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Cerebellum Structure Differences and Relationship to Speech in Boys and Girls With Nonsyndromic Cleft of the Lip and/or Palate

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Abstract

Objective—To identify regional cerebellar structural differences in boys and girls with nonsyndromic cleft of the lip and/or palate and determine whether these differences are related to speech impairment.

Design—Between 2003 and 2007, measures on cerebellar volume were obtained on 43 children with nonsyndromic cleft of the lip and/or palate and 43 age- and sex-matched, healthy controls. Children with the cleft condition also received speech evaluations. Children with nonsyndromic cleft of the lip and/or palate were recruited from clinic records, and controls (screened for medical, psychiatric, speech/language, and behavioral concerns) were recruited from the local community. All tests were administered at a large midwestern hospital. Boys and girls with nonsyndromic cleft of the lip and/or palate were compared with the healthy controls on global and regional measures of cerebellar volume. Areas of significant difference were then correlated with measures of speech to assess relationships in children with nonsyndromic cleft of the lip and/or palate.

Results—Boys with nonsyndromic cleft of the lip and/or palate had smaller cerebellums than controls (p = .002); whereas, for girls, only regional reductions in size reached significance (corpus medullare, p = .040). Cerebellum size was correlated with articulation for boys (p = .045).

Conclusions—These findings lend support to previous research documenting abnormal brain structure in children with nonsyndromic cleft of the lip and/or palate and suggest that the cerebellum may play a role in speech deficits along with other structural causes, at least in boys.

Keywords

cerebellum; children; cleft; speech

Orofacial clefts are the most common major birth defect in America, affecting more than 6800 births annually (Canfield et al., 2006). Types of oral clefts differ by location (i.e., lip, palate, or both; unilateral or bilateral) and extent (i.e., complete or incomplete; soft palate only or soft and hard palates; Burdi, 2006). Only 30% of cases of cleft are associated with a

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known genetic syndrome; the remaining 70% occur in isolation or without an identified genetic syndrome (Jones, 1988). These instances are often referred to as nonsyndromic cleft of the lip and/or palate (NSCL/P). In addition to the cleft, NSCL/P manifests as abnormalities in three areas: speech, cognition, and behavior (Riski, 2006). Here we focus on one of these domains—speech abnormalities.

The primary palatal closure surgery typically occurs prior to 18 months of age in most U.S. centers. Due to structural abnormalities (e.g., oral cavity and velopharyngeal insufficiency) and/or hearing loss (as a result of frequent otitis media), 20% to 30% of children will require secondary treatment to manage speech problems. Regular monitoring by speech and language pathologists is necessary to identify children at risk as soon as possible (Riski, 2006). Research has demonstrated that quality of speech in toddlers is correlated with lower cognitive abilities in addition to poorer hearing (Jocelyn et al., 1996; Broen et al., 1998; Baylis et al., 2008). However, research in the correlation of early speech—or even current speech—performance with cognitive abilities in middle childhood and adolescence is lacking. One recent study found that in children ages 7 to 17 years with NSCL/P, current speech performance was not correlated with verbal or performance IQ or to other measures of neuropsychological functioning (Conrad et al., 2009).

Speech difficulties commonly observed in children with cleft include those associated with velopharyngeal inadequacy (VPI) such as hypernasality, nasal emission, and compensatory articulation (Golding-Kushner, 2001). Treatment may include speech therapy, prosthetics (e.g., speech bulb or palatal lift), or surgery (e.g., pharyngeal flap or pharyngoplasty; Cleft Palate Foundation, 2006). Success rates for pharyngeal surgeries increase when done prior to 6 years of age (90.0% success rate compared with 73.9% for those done between 6 and 12 years of age) and the speech dysfunction is less severe (Riski, 2006).

In addition to abnormal oral structures, recent research has demonstrated abnormal brain development occurring concurrently with abnormal facial development. A series of studies on both children (Nopoulos et al., 2007) and adults (Nopoulos et al., 2002; Nopoulos et al., 2005) with NSCL/P report a pattern of abnormal brain structure in which the cerebrum is abnormally small early in development and more normal in volume by adulthood, but with tissue abnormally shifted anteriorly with larger volumes of the frontal and parietal lobes and smaller than normal volumes of the temporal and occipital lobes. One of the most robust structural changes seen in both children and adults with NSCL/P is an abnormally small cerebellum, and this difference remains strong after controlling for total brain volume (Nopoulos et al., 2002; Nopoulos et al., 2007).

Historically, the cerebellum has been noted for its role in motor coordination and balance (Holmes, 1939). Recently, research has emerged suggesting that due to the evolutionary parallel development of the frontal cortex and cerebellum, the cerebellum may share some responsibility in higher cognitive functions (e.g., speech, working memory, and executive functioning; Leiner et al., 1986). Functional and imaging studies in persons with cerebellum injury or lesions have documented deficits, including dysprosodia, agrammatism, mild anomia (Schmahmann and Sherman, 1998), verbal working memory deficits (Ben-Yehudah et al., 2007), and slowed speech pacing and articulation deficits (Ackermann et al., 2007). Atypical cerebellum development is not limited to people with clefts; abnormalities occur in conjunction with several other neurodevelopmental conditions. Further research in children with developmentally abnormal cerebellum structures (e.g., children with fragile X, Down, and Williams syndromes) have documented volumetric differences in the cerebellum associated with cognition, language, and speech. These deficits were more substantial in children with developmental differences than in those with acquired injuries to the cerebellum (Steinlin, 2007). The cerebellum's role in cognition and speech is hypothesized

to be one of temporal computations of motor and perceptual actions, including internal speech (Ackermann et al., 2007). Moreover, the superior/inferior posterior regions of the cerebellum have been attributed specifically to speech (Ackermann et al., 2007).

Given that children with NSCL/P have significant problems with speech (even after surgical correction) along with reports of structurally abnormal cerebellum in NSCL/P, it is natural to hypothesize that abnormal speech and abnormal cerebellum might be related in NSCL/P. The current study represents the first attempt to evaluate cerebellum morphology, speech, and their relationship in a sample of children with NSCL/P. In our previous study on brain morphology in children with NSCL/P, only global volumes of cerebellum were obtained (Nopoulos et al., 2007). Here, both global and regional morphology of the cerebellum are evaluated in a subset of NSCL/P children who also underwent an extensive and quantitative speech evaluation. There is no previous research evaluating speech and cerebellum structure in children with cleft, but based on our previous structural findings we hypothesized that abnormal structure of the cerebellum will be directly related to speech abnormalities. Specifically, lower volumes in the cerebellum would correlate with poorer performance on speech measures.

Methods

Procedure

This was a subsample of children who received a speech evaluation in addition to structural MRI (Nopoulos et al., 2007). Children with NSCL/P were recruited from clinic records, and those with a diagnosed genetic syndrome were excluded. Parents were sent a letter, inviting their child to participate. Those who were interested were screened for significant hearing loss (requiring a hearing aid), braces, and history of head trauma, brain tumor, or epilepsy. (Screening consisted of asking the parent if the aforementioned applied to his or her child and was conducted to ensure safety and quality of picture for the MRI scan.) The final group of children with NSCL/P consisted of 24 boys and 19 girls, ages 7 to 17 years. Of these, seven had nonsyndromic cleft lip only (NSCL), 11 had nonsyndromic cleft palate only (NSCP), and 25 had nonsyndromic cleft lip and palate (NSCLP). Although children with NSCL are not known for speech concerns due to oral structure, the focus of this study was on potential speech differences due to abnormal cerebellum development. Therefore, these seven individuals were included in analyses.

Through advertisements, 43 participants without cleft (matched by age and sex) were recruited from the community. These children were screened for learning, attention, speech/language, and health problems. Screening consisted of asking the parent if there was a history of the aforementioned in the child, and those with problems were excluded. Although this method has the potential risk of false negatives (i.e., a child with a disorder being included), it was the most efficient screening method for the number of control subjects recruited for the parent study (Nopoulos et al., 2007).

The average ages of the group with NSCL/P (13.27 ± 3.28 years) and controls (13.28 3.27 years) were not significantly different ($F_{1,84} = 0.0004$, $p = .983$). The majority of both groups, but more so for controls, were white (70% for NSCL/P and 86% for controls), consistent with demographics in the region. Social class was significantly higher for the control group (2.34 versus 2.59) based on ratings made by parents (Hollingshead and Redlich, 1958), where a lower number indicated higher socioeconomic status (SES; $F_{1,84} = 4.637$, $p = .034$). For this reason, SES was covaried in analyses. For detailed demographic data by cleft type, see Table 1.

This study was approved by the hospital's institutional review board; guardians signed consents and subjects signed assent documents to participate. After scheduling, subjects came to the hospital research clinic where parents completed a demographic questionnaire and children underwent the 35-minute MRI sequence. Children with cleft underwent a speech assessment, conducted by an experienced speech pathologist with extensive expertise in children with clefting (S.D.).

Measures

Speech Assessment—Speech and resonance assessments were provided by a certified speech pathologist with extensive experience evaluating children with speech disorders associated with VPI. Assessments included perceptually based and age-adjusted speech ratings of hypernasality and hyponasality on a 6-point scale (where 1 is "normal" and 6 is "severely abnormal") after each subject produced single words, sentence repetition, and spontaneous conversational speech. Previously reported (Dailey et al., 2006) interrater reliability was .96 for hypernasality and .91 for hyponasality. Perceptual ratings also were made on the same 6-point scale for articulation proficiency, intelligibility, and voice quality. Velopharyngeal function was rated as "competent," "marginal," or "incompetent." Presence of compensatory articulation and specific compensatory articulation errors were noted. Objective acoustic measurements of nasalance were obtained with a Nasometer II (model 6400; Kay-Pentax, Lincoln Park, NJ). Each subject was recorded while producing sentences loaded with pressure consonants excerpted from the Zoo Passage (Adams, 1988). Scores from this measure will be referred to in this report as nasalance. It is well established that children with clefts who have disorders of speech production are at highest risk for hypernasality and articulation errors (Peterson-Falzone et al., 2009). Therefore, for the purposes of this report, only perceptual judgments of hypernasality, articulation proficiency, and nasometric assessment of resonance were considered for analyses.

MRI

Imaging Methods: Images were obtained on a 1.5 Tesla GE Signa MR scanner (GE Medical Systems, Milwaukee, WI). Three different sequences were acquired for each subject. T1-weighted images, using a spoiled gradient recalled sequence, were acquired with the following parameters: 1.5-mm coronal slices, 40° flip angle, 24-millisecond repetition time (TR), 5-millisecond echo time (TE), two excitations (NEX), 26 cm field of view (FOV), and a 256 × 192 matrix. The proton density (PD) and T2-weighted images were acquired with the following parameters: 3.0-mm coronal slices, 36-millisecond TE (for PD) or 96-millisecond TE (for T2), 3000 millisecond TR, one NEX, 26-cm FOV, 256 × 192 matrix, and one echo train length.

Processing of the images after acquisition was done using a locally developed family of software programs called Brain Research: Analysis of Images, Networks, and Systems (BRAINS). Details of the image analysis are published elsewhere (Andreasen et al., 1992; Cohen et al., 1992; Andreasen et al., 1993; Andreasen et al., 1994; Magnotta et al., 2002). In brief, a three-dimensional data set is created (using all three sequences) and the images are realigned, resampled, and transformed into Talairach Atlas space (Talairach and Tournoux, 1988).

Brain Volume Measures: Within the stereotactic space, boxes were assigned to specific brain regions. Intracranial volume was subdivided into total brain tissue and cerebral spinal fluid. Brain tissue was subdivided into the cerebrum and cerebellum. The cerebellum was then subdivided into four main areas as described by Pierson and colleagues (2002). The anterior lobe (anatomical regions I, II, III, IV, and V) was separated from the superior posterior lobe (VI and Crus I of VIIA) by the primary fissure. The superior posterior lobe

was separated from the inferior posterior lobe (Crus II of VIIA, VIIB, VIII, IX, and X) by the horizontal fissure. The central white matter and output nuclei were included as part of the corpus medullare. These regions of the cerebellum were parcellated semiautomatically in BRAINS2 and corrected by hand by a trained tracer. Tracers were trained on five scans and tested on an independent sample of 10 scans. Regional volumes were compared and tracers were required to reach more than .90 intraclass correlation before tracing the current sample. Tracers averaged r = .93 (range, .90 to .98).

Analysis—All analyses were conducted with Statistical Package for the Social Sciences (SPSS), version 15.0 for Windows (SPSS Inc., Chicago, IL). Because there are brain structure differences between sexes, analyses were performed separately. Although this decision reduced the power of the analyses, it ensured that gender differences in structure/function relationships would not be masked by a global analysis. Structural comparisons between children with NSCL/P and controls began with a comparison of total cerebellar volume and then smaller, regional volumes were examined. An analysis of covariance (ANCOVA) was performed with patient type as the independent variable and cerebellar volume as the dependent variable. Intracranial volume (ICV) and SES were included as covariates in the model. For boys, Levene's test of equality of error variances indicated heterogeneity of variance ($F_{1,46}$ = 7.201, p = .010). A Brown-Forsythe robust test of equality of means was conducted with cerebellar volume/ICV as the dependent variable (this ratio was created to control for ICV in the analysis). Next, a multivariate analysis of covariance (MANCOVA) was examined with patient type as the independent variable and each region (i.e., anterior lobe, corpus medullare, inferior posterior lobe, and superior posterior lobe volumes) as the dependent variable. Cerebellar volume and SES were covaried in the model, providing an analysis that evaluated the morphology of each region in proportion to overall cerebellum size rather than in proportion to the brain.

The next analysis compared children who had received a corrective surgery (i.e., pharyngeal flap or z-plasty) to those who did not have a surgery. This evaluated the ability to merge the two groups for further analysis. Ten of the 24 boys with NSCL/P had surgery (seven had a pharyngeal flap and three had a z-plasty) and 1 of the 19 girls had surgery (a pharyngeal flap). Because so few girls had a history of surgery, only boys were used in the following analysis. There were no age ($F_{1,22}$ = 0.149, p = .703) or SES differences ($F_{1,22}$ = 0.013, p = .909) between boys with and without surgery. Cerebellar volume (controlling for ICV and SES) in boys with and without a history of surgery were compared with an ANCOVA.

Finally, correlations were run to determine the relationship between cerebellum structure and speech function. Correlations were run separately for each sex on speech measures and brain volume, limiting the analysis to only those regions where significant differences between groups were found. The limited comparisons were used to reduce chances for type I error. Because speech measures were skewed in the direction of good speech, a Spearman correlation was chosen.

Results

Speech results on the three measures (i.e., hypernasality, articulation proficiency, and nasalance) were skewed toward limited speech impairment (see Table 2). The majority of children with cleft (67% to 79%) were in the average range for all measures. A *post hoc* MANOVA with Bonferroni correction showed significant differences between boys and girls ($F_{3,38}$ = 2.845, p = .05). In general, boys had more impaired speech than girls had in all three domains. These differences reached significance only for the hypernasality rating (Brown-Forsythe; $F_{1,32.118}$ = 10.725, p = .003).

Means and standard deviations for cerebellar and regional volumes are presented in Table 3. The ANCOVA of cerebellar volume, controlling for ICV and SES, showed significant differences in boys (Brown-Forsythe; $F_{1,34.294} = 11.752$, $p = .002$). Boys with NSCL/P had significantly smaller cerebellar volumes than controls had. Differences for girls were nonsignificant ($F_{1,34} = 3.808$, $p = .059$).

The MANCOVA for cerebellar regional volumes, with Bonferroni correction and controlling for global cerebellar volume and SES, was nonsignificant for differences in boys ($F_{4,41} = 0.589$, $p = .672$), but significant for girls ($F_{4,31} = 3.458$, $p = .019$). This indicates that the overall decrement in cerebellar volume in boys does not seem to be regionally specific, but in girls, the structural abnormality may be more confined to certain regions. For girls with NSCL/P only the corpus medullare ($F_{1,34} = 5.043$, $p = .031$) was proportionately smaller than that of controls.

The ANCOVA assessing cerebellar volume differences between boys who received corrective surgery for speech concerns and those who did not was not significant ($F_{1,20} = 2.924$, $p = .103$). This finding indicates there were no significant structural differences between these groups based on surgery status and supports the merging of both groups in the subsequent analyses (i.e., comparing structure and speech function relationships).

Speech and structure correlations for boys were significant for cerebellar volume and articulation proficiency ratings (Spearman $\rho = -.490$, $p = .015$). Boys who were rated with worse articulation had lower cerebellar volume. This finding remained significant after a Bonferroni correction for multiple comparisons ($p = .045$). Speech and structure correlations for girls were nonsignificant after a Bonferroni correction (see Table 4).

A *post hoc* correlation was run on the three speech measures and total intracranial volume to rule out the possibility of deficits correlating to global brain differences. Spearman correlations were nonsignificant for hypernasality (Spearman $p = .126$, $p = .559$), articulation proficiency (Spearman $p = -.242$, $p = .254$), and nasalance (Spearman $p = -.027$, $p = .889$).

Discussion

Similar to a previous study that used a larger sample (Nopoulos et al., 2007), boys with NSCL/P had smaller cerebellar volumes than controls, even after accounting for ICV and SES. The subregions of the corpus medullare and superior posterior volume were proportionately smaller. These findings lend further support to the hypothesis of differential brain structure in children with NSCL/P.

To assess the potential confound of surgery, subjects with NSCL/P were grouped based on surgery status. It was hypothesized that those with more severe speech problems (requiring surgery) might show more difference in cerebellar volume. However, no differences were found.

In the final analyses of the relationship between brain region volumes and speech performance, one significant correlation was found: For boys, the decrement in overall volume of the cerebellum was related directly to worse articulation proficiency.

The *post hoc* correlation run to rule out the possibility of deficits correlating with global brain differences demonstrated no relationship between speech ratings and total ICV. This further supports the specificity of relationship between the cerebellum and speech. It is possible that other variables not measured in this study, related to both cerebellum and speech production, could be the cause of this relationship. Although these associations do

not provide evidence that abnormal cerebellum structure causes the speech deficit, these findings are the first ever to report this relationship in the NSCL/P population. Even though one significant finding can not be generalized as supportive to the hypothesis of a cerebellar role in speech production, this study establishes the need for further research.

The brain and face grow together, originating from the same embryonic cells and migrating to form their respective selves at the same developmental stage (Burdi, 2006). If the migration process of facial development is interrupted (resulting in cleft lip and/or palate), it is possible that similar mechanisms may cause abnormal migration and development of the brain. Several studies in NSCL/P have reported relationships between abnormal brain structure and function. The enlarged anterior and decreased posterior cerebrum of adult men with NSCL/P was found to be pathological, correlating to lower Full Scale IQ and Verbal IQ scores (Nopoulos et al., 2002). Abnormal temporal lobe morphology was associated with performance on language tests (Shriver et al., 2006). Finally, in an evaluation of social function and regional brain volume measures, lower levels of social functioning in men with NSCL/P were correlated with structural abnormalities of the ventral frontal region of the brain (Nopoulos et al., 2005). This same relationship was replicated in a sample of boys with NSCL/P (Boes et al., 2007). Therefore, the current study is another piece of evidence to support the notion that structural abnormalities in the brain of subjects with NSCL/P are associated directly with the three domains of functional deficit in this population: speech, cognition, and behavior. Although abnormal oral structure and early hearing problems are obvious main causes for potential speech problems, abnormal brain development also may play some role in speech concerns.

There are several limitations of the current study that should be addressed. The screening process for control subjects was brief, and some children with a learning/attention, speech, or health concern may have been included. Also, the lack of variability in the speech measures may have decreased the power to detect correlations. The overwhelming majority of subjects were rated at a 1 or 2 for hypernasality and articulation proficiency, and 71% were within the normal range for high oral pressure. Our imaging methods did not quantify the vermis (due to low reliability of vermis measures in the current protocol). Some differences within the vermis, a subregion of the cerebellum found to be abnormal in other developmental syndromes such as autism (Stanfield et al., 2008), may be overlooked due to this. Finally, cerebellum influence on speech may be related to different aspects of speech not assessed in this study. Perceptual ratings of nasality and articulation proficiency and acoustic measures of nasalance may not reflect the potential temporal role of the cerebellum.

There is compelling evidence of concurrent abnormal brain and facial development and the possibility of brain abnormalities playing some role in deficit patterns seen in children and adults with NSCL/P. Future research is needed to determine what this role may be. Studies should include larger number of children with a greater variety of speech levels. More detailed measures of speech pacing and articulation, verbal working memory, and other fine-motor functions (e.g., hand dexterity) will be needed. With further research, it will be possible to better understand the role of the cerebellum in higher cognitive functions. Also, it will be important to determine how much speech deficits are due to abnormal oral structure and function in children with cleft and how much of a role the brain may have.

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TABLE 1

Demographic Measures

	Control (n = 43)		NSCL/P (n = 43)		NSCL (n = 7)		NSCP (n = 11)		NSCLP (n = 25)	
	M	**SD**	**M**	**SD**	**M**	**SD**	**M**	**SD**	**M**	**SD**
Age	13.28	3.27	13.27	3.28	12.86	3.52	12.56	3.44	13.69	3.21
SES	2.33	0.56	2.86	0.69	2.86	0.69	2.59	0.49	2.52	0.59
	n (%)		*n (%)*		*n (%)*		*n (%)*		*n (%)*	
Gender										
Boys	24 (59)		24 (59)		4 (57)		4 (36)		16 (64)	
Girls	19 (41)		19 (41)		3 (43)		7 (64)		9 (36)	
Ethnicity										
White	37 (86)		30 (70)		5 (72)		11 (100)		14 (56)	
Asian American	—		4 (9)		—		—		4 (16)	
Hispanic	—		2 (5)		—		—		2 (8)	
Multiracial	1 (2)		3 (7)		1 (14)		—		2 (8)	
Unknown	5 (12)		4 (9)		1 (14)		—		3 (12)	

* NSCL/P = nonsyndromic cleft of lip and/or palate; NCSL = nonsyndromic cleft lip only; NSCP = nonsyndromic cleft palate only; NSCLP = nonsyndromic cleft of lip and palate; SES = socioeconomic status.

TABLE 2

Distribution of Scores for Speech Assessment in Children With NSCL/P*

	Boys		Girls		Total	
Measure	**% Normal**	**Mean (SD)**	**% Normal**	**Mean (SD)**	**% Normal**	**Mean (SD)**
Hypernasality†‡	50	1.67 (0.76)	90	1.11 (0.32)	67	1.42 (0.66)
Artic Prof‡	75	1.38 (0.77)	84	1.16 (0.37)	79	1.28 (0.63)
Nasalance§	58	0.26 (0.15)	89	0.19 (0.12)	71	0.23 (0.14)

*NSCL/P = nonsyndromic cleft lip and/or palate; Artic Prof = articulation proficiency.

†n = 43. Boys rated significantly higher than girls at $p = .003$.

‡n = 43. Scores are based on a scale of 1 to 6, with 1 indicating “normal” and 6 “severe.”

§n = 42. Nasalance measures >0.30 consistent with hypernasal resonance.

TABLE 3

Means, Standard Deviations, and Ratios of Cerebellar and Regional Volume Measures*

	Boys						
	NSCL/P†		Control				
Measure	Mean (SD)	Ratio	Mean (SD)	Ratio	F	p	η_p^2
Cerebellum	127.54 (13.06)	.09	143.25 (10.23)	.10	11.752	.002	—
AL	15.34 (2.55)	.12	16.06 (2.44)	.11	0.877	.354	.020
CM	12.52 (2.03)	.10	14.15 (1.32)	.10	0.351	.557	.008
IPL	56.43 (7.48)	.44	63.27 (5.99)	.44	0.488	.489	.011
SPL	43.94 (5.53)	.35	50.53 (5.26)	.35	0.378	.247	.030

	Girls						
	NSCL/P		Control				
Measure	Mean (SD)	Ratio	Mean (SD)	Ratio	F	p	η_p^2
Cerebellum	118.95 (8.92)	.09	128.69 (9.62)	.09	3.808	.059	.101
AL	14.80 (2.91)	.13	13.82 (2.18)	.11	1.052	.312	.030
CM	11.61 (1.53)	.10	13.73 (1.75)	.11	5.043	.031	.129
IPL	52.99 (6.21)	.45	55.31 (3.77)	.43	1.253	.271	.036
SPL	41.84 (5.16)	.35	47.65 (4.01)	.37	3.382	.075	.090

*Volumetric measures are presented in cubic centimeters; this unit of measurement was used in analyses. Ratio values are total cerebellum/total intracranial volume and regional volume/total cerebellum.

†NSCL/P = nonsyndromic cleft lip and/or palate; AL = anterior lobe; CM = corpus medullare; IPL = inferior posterior lobe; SPL = superior posterior lobe, η_p^2 = partial eta squared.

TABLE 4

Relationship of Cerebellar Volumes and Speech Performance in Children With NSCL/P

	Boys (n = 24)		Girls (n = 19)	
	Cerebellum		Corpus Medullare	
Measure	**Spearman p**	**p**	**Spearman p**	**p**
Hypernasality	.301	0.459	.063	4.794
Artic Prof	−.490	0.045	.211	2.316
Nasalance	.348	0.285	−.239	2.040

* NSCL/P = nonsyndromic cleft lip and/or palate; Artic Prof = articulation proficiency. *p* values are after Bonferroni correction.