̩um koʈʃʈʃu nʌgʌɾʌŋʌl̩um təŋiɳ to:ppukʌl̩um niɽʌɲɲʌ ənʈə i: koʈʃʈʃu ke:ɾʌl̩ʌm sʌɳcʌ:rikʌl̩kkə svʌrggʌsʌmʌ:ɳʌmʌ:ɳə. dəivʌttiɳʈə svəndəm nʌ:ʈʌ:ɳə ke:ɾʌl̩ʌm ənnʌ collə ke:ʈʈirikkumʌllo:. oru tʌrʌttil pʌɽʌɲɲʌ:l ʌtu vəɽum ʧollʌllʌ kʌ:ɾjəm tʌnnəjʌ:ɳə. ʌtɽamʌ:tɽʌm bʰʌŋijuɲʈə ənʈə ke:ɾʌl̩ʌttiɳə. ʌ:ɽkkum ke:ɾʌl̩ʌttiɳʈə prʌkɽitibʰʌŋi ʌppʌ:ʈə ʌvʌɾuʈə vʌ:kkukʌl̩ilu:ʈə niŋʌl̩kkə pʌɽʌɲɲu tʌɾʌ:ɳo: ezhutippiʈippiʈʃʈʃu kʌ:ɳikkuvʌ:ɳo: pʌttukʌjillʌ. ʌtə ne:ɾiʈʈə no:kkikkʌnʈə oru sundʌɾʌmʌ:jʌ ʌnubʰu:tijʌ:ji nuɳʌɲiɽʌkke:ɳʈʌtʌ:ɳə. əŋkil mʌ:tɽʌme: ke:ɾʌl̩ʌttiɳʈə muzhvʌnʌ:jul̩lʌ ʧitɽəm niŋʌl̩kkə kiʈʈukʌjul̩lu:.

sʌsjəŋʌl̩ʌ:l pʌʧʃʌ niɽʌɲʌ ke:ɾʌl̩ʌttə mʌlʌnʌ:ʈə iʈʌnʌ:ʈə sʌmʌtʌlʌm ənnə mu:nnʌ:ji bʰʌ:gikkʌ:m. pʌɽvvʌtʌpɽʌde:ʃʌmʌ:jʌ mʌlʌnʌ:ʈʈil ɽʌbbəɽ te:jilʌ kʌ:ppi e:lʌm ənnivʌ kɽiśi ʧəjjunnu. iʈʌnʌ:ʈʈil iɳʧl mʌɲʌl̩ kurumul̩ʌkə mutʌlʌ:jʌvʌjum sʌmʌtʌlʌttil təŋum nəlluмʌ:ɳə prʌdʰʌ:ɳʌvilʌkʌl̩. bʰʌ:ɾʌtʌttiɳʈə pʌʈiɲʌ:ɽəɳ ti:ɾʌttə ʌ:djʌmʌ:jl̩ pʌ:ʃʧʌ:tjʌ nʌ:ʈumʌ:jl̩ bʌɳdəm stʰʌ:piʧʃʌtə i: nʌ:ʈʈukʌ:ɾʌ:ɳənnə pʌɽʌjʌppəʈuɳɳu. təŋə ke:ɾʌlʌttil əllʌ:jiʈʌttum kʌ:ɳʌ:vunnʌtʌ:ɳə. ke:ɾʌl̩i:jəɽ təŋintʌʈi vi:ʈə vəkkʌ:ɳum te:ŋʌ ʌ:hʌ:ɾʌttiɳum vəl̩iʧʃʧəɳɳʌ tʌlʌjil te:kkʌ:ɳum o:lʌ vi:ʈə me:jʌ:ɳum upʌjo:gikkunnu. ʌvʌɽkkə təŋə oru kʌlpʌvɽəkśəm tʌnnəja:ɳə.

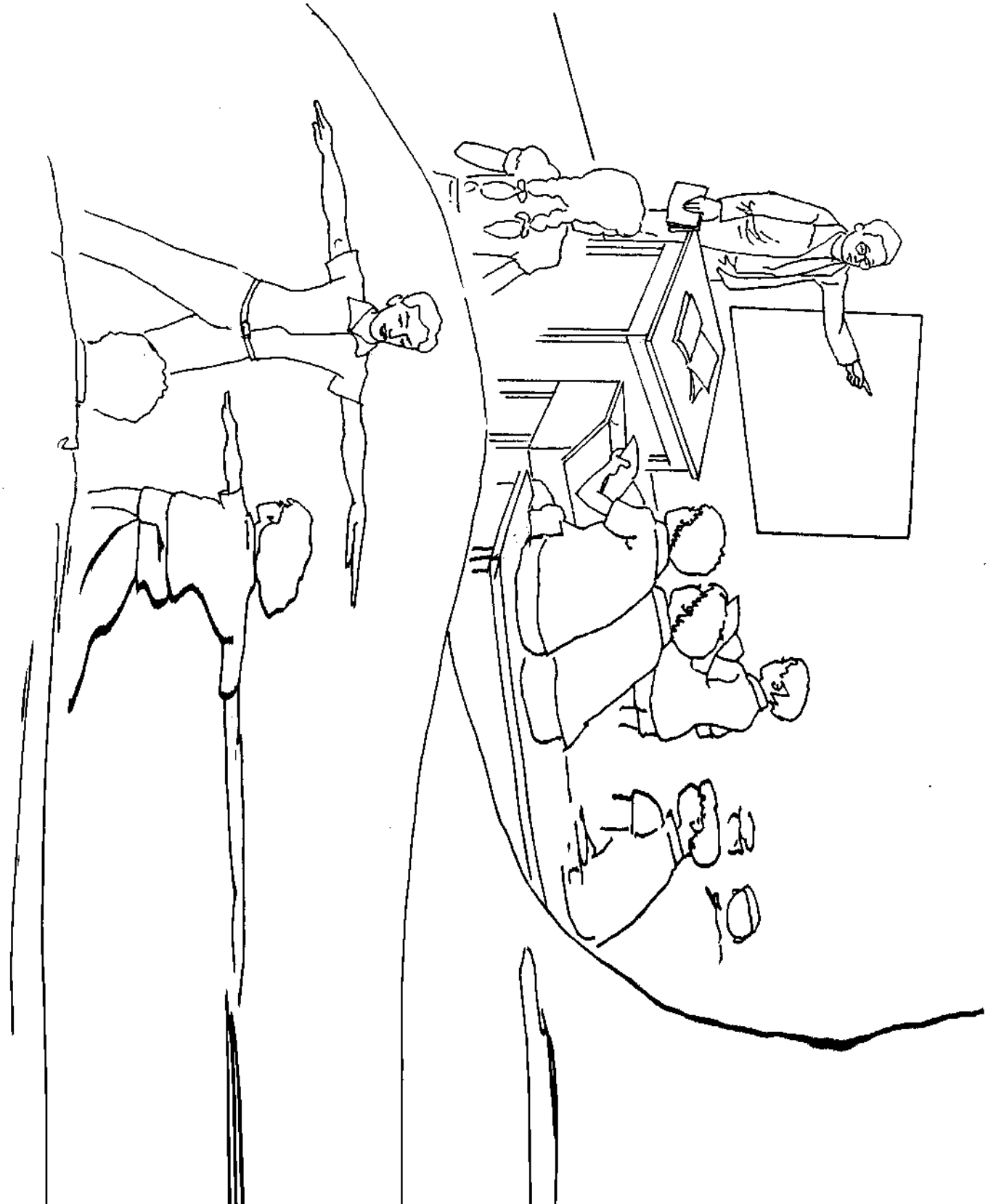
Reading passage in Malayalam (Meaning translated to English)

Everyone will have special liking for their country and their language. Without exception, I too like my country and language. Even the memories of my country and language are sweet. The pleasure of ones country and language is not comparable to anything else. This little Kerala state with lots of rivers, backwaters, villages, towns, townships, coconut groves and dense forests is like heaven for travellers. You must have heard of the saying that 'Kerala is Gods own country'? In one way it is just not a saying, it is a reality. My Kerala is that beautiful. No one can explain Kerala's beauty orally or through writing. You have to see it and enjoy the feeling of its beauty. Then alone will you get a wholistic picture of Kerala.

Kerala which is rich in greenery can be divided into three regions based on whether it is hilly region, plateau or plains. Plantation in the hilly regions includes rubber, tea, coffee and cardamom. In plateaus they cultivate ginger, turmeric, pepper and tapioca. In the plains they cultivate coconut and paddy. It is believed that the people of Kerala were the first ones from the Southern part of India to establish contact with people from the West. You can see coconut in all regions in Kerala. People of Kerala use the trunk of coconut tree to build houses, coconut for food, coconut oil as edible and hair oil and coconut leaf for thatching roof. Coconut tree is indeed a boon for people of Kerala.

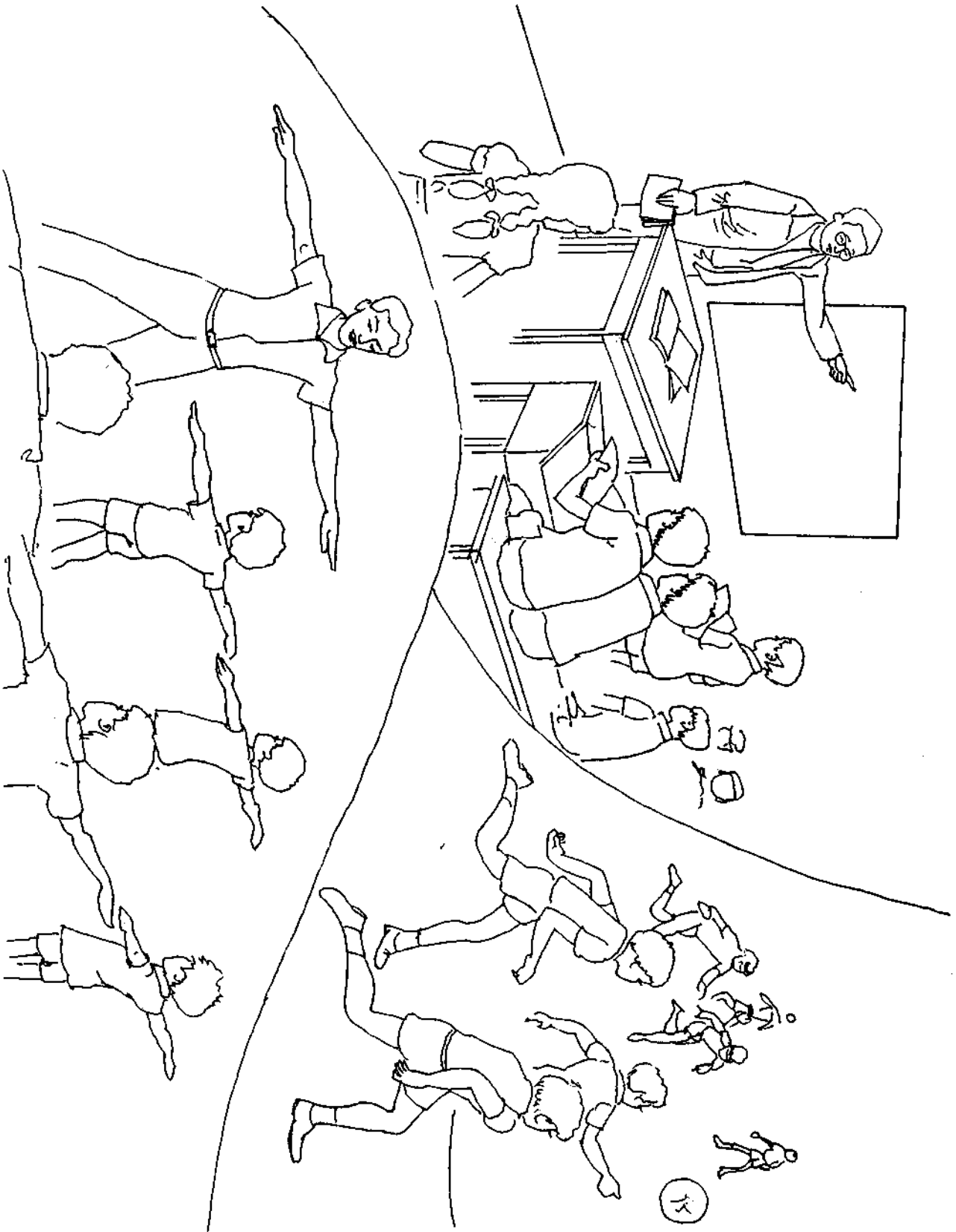
APPENDIX 5

Picture stimuli used for narration task
[Adapted from Linguistic Profile Test in Kannada by Karanth (1980)]



APPENDIX 5

Picture stimuli used for narration task
[Adapted from Linguistic Profile Test in Kannada by Karanth (1980)]



APPENDIX 5

Picture stimuli used for narration task

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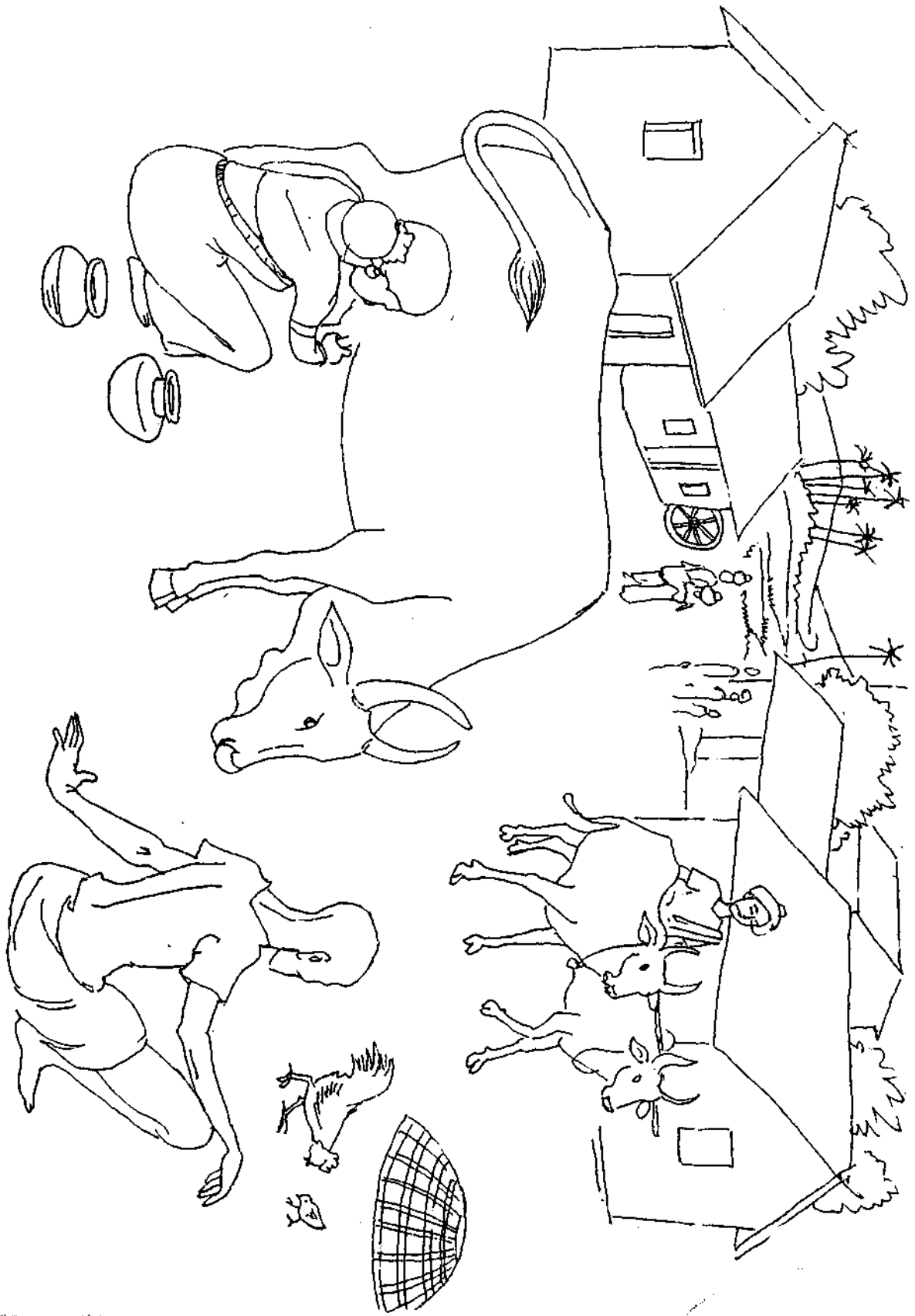

APPENDIX 6

Dimensions of speech for perceptual analysis

[Based on Darley, Aronson and Brown (1975)]

No.	*Abbreviation*	*Description*
1.	Pitch level	Pitch of voice sounds consistently too low or too high for individual's age and sex.
2.	Pitch breaks	Pitch of voice shows sudden and uncontrolled variation (falsetto breaks)
3.	Monopitch	Voice is characterized by a monopitch or mono-tone. Voice lacks normal pitch and inflectional changes.
4.	Voice tremor	Voice shows shakiness or tremulousness
5.	Monoloudness	Voice shows monotony of loudness. It lacks normal variations in loudness
6.	Excess loudness variation	Voice shows sudden, uncontrolled alterations in loudness, sometimes becoming too loud, sometime too weak
7.	Loudness decay	There is progressive diminution or decay of loudness
8.	Alternating loudness	There are alternating changes in loudness
9.	Loudness (overall)	Voice is insufficiently or excessively loud
10.	Harsh voice	Voice is harsh, rough and raspy
11.	Hoarse (wet) voice	Wet, "liquid sounding" hoarseness
12.	Breathy voice (continuous)	Continuously breathy, weak and thin
13.	Breathy voice (transient)	Breathiness is transient, periodic, intermittent
14.	Starined-strangled voice	Voice (phonation) sounds strained or strangled (an apparently effortful squeezing of voice through glottis)
15.	Voice stoppages	There are sudden stoppages of voiced air stream (as if some obstacle along vocal tract momentarily impedes flow of air).

No.	*Abbreviation*	*Description*
16.	Hypernasality	Voice sounds excessively nasal. Excessive amount of air is resonated by nasal cavities
17.	Hyponasality	Voice is denasal
18.	Nasal emission	There is nasal emission of airstream
19.	Forced inspiration-expiration	Speech is interrupted by sudden, forced inspiration and expiration sighs
20.	Audible inspiration	Audible, breathy inspiration
21.	Grunt at end of expiration	Grunt at end of expiration
22.	Rate	Rate of actual speech is abnormally slow or rapid
23.	Short phrases	Phrases are short (possibly due to fact that inspirations occur more often than normal). Speaker may sound as if he has run out of air. He may produce a gasp at the end of a phrase
24.	Increase of rate in segments	Rate increases progressively within given segments of connected speech
25.	Increase of overall rate	Rate increases progressively from beginning to end of sample
26.	Reduced stress	Speech shows reduction of proper stress and emphasis patterns
27.	Variable rate	Rate alternatively changes from slow to fast
28.	Prolonged intervals	Prolongation of interword or intersyllable intervals
29.	Inappropriate silences	There are inappropriate silent intervals
30.	Short rushes of speech	There are short rushes of speech separated by pauses

No.	*Abbreviation*	*Description*
31.	Excess and equal stress	Excess stress on usually unstressed parts of speech, e.g., (1) monosyllabic words and (2) unstressed syllables of polysyllabic words
32.	Imprecise consonants	Consonant sounds lack precision. They show slurring, inadequate sharpness, distortions, and lack of crispness. There is clumsiness from one consonant sound to another.
33.	Phonemes prolonged	There are prolongation of phonemes
34.	Phonemes repeated	There are repetitions of phonemes
35.	Irregular articulatory breakdown	Intermittent nonsystematic breakdown in accuracy of articulation
36.	Vowels distorted	Vowel sounds are distorted throughout their total duration
37.	Intelligibility (overall)	Rating of overall intelligibility or understandability of speech
38.	Bizareness	Rating of degree to which overall speech calls attention because of its unusual, peculiar, or bizarre characteristics