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Phonatory Characteristics in Subjects with Focal Cerebellar Lesions Preliminary Findings

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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). 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.

Phonatory 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; Gilman & Kluin, 1992) as well as acoustic attributes 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 (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 F0 for /a/ as compared to high vowels /i/ and /u/.

Zwirner, et al. (1991) found no significant difference between the mean F0 values of male ataxic subjects [123 Hz (SD 32, range 83-176)] compared to the mean F0 values of normal male controls [118 Hz (SD 15, range 99-147)]. They found that the standard deviation of F0 (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 (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, 1983). Ackermann and Ziegler (1994) found a quasi-rhythmic modulation of the F0 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 3Hz 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 (vF0) 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.

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 F0 were observed in ataxic dysarthria. The phonatory deviations, in general, may be due to underlying dysmetria in subjects with cerebellar lesion.

Method

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) They were in the age range of 20 to 51 years. (c) They were native speakers of Malayalam language which is a Dravidian language spoken in Kerala state in India. (d) The education qualification of the subjects varied from matriculation to graduation. (e) 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. (f) The speech and voice samples of the subjects were recorded within 4 months of the onset of dysarthria. (g) All subjects were subjected to computerized tomography and / or magnetic resonance imaging investigations and the evidence for lesions in the cerebellum was established. (h) 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 site of lesion within the cerebellum of the seventeen subjects were recorded based on CT & / or MRI 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 MRI 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 1.

Table 1: 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 51years with a mean age of 37.5 years.

Material

1) Proforma for assessment of dysarthria
2) Proforma for neurological examination of dysarthria
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.

An overview of the tasks included in the study is given in Table 2.

Table 2: *Overview of the tasks and the measures obtained from these tasks*

	Task	Dimensions measured		
		Spectral	Temporal	Perceptual
Phona	Maximum phonation duration	• Fundamental frequency		-

	(MPD) for vowels /a/, /i/ and /u/	• Frequency perturbation • Amplitude perturbation • Noise related	MPD (s)	
	Maximum fricative duration for /s/ and /z/	-	Maximum fricative duration and s/z ratio	-

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.

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.

Test Protocol and Analysis

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 3.

Table 3: *Protocol for phonatory tasks*

Domain tested	Task	Purpose
I A	Sustained vowel prolongation of /a/, /i/ and /u/	To assess laryngeal function
II A A. Phonation	Sustained prolongation of /s/ and /z/ and s/z ratio	To infer on laryngeal 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 mid-portion 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 4.

Table 4: 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	vF0 (%)
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

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 /z/ 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 (Boone, 1977), indicating that voiceless expiration time closely matches the maximum phonation time.

RESULTS AND DISCUSSION

The aims of the study are to analyze the voice dimensions in subjects with ataxic dysarthria due to lesions in various sites of the cerebellum, using acoustic 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 5.

Table 5: *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 Measures: 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/ and /u/. The measure of Maximum Phonation Duration reflects on the coordination of respiratory and laryngeal processes.

The results in Table 6 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 6*: Mean (sec) and SD for measures of maximum phonation duration for /a/, /i/ and /u/ of normal controls 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)	17.51 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 /u/										
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 (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 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 /z/. Production of voiceless fricative /s/ requires the coordination of respiratory and supralaryngeal structures, while the production of voiced fricative /z/ 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 /z/ sound contrasted with a sustained expiration without phonation for voiceless sound /s/. 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 7 and 8 gives the confidence intervals (CI), Mean (sec) and SD for /s/, /z/ measures and s/z ratio respectively.

Table 7*: 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	4.53	9.87	8.62	8.12
CI	19.54 to 21.97	18.81to 22.20								

Table 8: *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	RSP	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 7 indicate that the duration of /s/ and /z/ are reduced in all the experimental subgroups, with the exception of right posterosuperior (RPS) and right anterosuperior (RAS) subjects. The duration of /s/ and /z/ 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 /z/, 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 8 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.

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/ 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 F0 (vF0) (%)

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 9 to Table 18.

a. Fundamental Frequency Related Parameters

(i) Fundamental Frequency (F0) Hz

Fundamental frequency (F0) for normal controls and experimental subjects are given in Table 9.

Fundamental frequency (F0) 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. F0 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 9).

Table 9: *Mean (Hz) and SD for normals (N) and experimental groups and confidence intervals (CI) for normals for fundamental frequency (F0) in Hz. [(*) indicate values outside the confidence interval]*

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

F0 is reportedly a highly inconsistent feature in ataxic dysarthria with some studies reporting lower F0 (Brown, et al. 1970; Chenery, et al. 1990) and others a higher F0 (Joanette & Dudley, 1980). Ackermann and Ziegler (1994) reasoned that higher F0 is the general trend seen in subjects with ataxic dysarthria. In this study none of the experimental groups showed

a higher F0 compared to normal control subjects. The F0 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 F0.

The findings of this study does not confirm the general impression of a higher F0 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 F0, 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 10 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 anteroinferior), superior vermis and right superior paravermal groups are higher than the normal control subjects (Table 10), 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 F0 (Ladd, 1984).

Table 10: *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	-	-	-	-
/u/	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 F0 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 F0 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 F0 due to phonatory dysmetria and hence there could be variations in F0. 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 F0 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. F0 is controlled mainly by laryngeal muscles (stiffness and vibratory pattern of vocal folds), whereas F0 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 F0 range.

b] Frequency Perturbation Parameters

(i) Jitter Percentage (%)

Jitter percentage indicates cycle to cycle variation in pitch and is a short term measure of F0. Higher values in Jitter percentage indicate irregular vocal fold vibration. Table 11 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 11). 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).

Table 11: *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 (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 F0 variations (frequency range and jitter) are localized within left superior paravermal, left anteroinferior, superior vermis and right superior paravermal regions of the cerebellum.

(ii) Smoothed Pitch 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 12 gives the Mean and SD for SPPQ for normal and experimental groups along with confidence intervals for normals.

Table 12: *Mean (%) and SD for 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	-	-	-	-
/u/	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	-	-	-	-
/u/	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 12). In other words it may be inferred that left superior paravermal, left anteroinferior, superior vermis and right superior paravermal groups demonstrated increased F0 variations even after a smoothing factor is applied. The smoothing factor helps to smooth out most of the variations in F0, except for

major variations in F0. Hence it may be speculated that pitch variations due to irregular vocal fold vibrations or irregular changes in subglottal pressure (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 F0 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 F0 variations. It may be inferred that anatomical correlates related to cycle to cycle variations in F0 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 F0 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 F0 measures (PFR) and short term F0 measures (Jitter percentage and variation in fundamental frequency).

(ii) Variation in Fundamental Frequency (vF0) (%)

Variation in fundamental frequency (vF0 %) is calculated as the relative standard deviation of the period-to-period calculated fundamental frequency (standard deviation of F0 / F0). It reflects the very long term variations of F0 for the analyzed sample. Table 13 gives the Mean and SD for vF0 (%) for normal and experimental groups and the confidence intervals for normals.

Table 13: *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 (%)	1.23	6.26*	14.15*	2.54*	1.45*
	(SD)	(0.98)	(2.41)	(5.62)	(3.42)	(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	-	-	-	-

vF0 (%) 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 13). Thus all the cerebellar regions included in this study seem to be involved in controlling aspects related to vF0 (%). It may be presumed that anatomical correlates underlying long term variations in F0 may be localized in more number of cerebellar regions compared to localization of neural correlates related to absolute measures of F0 and short term variations of F0. vF0 (%) is increased in subjects with right as well as left superior paravermal lesions. Interestingly, vF0 (%) 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 vF0 (%) is a characteristic finding reported in ataxic dysarthric subjects with nonfocal lesions (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 vermis 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 F0) obtained from sustained phonation of vowels /a/, /i/ and /u/. Neural correlates underlying the production of F0 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 (vF0 %) is increased in all the experimental groups compared to normal controls.

Neural correlates for precise coordinated movements as is required for steady F0 (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 (vF0 %) seem to be localized in more regions of the cerebellum. These differences may be due to the differences in the subsystems controlling these parameters. F0 control mainly involves laryngeal control whereas variations in F0 (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 (F0 variations),

whereas absolute F0 which principally involves laryngeal control seem to be represented only in the superior vermis region of the cerebellum. All 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 (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 14 gives the Mean and SD for Shimmer percentage for normal and experimental groups and the confidence intervals for normals.

Table 14: *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 (3.12)	*10.32 (4.12)	3.36 (1.51)	3.23 (2.56)
CI	CI	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)
	CI	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)
	CI	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..90 (3.82)	*5.15 (5.71)
	CI	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)
	CI	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)
	CI	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 14). 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 (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 (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 15 gives the Mean and SD for SAPQ for normal and experimental groups, including the confidence intervals for normals.

Table 15: *Mean (%) and SD for 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 vermis group (Table 15). SAPQ is comparable to normal control subjects in all the groups with right hemispheric lesions except for increased for vowel /i/ 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 15 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 (vF0 %) 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.

(iii)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 16 gives the Mean and SD for variation in amplitude for normal and experimental groups and confidence intervals for normals.

Table 16: *Mean (%) and SD for 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 to13.28	-	-	-	-
/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 (%)	13.83	*18.25 (9.83)	*24.32 (6.04)	*26.19 (9.13)	14.50 (9.11)

	(SD)	(1.03)				
	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 16). 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 (vF0). 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 16 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
F0 (Hz)			*			
PFR	*	*	*	*		
Frequency perturbation related parameters						
	LSP	LAI	SV	RSP	RPS	RAS
Jitt (%)	*	*	*	*		
SPPQ (%)	*	*	*	*		
vF0 (%)	*	*	*	*	*	*
Amplitude perturbation related parameters						
Shim (%)	*	*	*	*		
SAPQ (%)	*	*	*			
vAm (%)	*	*	*		*	

F0 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 F0 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 F0 and amplitude variations. The right posterosuperior region of the cerebellum seems to be involved in controlling only long term F0 and amplitude measures. Right anterosuperior region is involved only in the control of long term F0 perturbation (vF0).

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 17 gives the Mean and SD for NHR for normal and experimental groups and confidence intervals for normals.

Table 17: *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 (%)	0.14	*0.16	*0.21	*0.18	0.14
	(SD)	(1.18)	(3.09)	(5.10)	(4.14)	(2.17)
	CI	0.13 to 0.15	-	-	-	-
/i/	Mean (%)	0.12	0.13	*0.31	*0.15	0.124
	(SD)	(2.27)	(3.62)	(4.18)	(5.19)	(3.83)
	CI	0.11 to 0.13	-	-	-	-
/u/	Mean (%)	0.13	*0.15	*0.18	*0.15	*0.10

	(SD)	(1.09)	(3.92)	(2.64)	(3.56)	(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 17), 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 vermis, 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 (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 18 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 18. 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 18: *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 (%)	14.13	*23.11	*36.66	*20.03	15.01
	(SD)	(4.12)	(8.56)	(12.16)	(9.87)	(5.33)
CI	CI	12.30 to 15.96	-	-	-	-
/i/	Mean (%)	14.05	14.91	*28.12	*26.04	17.21
	(SD)	3.54	(4.92)	(11.01)	(12.26)	(7.01)
	CI	10.51 to 17.59	-	-	-	-
/u/	Mean (%)	15.45	*36.28	*31.14	*24.94	*32.76
	(SD)	5.02	(15.14)	(9.87)	(10.51)	(10.87)
	CI	13.39 to 17.51	-	-	-	-
Groups		N	LAI	SV	LSP	RSP
Gender		*Female*	*Female*	*Female*	*Female*	*Female*
/a/	Mean (%)	12.58	14.62	*52.50	*34.23	*31.33
	(SD)	5.58	(5.44)	(19.21)	(9.42)	(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 (F0 & 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 F0, whereas subjects with superior vermis lesion had reduced F0. 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 vF0 (%). 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 (vF0 %). 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.

Summary

I. 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 /z/ 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).

2) Spectral Measures

- Fundamental frequency (F0) 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. F0 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 (vF0 %) 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 anteroinferior (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/ 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 /i/ were comparable to normal control subjects for female subject with left anteroinferior lesion (LAI) and subject with right anterosuperior lesion (RAS).

An attempt is made to throw some light on the differential contribution of different cerebellar regions to phonation. 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 phonatory characteristics in the different regions of cerebellum, based on phonatory task based profile of subjects with ataxic dysarthria with lesions in different regions of the cerebellum.

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. The possible contribution of cerebellar nuclei to phonatory aspects could not be considered. Future studies incorporating these variables are necessary.

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References

Ivry, R. B. (1996). The representation of temporal information in perception and motor control. *Current Opinion in Neurobiology, 6,* 851 - 857.

Boutsen, F., & Christman, S. (2002). Prosody in apraxia of speech. *Seminars in Speech and Language, 23,* 245 - 256.

Van Lancker, D., & Sidtis, J. J. (1992). The identification of affective-prosodic stimuli by left - and right - hemisphere - damaged subjects: all errors are not created equal. *Journal of Speech and Hearing Research, 35* (5), 963 - 970.

Lechtenberg, R., & Gilman, S. (1978). Speech disorders in cerebellar disease. *Annals of Neurology, 3* (4), 285 - 290.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1969a). Differential diagnostic patterns of dysarthria. *Journal of Speech and Hearing Research, 12* (2), 246 - 269.

Gilman, S., & Kluin, K. J. (1992). Speech disorders in cerebellar degeneration studied with positron emission tomography. In A. Blitzer, M. F. Brin, C. T. Sasaki, S. Fahn, & K.S. Harris (Eds.), *Neurologic disorders of the larynx (*pp. 279 -285) NewYork: Thieme Medical Publishers.

Ackermann, H., & Ziegler, W. (1994). Acoustic analysis of vocal instability in cerebellar dysfnctions. *Annals of Otology, Rhinology, and Laryngology, 103*, 98 - 104.

Joanette, Y., & Dudley, J. G. (1980). Dysarthric symptomatology of Friedreich's Ataxia. *Brain and Language, 10,* 39 - 50.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1969a). Differential diagnostic patterns of dysarthria. *Journal of Speech and Hearing Research, 12* (2), 246 - 269.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1969b). Clusters of deviant speech dimensions in dysarthrias. *Journal of Speech and Hearing Research, 12* (3), 462 -496.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1975). *Motor Speech disorders*. NewYork: W.B.Saunders, Philadelphia.

Brown, J. R., Darley, F. L., & Aronson, A. E. (1970). Ataxic dysarthria. *International Journal of Neurology, 7,* 302 - 318.

Kluin, K. J., Gilman, S., Markel, D. S., Koeppe, R. A., Rosenthal, G., & Junck, L. (1988). Speech disorders in olivopontocerebellar atrophy correlate with positron emission tomography findings. *Annals of Neurology, 23,* 547 - 554.

Chenery, H., Ingram, J., & Murdoch, B. (1990). Perceptual analysis of the speech in ataxic dysarthria. *Australian Journal of Communication Disorders, 34*, 19 - 28.

Kent, R. D., Kent , J. F., Rosenbek , J. C., Vorperian, H. K., & Weismer, G. (1997). A speaking task analysis of the dysarthria in cerebellar disease. *Folia Phoniatrica et Logopaedica, 49*, 63 - 82.

Hertrich, I., Spieker, S., & Ackermann, H. (1998). Gender-specific phonatory dysfunctions in disorders of the basal ganglia and the cerebellum: Acoustic and perceptual characteristics. In W. Ziegler & K. Deger (Eds.), *Clinical Phonetics and Linguistics* (pp 448 - 457). London: Whurr publications.

Ackermann, H., & Ziegler, W. (1994). Acoustic analysis of vocal instability in cerebellar dysfnctions. *Annals of Otology, Rhinology, and Laryngology, 103*, 98 - 104.

Horii, Y. (1983). Vocal shimmer in sustained phonation. *Journal of Speech and Hearing Research, 23*, 202 -209.

Boutsen, F., Duffy, J. R., & Dimassi, H. (2004) .*An analysis of vocal unsteadiness and tremor in ataxic dysarthria.* Manuscript submitted for publication.

Duffy, J. R. (1995). *Motor Speech disorders. Substrates, differential diagnosis and management.*, St.Louis: Mosby Publications.

Kent, R. D., Kent, J. F., Duffy, J. R., Thomas, J. E., Weismer, G., and Stuntebeck , S. (2000). Ataxic dysarthria. *Journal of Speech, Language, and Hearing Research, 43* (5), 1275 - 1289.

Cannito, M. P., & Marquardt, T. P. (1997) Ataxic dysarthria. In M. R. McNeil (Eds.), *Clinical management of sensorimotor speech disorders* (pp. 217 - 247). NewYork: Thieme Medical Publishers Inc.

Gremy, F., Chevrie-Muller, C., & Garde, E. (1967). Etude phoniatrique clinique et instrumentale des dysarthries. Technique 1. Resultats chez le maladies presentant unsyndrome cerebelleux. *Revue Neurologie , 116,* 410 - 424.

Boone, D. R. (1977). *The voice and voice therapy.* Englewood Cliffs, NJ: Prentice - Hall.

Ludlow, C. L., & Bassich, C. J. (1983). The results of acoustic and perceptual assessment of two types of dysarthria. In W.R.Berry (Eds.), *Clinical dysarthria,* (pp. 283 - 294). SanDiego: College-Hill Press.

Murdoch, B. E., Chenery, H. J., Stokes, P. D., & Hardcastle, W. J. (1991). Respiratory kinematics in speakers with cerebellar disease. *Journal of Speech and Hearing Research, 34,* 768 - 778.

Maurice - Williams, R. S. (1975): Mechanism of production of gait unsteadiness by tumours of the posterior fossa. *Journal of Neurology, Neurosurgery and Psychiatry, 38*:143.

Amici, R., Avanzini, G., & Pacini, L. (1976). Cerebellar tumours. In *Monographs in Neural Sciences,* Vol. IV. Switzerland: Karger Publishers.

Keller, E., Vigneux, P., & Laframboise, M. (1991). Acoustic analysis of neurologically impaired speech. *British Journal of Disorders of Communication, 26:* 75 - 94.

Boone, D. R. (1977). *The voice and voice therapy.* Englewood Cliffs, NJ: Prentice - Hall.

Kent, R. D., & Netsell, R. (1975). A case study of an ataxic dysarthria: Cineradiographic and spectrographic observations. *Journal of Speech and Hearing Disorders, 40,* 115 - 134.

Bowman, J. P. (1971). *The muscle spindle and neural control of tongue: Implications for Speech.* Springfield, IL: Charles Thomas.

Mills, C. K., & Weisenburg, T. H. (1914). Cerebellar symptoms and cerebellar localization. *Journal of American Medical Association, 63,* 1813 - 1818.

Holmes, G. (1917). The symptoms of acute cerebellar injuries due to gunshot injuries. *Brain, 40* (4)*:* 461-535.

Holmes, G. (1922). The Croonian lectures on the clinical symptoms of cerebellar disease and their interpretation. *Lancet 1*: 1177-1182 and 1231 - 1237.

Chiu, M. J., Chen, R. C., & Tseng, C. Y. (1996). Clinical correlates of quantitative acoustic analysis in dysarthria. *European Neurology, 36,* 310 – 314

Ladd, D. R. (1984). Declination: a review and some hypotheses. In *Phonology Yearbook,* 1, 53 - 74.

Collier, R. (1974). Laryngeal muscle activity, subglottal air pressure, and the control of pitch in speech. *Haskins Laboratory Status Report on Speech Research,* SR-39/40, 137 - 170.

Maeda, S. (1976). *A characterization of American English Intonation*. Ph.D. thesis, MIT, Cambridge, MA.

Strik, H. & Boves, L. (1992). Control of fundamental frequency, intensity and vocal quality in speech. *Journal of Phonetics, 20,* 15 - 25.

Cannito, M. P., & Marquardt, T. P. (1997) Ataxic dysarthria. In M. R. McNeil (Eds.), *Clinical management of sensorimotor speech disorders* (pp. 217 - 247). NewYork: Thieme Medical Publishers Inc.

Lieberman, P., & Blumstein, S. E. (1988). *Speech physiology, speech perception, and acoustic phonetics.* Cambridge, England: Cambridge University Press.

Deger, K., Ziegler, W., & Wessel, K. (1999). Airflow tracking in patients with ataxic disorders. *Clinical Linguistics and Phonetics, 13*, 433 - 447.

Krom, G. de (1993). A cepstrum - based technique for determining a harmonics - to - noise ratio in speech signals. *Journal of Speech and Hearing Research, 36,* 254 - 266.

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