

Relational Speech Timing in Dysarthria Associated with Cerebellar Lesions in Different Loci: Word Context

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ABSTRACT

Cerebellum plays an important role in speech motor control. Various tasks like sustained phonation, diadochokinesis and conversation have been used to tap the speech timing abilities of dysarthric clients with cerebellar lesion. It has recently been proposed that not all areas of the cerebellum may be involved in speech motor control; especially aspects concerned with timing of speech events. In the present study, the timing of articulatory movements in three clients with lesions restricted to different cerebellar loci was investigated. A relational speech timing paradigm was used to investigate initial syllable vowel duration (ISVD) in these three clients. The findings were compared with 15 age and gender matched controls. The results suggest that subjects with cerebellar right hemispheric cerebellar lesion showed reduced preservation of relational timing measures as compared to cerebellar vermis lesion i.e. midline structures and cerebellar left cerebellar hemispheric lesion, thus indirectly suggesting a differential loci specific control in cerebellum for relational speech timing.

Keywords: Cerebellum, Cerebellar Hemisphere, Cerebellar Vermis, Relational Speech Timing.

INTRODUCTION

“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, lack of coordination and/or altered tone of speech musculature (Darley, Aronson & Brown 1969a, 1969b, 1975). Depending on the site of lesion, different types of dysarthrias have been identified and each of them have a characteristic symptomatology. Ataxic dysarthria, which is caused due to cerebellar disorders, is characterized by abnormalities mainly in the temporal domain of speech. Temporal deficits are reflected in the form of articulatory inaccuracy, prosodic excess and phonatory – prosodic insufficiency (Darley, Aronson & Brown 1975). Abnormalities in the control of rate, range, direction, force and timing of voluntary movements (Kent & Netsell 1975) and distortion of the overall time program for speech (Kent & Netsell 1975), reduced durational contrasts (Ackermann and Hertrich 1994) and hypotonia are considered as factors contributing to articulatory inaccuracy, prosodic excess and phonatory – prosodic insufficiency.

Supportive evidences in the literature suggest that different aspects of speech are differently lateralized in the cerebellum. Lechtenberg & Gilman (1978), observed that speech was more strongly represented in the cerebellar hemispheres than in the vermis. However, the details of which feature of speech in particular are represented in the cerebellar hemispheres were not reported in this study. The speculation that different features of speech, are controlled at different loci in the cerebellum and thus ataxic dysarthria is not homogenous has been supported by various investigators (Duffy 1995; Kent, Duffy, Slama, Kent & Clift 2001). This is further strengthened by different reports available on the description of ataxic dysarthria. For example, the feature, “excess and equal stress” was reported as the prominent feature by (Darley, Aronson & Brown 1975). In contrary, Ackermann, Vogel, Peterson & Poremba (1992) described articulatory deficits and slow speech tempo as the most prominent feature in ataxic dysarthria. These differences may have a common underlying factor, that is, it is quite possible that inconsistency in the reported features is because of the differential localization of speech timing in different loci of the

cerebellum. If this is true, there is no report available which describes the exact nature of timing abnormalities in cerebellar lesions. The study attempts to address this aspect. It aims to study the difference if any in the speech timing control in subjects with lesions in different cerebellar loci.

One of the specific measures in speech is the Initial Syllable Vowel Shortening (ISVS), which reflects the timing control in speech. Initial syllable vowel shortening is the systematic shortening of base word and base word vowels with the addition of syllables to the base word (Port 1981). This reflects the intrinsic timing control that is executed by the central control mechanism during speech production, in order to keep up the temporal accuracy and appropriateness in natural speech. Any lesion in cerebellum may disrupt this subtle intrinsic control in speech timing, since principally, cerebellum is said to be the internal time clock for all activities in the human being including that of speech (Ivry, Keele & Diener 1988; Ivry & Keele 1989). Further, the study assumes that initial syllable vowel shortening may be differently affected in individuals with lesions in different sites of cerebellum. Thus far, no study has been carried out to investigate the relational speech timing in individuals with cerebellar lesions in different loci.

Aims of the study

To study the feature of initial syllable vowel shortening (ISVS) as a parameter of relational speech timing abilities in dysarthric individuals with cerebellar lesions in different loci and to compare with that of normal controls

METHOD

Subjects

A. Experimental group

The experimental subjects included three patients with cerebellar lesions in different loci. Demographic data of the subjects are in Table 1.

Table 1. *Demographic data of experimental subjects*

Subject	Age/Sex	Lesion
S1	28 / F	Cerebellar vermis lesion
S2	41/ M	Left cerebellar hemispheric lesion
S3	34/ M	Right cerebellar hemispheric lesion



Figure 1(a)



Figure 1(b)

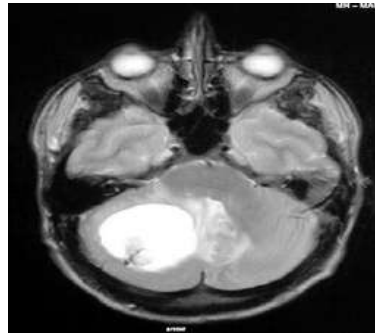


Figure 1(c)

All the subjects had cerebellar lesions of neoplastic origin as confirmed by CT/MRI. All the subjects were evaluated within one to two weeks after admission to the hospital and prior to surgery and before use of medication prior to the surgery. Subjects with any associated illnesses/ lesions, VP shunt or hydrocephalus were not included in the study.

B. Control group

Three normal control groups, C1, C2 and C3, each group with five subjects who were matched for age and gender of each of the three experimental subjects (total 15 controls) were included. They were screened for neurologic, physical, sensory and oromotor status prior to the inclusion in the study. Subject consent for participation in the study was obtained before collecting the speech sample.

Stimuli

The test stimuli included twelve sets of words in Malayalam, a Dravidian language spoken in Kerala state in South India. Each set consisted of three words: a) the first word consisted of base word only (bw) with minimum of 2 and maximum of 4 syllables, b) the second consisted of base word with suffix (bw + s1). The length of the suffix added varied from one to two syllables and c) the third consisted of the base word with suffix (bw + s1 + s2). The length of the suffix varied from five to six syllables. The initial syllable of the first four sets (Sl No. 1 to 4 in Table 2) had vowel /a/ in the initial syllable, second four sets (Sl No. 5 to 8 in Table 2) had vowel /i/ in the initial syllable and the last four sets (Sl No. 9 to 12 in Table 2) had vowel /u/ in the initial syllable.

Table 2. *List of word sets included in the experiment*

Word set	Base word (bw)	Base word + suffix (Short) (bw+s1)	Base word + suffix (Long) (bw+s1+s2)
1	/ gaɳitəṁ /	/ gaɳitaɟ a:stRəṁ /	/ ganitaɟa:stRəɳjaɳma:rum/
2	/ bahu /	/ bahudʒanaɳal /	/ bahudʒanaɳaluɟe:ta:ja /
3	/ sahaɟɟi:vi /	/ sahaɟɟi:vikəl /	/ sahaɟɟi:vikəlo:ɟuku:ɟi /
4	/ kaɟal /	/ kaɟalinte /	/ kaɟalinteakkareninnə /
5	/ piɟi/	/ piɟikalil /	/ piɟikalila:ji /
6	/ kiɟakka /	/ kiɟakkanəṁ /	/ kiɟakkanatilninnə /
7	/ piɟitʃu /	/ piɟitʃuninu /	/ piɟitʃuninnukonɟirunnu /
8	/ diɟa /	/ diɟajil /	/ diɟailninnuma:ji /
9	/ kuɟam /	/ kuɟaɳalum /	/ kuɟaɳalo:ɟuku:ɟi /
10	/ sugəməṁ /	/ sugəməma:l /	/ sugəməma:ɟiti:Rnnu /
11	/ puɟəva /	/ puɟəvakal /	/ puɟəvakalilninnu /
12	/ suɟaktəṁ /	/ suɟaktama:ɟata:ja /	/ suɟaktama:ɟata:ɟatina:l /

Task

All the test stimuli were written on separate cards and the subjects were instructed to read the words in their habitual rate of speech as naturally and as intelligibly as possible. Three trials were given for each word. The best of the 3 trials was included as speech sample for analysis.

Recording

The speech samples were recorded using a Sony-MZ-55 digital recorder with the microphone kept at a distance of 10 cm from the subject's mouth.

ANALYSIS

The speech samples were analyzed using Spectrogram module in Computerized Speech Lab (CSL 4400) software. The following measures were extracted for each of the target words.

1. Total word duration (TD): defined as the time between the initial consonant burst for the voiceless consonants / initiation of voicing for the voiced consonants/ initiation of frication for fricatives to the end of the final vowel or consonant.
2. Initial syllable vowel durations (ISVS): Initial syllable vowel durations were obtained by placing the cursors at the beginning and end of the regular pulses and auditory cues were used to confirm that the vowel was isolated.

RESULTS AND DISCUSSION

The aim of the present study was to see if there is lesion specific difference in the Initial syllable vowel shortening (ISVS) in the speech of subjects with cerebellar dysarthria. The results are discussed under the following sections:

1. The mean values (in milliseconds) of initial syllable vowel duration (ISVD) and total duration (TD) for bw, bw+s1 and bw+s1+s2 for the vowels /a/, /i/ and /u/ for control and experimental group (Table 3 and Table 4)

2. Results of one to one comparison of ISVD values of experimental with the corresponding confidence intervals established for control group.
3. Correlation between the ISVD and TD for bw, bw+s1 and bw+s1+s2 for each word set of control and experimental group (Graph 1, 2, 3 & 4 and Table 5 & 6)

Section A

The mean value in milliseconds of initial syllable vowel duration (ISVD) and TD of target words for both control and experimental subjects were tabulated (Table 3 and 4)

Table 3. Mean of ISVD (m sec) and TD (m sec) for the vowels /a/, /i/ and /u/ for bw, bw+s1 and bw+s1+s2 for control subjects (total 15 control subjects)

Vowel		Word set	C1 ISVD (msec)	C1 TD (msec)	C2 ISVD (msec)	C2 TD (msec)	C3 ISVD (msec)	C3 TD (msec)
/a/	/a1/	bw	0.0684	0.5074	0.0417	0.5252	0.0532	0.5101
	/a2/	bw +s1	0.0557	0.7802	0.0538	0.8751	0.0464	0.6720
	/a3/	bw+s1+s2	0.0433	1.0130	0.0479	1.2110	0.0413	0.8387
/i/	/i1/	bw	0.0616	0.3975	0.0509	0.4276	0.0617	0.2517
	/i2/	bw +s1	0.0413	0.5338	0.0432	0.5177	0.0587	0.4599
	/i3/	bw+s1+s2	0.0349	0.8192	0.0292	0.5560	0.0401	0.9334
/u/	/u1/	bw	0.0465	0.3714	0.0391	0.3913	0.0558	0.4144
	/u2/	bw +s1	0.0342	0.7068	0.0401	0.7901	0.0499	0.6105
	/u3/	bw+s1+s2	0.0298	0.9496	0.0438	1.0120	0.0546	0.8478

Table 4. Mean of ISVD (milliseconds) and TD (milliseconds) for the vowels /a/, /i/ and /u/ for bw, bw+s1 and bw+s1+s2 for experimental subjects

Vowel		Word set	S1 ISVD (msec)	S1 TD (msec)	S2 ISVD (msec)	S2 TD (msec)	S3 ISVD (msec)	S3 TD (msec)
/a/	/a1/	bw	0.0662	0.5449	.0811	.4943	.1394	.4902
	/a2/	bw +s1	0.0465	0.7909	.0782	.6896	.1353	.9842
	/a3/	bw+s1+s2	0.0340	1.011	.0469	1.1606	.1611	1.8985
/i/	/i1/	bw	0.0664	0.5704	0.0752	0.5982	0.1141	0.5553
	/i2/	bw +s1	0.0357	0.7050	0.0561	0.7539	0.1055	0.8813
	/i3/	bw+s1+s2	0.030	1.1600	0.0386	1.0385	0.1192	1.2398
/u/	/u1/	bw	0.0572	0.5051	0.0712	0.5209	0.1911	0.5572
	/u2/	bw +s1	0.0435	0.8701	0.0573	0.7225	0.1891	1.0155
	/u3/	bw+s1+s2	0.0374	1.4460	0.0396	1.2430	0.1780	1.6020

From Table 3, it is seen that the control subjects consistently showed a decrease in initial syllable vowel duration (ISVD) with increase in total duration (TD) of the word. From Table 4, it is seen that the subject S1 with cerebellar vermis lesion showed a reduction in ISVD with increase in TD across bw, bw+s1 and bw+s1+s2. The Subject S2 with left cerebellar hemisphere lesion also showed a similar trend to that of the cerebellar vermis lesion. An opposite trend was observed in S3 with right cerebellar hemisphere lesion, wherein there is either an increase or stable ISVD with increase in TD.

Section B

Confidence limits were set up for the normal controls for ISVD and TD for all the 12 word sets having vowel /a/ in initial syllable (first four), /i/ in the initial syllable (fifth to eight) and /u/ in the initial syllable (the last four). Since the mean value of the experimental subjects as shown in Table 4 may not have represented the raw scores obtained in the four words i.e. 4 (bw), 4 (bw+s1) and 4 (bw+s1+s2) for each vowel /a/, /i/ and /u/, confidence limits were set up for the controls. Confidence limits are the value limits calculated for controls, within which the raw scores of experimental subjects have to fall in order to infer if there is a significant difference from the normal control subjects. The scores falling outside the confidence limits suggest a significant difference from normal scores. For example, if the confidence limit for /a/ in the bw of first word set is 0.0440 to 0.0701 and the corresponding raw score for experimental subject is 0.0901, then this value is significantly different from control group. One to one comparison of the individual values of the initial syllable vowel durations (ISVD) for the vowels /a/, /i/ and /u/ for the three patients were compared with the corresponding confidence intervals.

As there was no significance difference ($t=.42$, $p>.05$ level) between the three control groups (C1, C2 and C3) for ISVD and TD for all the three vowels, a composite confidence interval was obtained for ISVD and TD for all the 12 word sets (Table 5)

Table 5. *The composite confidence intervals established for normal control subjects*

	ISVD			TD		
	bw	Bw+s1	Bw+s1+s2	bw	Bw+s1	Bw+s1+s2
vowel	.0335- .0684	.0342- .0593	.0298- .0489	.3975- .5474	.6720- .7921	.7480- 1.018

Comparison of the individual values of experimental subjects for ISVS and TD with composite confidence intervals obtained for normal control subjects was made. From the comparison, it is evident that the number of values for S3 (right cerebellar) confined to the confidence intervals specified, is very less for both initial syllable vowel shortening and total duration when compared to that of the subjects S1 (vermis) and S2 (left cerebellar). The right cerebellar lesion subject showed deviant values for bw, bw+s1 and bw+s1+s2 for all the three vowels for ISVD as well TD, on comparison with the confidence intervals. All the values for ISVD and TD for this subject was increased in relation to normal control subjects. In the subject with left cerebellar lesion, the values of the bw was increased compared to normal control subjects for vowel /a/ for both ISVD and TD. The value of bw+s1+s2 was increased for vowel /a/ for ISVD and TD for the left cerebellar subject. For the subject with midline cerebellar lesion, the increased values on comparison with the confidence intervals for normals can be seen only for TD for bw+s1+s2 for /I/ and /u/, bw + s1 for /u/ and bw for /i/.

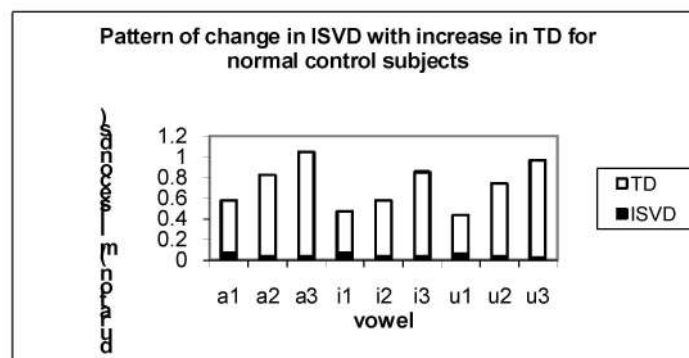
Section C

Pearson product moment correlations were found for the ISVD in bw, bw+s1 and bw+s1+s2 and their corresponding TD for each word set. This was done to see if there is significant correlation between ISVD and TD for each word set and if it is significant, whether it is negative or positive. The correlations were calculated for both the control and experimental subjects.

There was a significant negative correlation at $p < 0.05$ level between the ISVD and TD for 11 out of 12 word sets for control subjects as can be seen from Table 6. This shows that the controls showed a decrease in ISVD with increase in the TD.

Table 6. *Correlation of ISVD with their corresponding TD for bw, bw+s1 and bw+s1+s2 for control subjects*

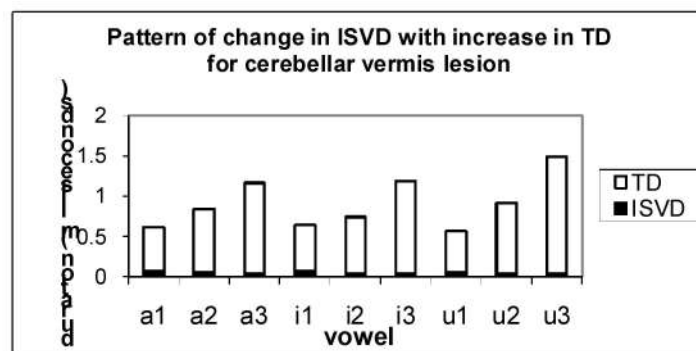
Vowel /a/	r	Vowel /i/	r	Vowel /u/	r
1	-0.9960	5	-0.9390	9	-0.4130
2	+0.6470	6	-0.4300	10	-0.8750
3	-0.9260	7	-0.9970	11	-0.8280
4	-0.9730	8	-0.4620	12	-0.8470



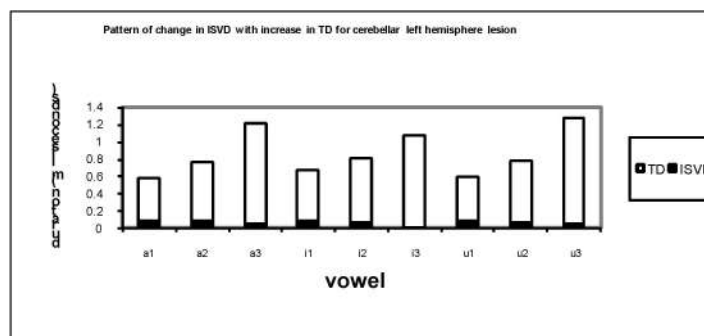
Graph 1

Table 7. *Correlation of ISVD with their corresponding TD for bw, bw + s1 and bw+s1+s2 separately for each of the 12 word sets for experimental subjects*

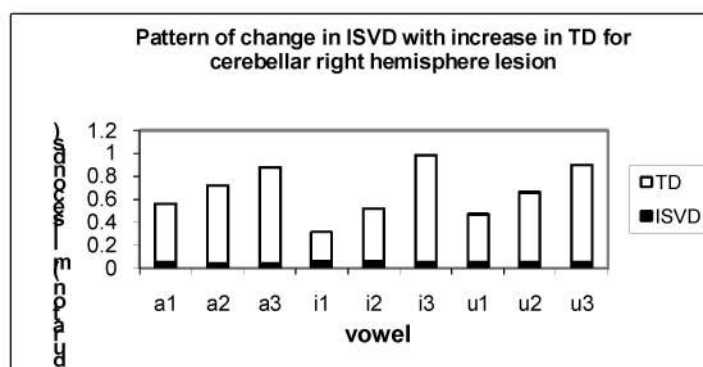
Word set	S1(vermis)	S 2 (left)	S3 (right)
/a / 1	-0.925	-0.969	+0.355
2	+0.508	-0.515	+0.956
3	-0.986	-0.340	+0.572
4	+0.066	+0.894	+0.862
/i/ 5	-0.189	-0.926	+0.984
6	-0.363	-0.200	+0.329
7	-0.902	-0.257	+0.584
8	-0.274	-0.934	-0.236
/u/ 9	-0.997	-0.908	+0.588
10	-0.935	-0.819	+0.986
11	+0.931	+0.430	+0.937
12	-0.605	-0.761	+0.624



Graph 2



Graph 3



Graph 4

In experimental subjects, a significant negative correlation is evident for subject S1 and S2 i.e. the subjects with cerebellar vermis lesion and cerebellar left hemisphere lesion respectively; as can be seen from the Graph 2 and Graph 3. From Table 7 it can be observed that nine out of twelve word sets for cerebellar vermis lesion and ten out of twelve word sets for cerebellar left hemisphere lesion, showed a significant negative correlation at $p < 0.05$ level for these subjects. This indicates that for S1 and S2 there is a significant reduction in vowel length with increase in total word duration. This implies that the relational timing seems to be preserved in these clients. The subject S3 showed a positive correlation for eleven out of twelve word sets, which was again significant at $p < 0.05$ level. This is in contrast to the trend shown by S1 and S2. Thus in S3 there is either an increase or maintenance of ISVD with increase in TD. Thus, it may be implied that right cerebellar hemispheric lesion seems to have less preserved relational speech timing abilities.

DISCUSSION

To summarize the findings, it can be seen that the subject with vermis lesion and cerebellar left hemispheric lesion showed a decrease in vowel length with increase in word length. Subject S3 showed an opposite trend for almost all the stimuli. This suggests that impairment in right hemisphere of cerebellum may reflect in impaired relational speech timing. This finding is in congruence with the findings of Urban, Marx, Hunsche, Gawehn, Vucurevic, Wicht, Massinger, Stoeter & Hopf (2003), that the right cerebellar hemisphere is more involved in the coordination of articulatory movements of the tongue and orofacial muscles. Ackermann et al. (1992) also implicated the role of the right cerebellum in causing dysarthric deficits. It can also be speculated that the impaired relational speech timing abilities in right cerebellar hemisphere damage may be because of the crossed cerebello-cerebral interaction in terms of speech timing. Boutsen & Christman (2002) opined that the timing of intrinsic prosodic structures such as syllable timing is represented in the left cerebral hemisphere. The deviant relational speech timing in the right cerebellar hemisphere

lesion may be attributed to the reciprocal connection of the right cerebellar hemisphere to the left cerebral hemisphere and the role of the left cerebral hemisphere in controlling syllabic timing. Moreover, Boutsen & Christman (2002), also speculated that the right cerebellar hemisphere is involved in the fast coordination of supralaryngeal and laryngeal movements especially those involving syllable integrity and tonic stress. This might have led to the finding of altered values or reduced preservation of relational speech timing abilities in the right cerebellar subject in this study.

CONCLUSION

The study provides some preliminary indications about the localization of speech timing in the cerebellum. The cerebellar vermis and cerebellar left hemisphere seems to be less involved in the control of speech timing. The right cerebellum seems to be more representative of relational speech timing control. The involvement of right cerebellar hemisphere in relational speech timing may be reasoned on the basis of the cerebello-cerebral interactions and the role of the left cerebral hemisphere in the control of intrinsic prosody such as syllabic timing. This finding also throws light on one of the possible reasons for inconsistencies in the reported findings in the literature as to the presence or absence of speech symptoms in cerebellar lesions. That is, it may offer an explanation as to why some clients with cerebellar lesions present with speech symptoms and some do not present speech symptoms. The study has included only three experimental subjects with focal and isolated cerebellar lesions localized to specific regions of the cerebellum. The study needs to be replicated with more number of subjects.

IMPLICATIONS

It is important to explain why distinct forms of cerebellar dysarthrias are seen in the clinical population. The finding has relevance in clinical assessment and management of cerebellar dysarthrics. The study provides an insight into a preliminary

theoretical basis to localization of functions as related to speech timing within the cerebellum.

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