Original Paper

European Neurology

Eur Neurol 1996;36:310–314

Ming-Jang Chiu
Rong-Chi Chen
Chiu-yu Tseng

Department of Neurology, National Taiwan University Hospital, College of Medicine, National Taiwan University, and Institute of History and Philology, Academia Sinica, Taipei, Taiwan

Clinical Correlates of Quantitative Acoustic Analysis in Ataxic Dysarthria

Key Words
Ataxic dysarthria
Acoustic analysis
Instrumental measurement
Cerebellum
Fundamental frequency
Voice onset time
Tone language
Taiwanese

Abstract
The speech of 15 Taiwanese patients with cerebellar dysfunction and ataxic dysarthria was investigated utilizing techniques of acoustic analysis and instrumental measurement. Semiquantitative assessment of cerebellar function (SQACF) was also performed. The results of the acoustic analysis of the cerebellar speech were then correlated with the findings in the SQACF. The overlapping of voice onset time highly correlates with truncal ataxia as well as eye movement abnormality and eye-hand coordination. The prolongation of vowel duration also correlates significantly with tandem walking, dysdiadochokinesia and eye movement abnormality. The correlation of the speech function with other cerebellar motor functions provides further insight into the functional anatomy for cerebellar speech motor control. This study concludes that the midline structures – vermis and fastigial nucleus – seem to be the primary focus for the coordination of motor speech in the cerebellum.

Introduction

Lesion of the cerebellum or its connections results in a state called 'ataxia' in which movements become uncoordinated. Various uncoordinated movements may be related to different parts of the cerebellum or different mechanisms. Dysarthria has been described with cerebellar disease since the 19th century [1] using the term 'scanning speech'. However the role of the cerebellum in speech motor control is still not known with certainty. When ataxia affects the muscles of the speech mechanism, the production of speech becomes abnormal, leading to a cluster of deviant speech patterns referred to 'ataxic dysarthria' [2, 3]. In clinical medicine the following descriptors for ataxic speech have been used [4]: 'slow, slurred, irregular, labored, intermittent, jerky, explosive, staccato, singsong, and scanning'. But they are nonquantitative and unable to describe the severity of the deviant dimensions in terms of measurement. Therefore, instrumental acoustic analysis [5, 6], capable of quantification and good temporal resolution (up to milliseconds), should be applied. With such methodology the following acoustic deviant patterns in ataxic dysarthria have been derived in our previous study [unpublished]: marked prolongation of vowel duration, obvious elevation of the mean fundamental frequency (F_0), evident overlapping of the voice onset time (VOT) boundary and moreover significant decrease in the correct rates in all three categories of production, i.e., consonants, vowels and tone. This observation in Taiwanese, a tone language, supports in part pre-

KARGER
E-Mail karger@karger.ch
Fax + 41 61 306 12 34
http://www.karger.ch

0014–3022/96/0365–0310$10.00/0

Dr. Ming-Jang Chiu
Department of Neurology
National Taiwan University Hospital
No. 7 Chung-Shan S. Rd.
Taipei 100 (Taiwan)

Received:
July 28, 1995
Accepted:
March 7, 1996

vious spectrographic findings in English-speaking ataxic dysarthrics by Kent and Netsell [7] and Kent et al. [8]: alterations of the normal timing pattern with prolongation of a variety of segments.

The purpose of this study was to investigate the possible relationship between the acoustic deviation in the speech of ataxic dysarthria in terms of measurement and the concomitant motor dysfunction by semiquantitative clinical assessment, in the hope that new insight into possible functional anatomy of the cerebellar speech motor control could be obtained.

Patients and Methods

Subjects. Fifteen Taiwanese patients with cerebellar dysfunction and ataxic dysarthria were included in this study. There were patients with cerebellar degeneration (n = 7), cerebellar stroke (n = 5), sequelae of head injury (n = 1), demyelinating disease (n = 1) and cerebellar tumor (n = 1). Only patients with cerebellar dysfunction whose lesion was mainly localized in the cerebellum were included. Patients with apparent pyramidal and/or extrapyramidal symptoms and signs were excluded. All patients were tested after their acute illness. Fifteen control subjects matched for age, sex and language background were also recruited. All cases were subjected to a complete neurological examination with emphasis on cerebellar function. Semiquantitative assessment of cerebellar function (table 1) specified as tandem walking, truncal ataxia, limb ataxia, fine rapid alternative movement (RAM), eye-hand coordination (eye-hand), and eye movement disorder was performed. All patients with cerebellar dysfunction received at least one neuroimaging study (brain MRI or cranial CT scan) to validate their lesion localization. In some cases, electro-oculography was performed to confirm the existence of eye movement abnormalities.

Test Material. The test for speech function was comprised of 33 Taiwanese monosyllable words. All these words are CV (consonant + vowel) in structure, and are composed of an initial stop consonant /p, p^h, b, t, t^h, k, k^h, g/ plus the vowel /i/ with 5 different tones: high level (1st tone), rising (2nd tone), high falling (3rd tone), low falling (4th tone) and mid level (5th tone) tones. (There are 7 tones in Taiwanese, but the 2 short tones are CVC in structure, therefore not included in the study.) All subjects were instructed to repeat sentences after the examiner at a comfortable speaking rate, the target words embedded in the carrier sentences 'tse^1 çi^4 ___ ___ *(disyllabic word)* e^5 ___ *(monosyllabic target)*', which means 'This is the *target final syllable* of the target *disyllabic word*'.

Data Preprocessing. The analog data from the audio tapes was digitized at a sampling rate of 10.24 kHz with the resolution of 16 bits. Sound spectrograms were produced by using a PC-based self-developed software on which AR model was adopted for extracting pitch values.

Data Analysis. Tape-recorded speech data were listened and transcribed. The correct rates of each target word were scored by categorizing all phonemes into vowels, consonants, and tones for correct percentage counts. VOT (wide-band spectrogram), F_0 (narrow-band spectrogram), and vowel duration in the monosyllable target words located in the final position of the carrier sentence were measured on sound spectrograms.

Table 1. Semiquantitative assessment of cerebellar function

Tandem walking
0 Walk tandem without difficulty
1 Walk tandem with difficulty: more than two steps off in a 3-meter straight line
2 Walk in shoulder-width base
3 Walk only with assistance
4 Cannot walk even with assistance

Truncal ataxia
0 Stand without difficulty
1 Titubation but can stand alone
2 Stand only with assistance
3 Titubation but can sit alone
4 Sit only with assistance

Limb ataxia
Finger to nose to finger:
Nil 0, mild 1, moderate 2, marked 3
Rt____ Lt____ dysmetria
Rt____ Lt____ dyssynergia
Rt____ Lt____ intention tremor

Dysdiadochokinesia
RAM
Ask the patient to touch each finger tip with the thumb of the same hand sequentially and then repeat the sequence again and again 5 times; scored as number of seconds consumed for 5 cycles by either hand

Eye-hand coordination (A, B)
(A) Draw line between whirling circle; score as number of crossing points by dominant hand (or nonparalytic hand)
(B) Cross all line segments on test paper; score as number of seconds consumed by dominant hand

Eye movement disorder
Nil 0, mild 1, moderate 2, marked 3
___ Ocular dysmetria
___ Slow saccade
___ Gaze-evoked nystagmus
___ Saccadic pursuit
Handedness: dex___ sin___ ambi___

Statistics. Vowel duration and F_0 were clustered with respect to tonal groups to obtain their average in each individual. The VOT was classified to each different stop consonant. The natural boundary of VOT obtained in our previous study [unpublished] was applied to estimate the severity of the overlapping of VOT in ataxic speech. VOT delay was also computed by calculating the VOT errors resulting from delaying only. The relative average pitch height for each patient was computed by subtracting the average pitch height in controls of the same sex to remove confounding effects. Spearman rank correlation coefficient was used to examine the correlates between the clinical cerebellar dysfunction and the findings in perceptual acoustic analysis and in instrumental measurement.

Table 2. Spearman rank correlation matrix of cerebellar functions and acoustic measurements

	Trk	Lmb	Lmbc	RAM	EHA	EHB	EMV	CV	CC	CT	D_av	P_av	RPav	VOTo	VOTd
Tan	*****	*	NS	****	****	NS	****	NS	NS	NS	**	NS	NS	NS	NS
Trk		NS	NS	*	****	NS	****	NS	NS	n*	c***	NS	NS	***	NS
Lmb			****	***	*	NS	***	NS	NS	NS	NS	NS	NS	NS	NS
Lmbc				**	*	****	***	n*	n*	NS	NS	NS	NS	NS	NS
RAM					******	**	**	NS	NS	NS	c*	NS	NS	NS	NS
EHA						NS	***	NS	NS	NS	NS	NS	NS	**	NS
EHB							NS	NS	NS	NS	NS	NS	NS	NS	NS
EMV								NS	NS	NS	c****	NS	NS	**	NS
CV									*	NS	NS	NS	NS	NS	NS
CC										NS	NS	NS	NS	NS	NS
CT											NS	NS	NS	NS	NS
D_av												NS	NS	c***	NS
P_av													NS	nc**	NS
RPav														NS	c*
VOTo															*

Tan = Tandem walking; Trk = truncal ataxia; Lmb = limb ataxia; Lmbc = limb ataxia corrected for limb number; RAM = rapid alternative movement; EHA = eye-hand coordination A; EHB = eye-hand coordination B; EMV = eye movement disorder; CV = correct rate of vowel; CC = correct rate of consonant; CT = correct rate of tone; D_av = average duration of vowel; P_av = average pitch height; RPav = relative average pitch height; VOTo = overlapping of VOT; VOTd = delay of VOT; c = significant in some categories; n = negative correlation.
****** $p < 0.001$; ***** $p < 0.002$; **** $p < 0.005$; *** $p < 0.01$; ** $p < 0.02$; * $p < 0.05$; $p > 0.05$ = NS.

Results

The results were summarized in the form of a Spearman rank correlation matrix (table 2). In cerebellar motor functions, tandem walking correlates rather well with truncal ataxia $r_s = 0.75$, RAM $r_s = 0.71$, eye-hand A $r_s = 0.71$ and eye movement $r_s = 0.72$. At the same time truncal ataxia also correlates rather well with eye-hand A $r_s = 0.72$ and eye movement $r_s = 0.74$. Limb ataxia, represented by finger-nose-finger examination, shows good correlation with RAM $r_s = 0.67$ and eye movement $r_s = 0.65$. We also correct the effect of number of limbs involved by dividing scores from the degenerative patients by 2, because of their relatively symmetric affection. The corrected scores of limb ataxia correlate significantly with eye-hand B $r_s = 0.70$ and eye movement $r_s = 0.67$, while they moderately correlate with RAM $r_s = 0.63$. As for RAM it excellently correlates with eye-hand A $r_s = 0.81$ and moderately with eye movement $r_s = 0.61$ and eye-hand B $r_s = 0.62$. Besides eye-hand A has close correlation with eye movement $r_s = 0.69$.

With respect to speech parameters, the average duration of all vowels shows moderate correlation with tandem walking $r_s = 0.61$. Among the 5 tones investigated, the average duration of the 3rd tone (high falling) correlates excellently with tandem walking $r_s = 0.79$, followed by the 4th tone (low level) $r_s = 0.70$, and the 5th tone (mid level) $r_s = 0.60$. The average duration of the 4th tone also has a good correlation with truncal ataxia $r_s = 0.67$. In addition, the 4th tone has a good correlation with eye-hand A, $r_s = 0.67$ and rather high correlation with eye movement $r_s = 0.72$. Our data of VOT overlapping show good correlation with truncal ataxia $r_s = 0.69$ and also moderate correlation with eye-hand A and eye movement $r_s = 0.60$. Nevertheless pathological delay for VOT shows almost no significant correlation with all cerebellar motor functions.

Discussion

Clinical signs resulting from midline cerebellar lesions consist of disordered stance and gait, truncal titubation, and disturbance of extraocular movements [9]. Correspondingly, truncal ataxia, tandem walking and eye movement are closely related to each other in our study. They all are important motor functions related to the cerebellar vermis and fastigial nuclei [10]. The close correla-

tion of VOT overlapping and prolongation of vowel duration with the above-mentioned motor functions may imply that they are speech disorders closely related to cerebellar midline structure. On the other hand, lateral lesions result in motor abnormalities such as hypotonia, dysmetria, dysdiadochokinesia, excessive rebound and impaired check, intention tremor, dyssynergia, past pointing, and eye movement disorders. Accordingly, our data shows that limb ataxia correlates well with eye-hand B, eye movement and RAM. These are important functions related to cerebellar hemisphere and dentate nucleus. However no speech parameters ever show significant correlation with limb ataxia. This further supports that VOT overlapping and vowel duration prolongation are exclusively midline cerebellar dysfunction. From the above discussion we learned that ocular motor abnormalities could be found in cerebellar lesions of either midline or lateral locations. In essence, the syndrome of dorsal vermis and underlying fastigial nuclei would manifest the following ocular motor abnormalities: enduring saccadic dysmetria, mild deficits of smooth pursuit but not marked nystagmus [11]. Most of our patients' ocular motor abnormalities were dysmetric saccade or impaired pursuit, but less slow saccade and rarely nystagmus. Hence the ocular motor disorder in our cases mainly resulted from vermian-fastigial dysfunction.

The sites of cerebellar damage most commonly associated with abnormal speech have been reported. One earlier study [12] showed that dysarthria might result from injury to the superior cerebellar vermis. A more recent study [13] further demonstrated that if ataxic dysarthria was a prominent and early sign of cerebellar damage, it might indicate a lesion of the mid-portion of the vermis. Our observations in the present study are compatible with these reports. However some researchers [14] believed that the left hemisphere of the cerebellum, especially the superior portion [15], was most commonly affected in cases of cerebellar speech dysfunction. In our observation, although the superior portion of the cerebellar hemisphere may be the location affected, they are not limited to the left hemisphere. In fact in our nondegenerative cases, 4 were mainly on the right side, 2 on the left side and 2 with bilateral involvement. This was also supported by a report [16] on patients with superior cerebellar artery infarction.

What is the possible mechanism that underlies this ataxic dysarthria? VOT is the timing between the release of the occlusion of the supralaryngeal vocal tract and the initiation of phonation that characterizes stop-consonant distinctions in syllable-initial position such as the English /ba/ versus /pa/ [17]. Breakdown of the respiratory-laryngeal coordination may account for the timing errors [18, 19] manifested as VOT overlapping. On the other hand, several possibilities have been advocated for the prolongation of the vowel duration [8]. We propose two possible mechanisms directly involving these deficits. The first is the hypotonia hypothesis [20] and the second is the revisory control of the cerebellum [21, 22]. In the first hypothesis hypotonia involving the respiratory-laryngeal subsystems was proposed as the cause of slow movement in phonation and articulation. The prolongation may reflect a delay in the generation of muscular force related to improper spindle bias [23]. Cerebellar lesions may result in defective γ motor neuron regulation of muscle spindle output [24] leading to cerebellar hypotonia. Hypotonia is usually attributed to a cerebellar hemisphere function. However vestibulospinal and reticulospinal pathways, hence fastigial nucleus, also appear to be important relays of such function [24]. This hypothesis is compatible with our observation only in case of fastigial nucleus involvement. Nevertheless most of our data do not support this hypothesis. For if generalized hypotonia is the unique underlying mechanism, prolongation and slowing should occur in all phonemes or segments, and decreased rate of RAM should also be seen. Our data showed that VOT overlapping did not result from delaying alone. Furthermore no significant correlation could be found between VOT delay and other cerebellar motor functions nor correlation between RAM and VOT overlapping.

In the second hypothesis, the problem is in the revisory control function of the cerebellum. Commands from the motor cortex are imprecise and provisional, thus requiring refinement by the continuous action of the cerebellar loop. When the cerebellar short-loop revisory function, integrating and interpreting afferent signals, is handicapped by disease, the motor control system is forced to rely on longer loops. Consequently, segment duration in speech is increased to allow time for the longer loops to operate. With this strategy, there must be disproportionate increase in the duration of segments that are normally short. In our data the 4th tone (the shortest among the 5 investigated) as well as the 3rd tone (the second shortest) showed higher correlation with the midline cerebellar dysfunction than the other tones. Therefore the prolongation deficit was more severe in shorter tones. In sumamry, the long-loop motor control took over after the short-loop cerebellar mechanism broke down and resulted in lack of precision with regard to direction, range and timing of motion. This explains our VOT overlapping observation, among which the directions of the overlapping varied.

In conclusion, our study supports the notions that the anterior lobe of the cerebellum (vermis) and other midline structures (fastigial nucleus) may relate to speech abnormality observed in ataxic dysarthria. It also offers some insight into the mechanism, i.e., the defected cerebellar revisory function may result in slowdown of motor speech patterns as well as disturbance of normal temporal relationship.

Acknowledgment

This study was supported by grant (NSC-83-0412-B-002-123) from the National Scientific Committee Taiwan, ROC.

References

1 Charcot JM: Lectures on the Diseases of the Nervous System. London, The New Syndeham Society, 1877, vol 72, pp 192–193.
2 Darley FL, Aronson AE, Brown JR: Differential diagnostic patterns of dysarthria. J Speech Hear Res 1969;12:246–269.
3 Darley FL, Aronson AE, Brown JR: Clusters of deviant speech dimensions in the dysarthrias. J Speech Hear Res 1969;12:462–496.
4 Brown JR, Darley FL, Aronson AE: Ataxic dysarthria. Int J Neurol 1970;7:302–318.
5 Pickett JM: The Sounds of Speech Communication. Baltimore, University Park Press, 1980.
6 Wang WSY: Linguistics and Phonetics (in Chinese). Taipei, Wen-Ho Press, 1988.
7 Kent R, Netsell R: A case study of an ataxic dysarthric: Cineradiographic and spectrographic observations. J Speech Hear Disord 1975;40:115–134.
8 Kent R, Netsell R, Abbs JH: Acoustic characteristics of dysarthria associated with cerebellar disease. J Speech Hear Res 1979;22:627–648.
9 Gilman S: Cerebellum and motor dysfunction; in Asbury AK, Mckhann GM, McDonald WI (eds): Disease of the Nervous System, ed 2. Philadelphia, Saunders, 1992, chapter 23, pp 319–341.
10 Victor M, Adams RD, Mancall EL: A restricted form of cerebellar cortical degeneration occurring in alcoholic patients. Arch Neurol 1959;1: 579–688.
11 Leigh RJ, Zee DS: The Neurology of Eye Movement, ed 2. Philadelphia, Davis, 1991, chapter 10, pp 378–536.
12 Mills CK, Weinsburg TH: Cerebellar symptoms and cerebellar localization. JAMA 1914; 63:1813–1818.
13 Lothman EW, Ferrendelli JA: Disorders and diseases of the cerebellum; in Rosenberg RN (ed): Neurology. New York, Grune & Stratton, 1980, vol 5, chapter 19, pp 547–587.
14 Lechtenberg R, Gilman S: Speech disorder in cerebellar disease. Ann Neurol 1978;3:285–290.
15 Amarenco P, Chevrie-Mullerr C, Roullet E, Bousser MG: Paravermal infarct and isolated cerebellar dysarthria. Ann Neurol 1991;30: 211–213.
16 Amarenco P, Roullet E, Goujon C, Chéron F, Hauw JJ, Bousser MG: Infarction in the anterior rostral cerebellum (the territory of the lateral branch of the superior cerebellar artery). Neurology 1991;41:253–258.
17 Lisker L, Abramson A: A cross-language study of voicing in initial stops: Acoustic measurements. Word 1964;20:384–422.
18 Barlow SM, Netsell R, Hunker CJ: Phonatory disorders associated with CNS lesions; in Cummings CW (ed): Otolaryngology-Head and Neck Surgery. St Louis, Mosby, 1986, vol 3, pp 2087–2093.
19 Gentil M: Dysarthria in Freidreich disease. Brain Lang 1990;38:438–448.
20 Sheard C, Adams RD, Davis PJ: Reliability and agreement of ratings of ataxic dysarthria speech samples with varying intelligibility. J Speech Hear Res 1991;34:285–293.
21 Kornhauser D, Bromberg MB, Gilman S: Effects of lesions of fastigial nucleus on static and dynamic responses of muscle spindle primary afferent in the cat. J Neurophysiol 1982;47: 977–986.
22 Ito M: Neurophysiological aspects of the cerebellar motor control system. Int J Neurol 1970; 7:162–176.
23 Gilman S: The mechanism of cerebellar hypotonia. Brain 1969;92:621–638.
24 Gilman S, Bloedel JR, Lechtenberg R: Disorders of the Cerebellum. Philadelphia, Davis, 1981.