

Utility of the Delta/Alpha Ratio as a Neurophysiologic Marker for Children with Posterior Fossa Syndrome: A Blinded Controlled Pilot

Sharyl Samargia-Grivette

samargia@d.umn.edu

University of Minnesota, Duluth

Anne Bendel

Children's Minnesota Cancer and Blood Disorders Clinic

Maggie Skrypek

Children's Minnesota Cancer and Blood Disorders Clinic

Emma Olberg

University of Minnesota, Duluth

Caroline Dahlke

University of Minnesota, Duluth

Yu Liu

University of Minnesota Medical School

Callie Showalter

University of Minnesota, Duluth

Amanda Jackson

University of Minnesota, Duluth

Detlef H. Heck

University of Minnesota Medical School

Samantha Ertz

University of Minnesota, Duluth

Anna Schmidt

University of Minnesota, Duluth

Research Article

Keywords: Posterior fossa syndrome, posterior fossa tumor, electroencephalography, delta alpha ratio, ataxia

Posted Date: September 4th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10779195/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.
[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Purpose

To identify a potential neurophysiologic marker for posterior fossa syndrome (PFS) following resection of posterior fossa tumors in children. Methods: Spectral power was obtained across all frequency bands using resting state electroencephalography (EEG). This analysis focused on the delta/alpha ratio (DAR) from the left and right frontal cortical regions which was compared across groups of children who have had a posterior fossa tumor resected and 1) acquired PFS, 2) did not acquire PFS and 3) healthy, age-matched peers. Symptoms and severity of ataxia across gross and fine motor skills using the Scale for Assessment and Rating of Ataxia (SARA) and motor speech skills using a perceptual speech assessment were completed. Results: DAR values were consistently higher in the PFS group compared to the non-PFS and healthy controls with differences at F3, F4, F8 and Fp2 reaching statistical significance. The PFS group demonstrated a higher mean severity rating on the SARA and perceptual speech assessment. A positive correlation between DAR and 1) total SARA scores, 2) speech rate, 3) speech prosody and 4) overall speech severity was found at F3 and F7. Conclusion: DAR holds promise as a neurophysiologic marker for PFS following posterior fossa tumor resection. Increased DAR may be reflective of the integrity of the dento-thalamo-cortical tract and the presence of cross cerebellar-cortical diaschisis. Findings from this study lay the foundation for a larger trial to determine reliability and validity of DAR as a neurophysiologic marker for PFS.

Introduction

Posterior fossa syndrome (PFS) is an acquired brain injury occurring 8–31% of children who undergo resection of a posterior fossa tumor resulting in a devastating sequela of motor, neurobehavioral and linguistic deficits [1], [2], [3], [4]. Symptoms include emotional lability, dysmetria, ataxia, as well as cognitive and behavioral changes. However, delayed onset and transient mutism is the primary feature of PFS. Although recovery of many symptoms of PFS occurs, it is rarely complete, resulting in significant long-term communication, cognitive and motor impairments for many individuals [5], [6], [7], [8], [9], [10]. The diagnosis of PFS is perceptual, and currently, there is no one gold standard scoring tool used across all centers. Neurophysiologic measures such as EEG provide an opportunity to quantify neurologic changes due to this surgically-induced brain injury and hold potential as a neurophysiologic marker of PFS to improve accuracy of diagnosis and use as a tool for documenting the trajectory of recovery.

Damage to the efferent cerebellar cortical network has been implicated in the motor and cognitive impairments observed in children with PFS possibly due to cross cortical diaschisis in the frontal cortex [11], [12]. Electroencephalography has high temporal resolution and offers an affordable, non-invasive way to assess cortical activity. Quantitative EEG (qEEG), specifically spectral power, reflects synchronized oscillatory activity within and between cortical areas and has been deemed reliable in measuring the pathophysiology of acute ischemic brain injury [13], [14]. Differences in spectral power, specifically increases in slow-wave delta bands and decreases in higher-wave alpha bands appear to be

reflective of cortical impairment [15] and have been associated with decreased oxygen (ischemia) [16], cerebral blood flow [17], brain metabolism [18] and perfusion [19] potentially reflecting the penumbra region and/or regions of diaschisis [20]. The delta/alpha ratio (DAR), the relationship between slow-wave (delta) and faster-wave (alpha) oscillations, has recently been gaining attention. A higher proportion of spectral power in alpha versus delta, reflected in a DAR of ≤ 1.0 during an eyes open, resting state is expected in healthy adults [21] and children [22]. DAR has been used as a pathophysiologic marker for cerebral ischemia, acute [23] and delayed [24]. While not deemed sensitive for diagnostic purposes in mild traumatic brain injury [25], it appears to hold value as a predictor of rehabilitation potential [21], [23], [26], [27], [28].

To date, one report, a single case of a child with PFS, recorded resting state EEG over time and found initial delta band power to decrease then increase over time and theta, gamma and beta bands initially increased then decreased over time [29]. The purpose of this study was to compare DAR in the left and right frontal regions (frontopolar, frontal and anterior temporal) of the cortex, between individuals who had a history of posterior fossa tumor resection and 1) did acquire PFS, 2) did not acquire PFS to 3) healthy controls. We hypothesized the PFS group would demonstrate higher DAR values across all electrode sites. We also hypothesized DAR in the left electrode sites would be significantly higher than the right electrode sites in PFS because of the increased incidence of PFS in those with right sided tumors and damage to left cerebellar peduncle. Finally, we hypothesized there would be a significant relationship between DAR and ataxia severity scores.

Materials and methods

Participants

Individuals ages 10 years 0 months to 25 years 11 months who underwent resection for a posterior fossa tumor were recruited from a pediatric neuro-oncology center database. Inclusion criteria for PFS and non-PFS groups: Resection of posterior fossa tumor > 2 years prior to study participation, diagnosis of PFS (PFS group) or did not acquire PFS (Non-PFS group), as determined by the neuro-oncology team at the partnering institution, and > 3 months post chemotherapy and radiation treatment. Exclusion criteria for PFS and non-PFS groups: diagnosis of any developmental or genetic conditions prior to brain tumor diagnosis and history of stroke(s) or seizure(s). Exclusion criteria for healthy control group: diagnosis of developmental, genetic or neurologic conditions. The target sample size for this study was 24, 8 participants in each group. A total of 17 individuals post tumor resection were identified as potential participants of whom, 3 did not respond when contacted. Fourteen were screened, of whom 1 was deemed ineligible due to seizures and 2 were unable to enroll due to geographic location. Eleven participants with a history of posterior fossa tumor were enrolled and completed the study (PFS, $n = 6$ and non-PFS, $n = 5$). Age and gender match peer controls for the PFS group were recruited. Participants were assigned to one of three groups: PFS ($n = 6$, mean age = 17.32 years $\pm$ 6.12), non-PFS ($n = 5$, mean age = 14.38 years $\pm$ 3.84) and age and gender matched controls for the PFS group ($n = 6$, mean age = 17.15 years $\pm$ 6.60).

EEG Procedure

Each participant was seated comfortably in a chair while a 128-channel EEG saline sensor net (Hydrocel GCN 130, EGI; Eugene, OR) was placed. Minimal impedance was assured through manual repositioning and nesting of the electrodes and use of NetStation impedance testing. Resting state EEG was collected for 6 minutes during eyes open condition which required the participant to look at a black plus sign against a white background on a computer screen in a dimly lit room. Participants were instructed to sit as still and quietly as possible. The NetAmps 400 system (Magstim/EGI; Eugene, OR) was used and data acquisition at a sampling rate of 10,000 using NetStation 5.4.2 (Magstim/EGI; Eugene, OR). The EEG suite used is housed in a Faraday cage and was designed specifically for EEG acquisition.

Behavioral Ataxia Assessment Procedure

Following EEG, participants in the PFS and Non-PFS groups completed behavioral tasks including the Scale for the Assessment and Rating of Ataxia (SARA) and a recorded speech sample of connected speech for perceptual analysis. Due to the lack of acquired motor speech assessments created for pediatric populations, researchers created a visual analog scale of 100cm for each feature of motor speech: overall, resonance, respiratory, prosody, articulation, rate, fluency for perceptual analysis of dysarthria symptoms. Behavioral assessments were video recorded for later analysis. Three members of the research team blinded to group assignment of the participants then analyzed the video recordings and independently scored the SARA and the perceptual speech assessment. No large discrepancies were found in the scores across raters and the inter-rater reliability was high ($r = 0.80$). Scores across raters were then averaged and used in the analysis.

EEG Signals and Power Spectrum Analysis:

Following acquisition, raw EEG data were preprocessed in EEGLAB (v2024.0), including artifact rejection, channel labeling, and segmentation. Subsequent analyses were performed on preprocessed signals (200 s per recording) using custom scripts in MATLAB (R2024b).

Power spectral density (PSD) was estimated using Welch's method (sampling frequency: 500 Hz; window length: 1 s; 60% overlap; FFT length: 1 s). The resulting spectra were integrated into canonical frequency bands: delta (1–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), and beta (12–30 Hz). The gamma band was subdivided into low gamma (30–55 Hz) and high gamma (65–90 Hz). The 55–65 Hz range was excluded to minimize contamination from 60 Hz line noise, and frequencies above 90 Hz were excluded to reduce potential muscle artifact contamination. Band-limited PSD values were computed for each channel and subject. For interhemispheric analyses, homologous left–right electrode pairs were defined a priori to enable pairwise comparisons of spectral power across hemispheres.

Delta–Alpha Ratio (DAR) Computation:

The delta–alpha ratio (DAR) was computed for each subject and channel as the ratio of delta-band to alpha-band power derived from the PSD. To preserve physiological interpretability, band-limited powers expressed in decibels (dB) were converted back to linear scale prior to ratio calculation:

$$\text{DAR} = \frac{P_{\delta}}{P_{\alpha}}$$

DAR values were first calculated at the single-channel level for all electrodes. Region-level DAR was then obtained by averaging across predefined homologous channel pairs, yielding channel-pair–averaged measures for frontopolar (Fp1-Fp2), frontal (F3-F4), and anterior temporal (F7-F8) regions. For visualization, DAR distributions were summarized using box plots with overlaid individual subject values. Statistical analyses were conducted on subject-level DAR values without group averaging. Group differences were assessed using Kruskal–Wallis tests followed by Dunn–Šidák-corrected post hoc comparisons.

Results

EEG data from six participants were removed from the analysis. Despite little to no impedance during recording, 200s of clean data was not identified for these participants reducing the sample size to 11; PFS (n = 4, mean age 16.71 years, SD 5.94; range 10.92-23); non-PFS (n = 3, mean age 14.30 years, SD 4.16; range 11.08-19) and Healthy Control (n = 4, mean age 17.77 years, SD 6.31; range 12.33–24.67). The duration since surgery for the PFS group was 7.5 years $\pm$ 3.69 and the non-PFS group was 5.66 years $\pm$ 1.15. Of those enrolled who underwent posterior fossa tumor resection, four were diagnosed with a medulloblastoma (4 in the PFS group and 2 in the non-PFS group) and one was diagnosed with ependymoma in the non-PFS group (Table 1).

Table 1. Demographic characteristics of enrolled participants used in the final analysis across three groups, those who had a posterior fossa tumor resection and did acquire posterior fossa syndrome (PFS), did not acquire PFS (Non-PFS) and healthy controls.

		PFS (n = 4)	Non-PFS (n = 3)	Control (n = 4)
Gender				
	Male	n = 2	n = 3	n = 2
	Female	n = 2	n = 0	n = 2
Age (year)				
	n	4	3	4
	Mean ± Std Dev	16.71 ± 5.94	14.30 ± 4.16	17.77 ± 4.16
	Range (min-max)	10.92-23	11.0–19.0	12.33–24.67
	Median	16.46	12.83	17.04
Age at surgery (year)				
	n	4	3	4
	Mean ± Std Dev	9.91 ± 3.59	8.19 ± 3.30	n/a
	Range (min-max)	1.92-14.0	3.0–12.0	n/a
	Median	10.21	6.58	n/a
Duration since surgery (year)				
	n	4	3	n/a
	Mean ± Std Dev	7.5 ± 3.69	5.66 ± 1.15	n/a
	Range (min-max)	3.0–11.0	5.0–7.0	n/a
	Median	8.0	5.0	n/a
Tumor Type				
	Medulloblastoma	4 (100%)	2(66%)	n/a
	Ependymoma	0 (0%)	1 (33%)	n/a

Delta/Alpha Ratio (DAR)

Mean, median and range of DAR at each electrode site of interest (Fp1, F3, F7, Fp2, F4 and F8) were calculated for each group. Median differences can be seen in Fig. 1.

All statistical analyses were performed on subject-level metrics. Because data distributions were non-Gaussian, nonparametric methods were used throughout. Group differences were assessed using Kruskal–Wallis tests for each channel or channel pair and frequency band. When significant main effects were detected, post hoc pairwise comparisons were conducted using Dunn–Šidák correction for multiple comparisons and. Statistical significance was set at $\alpha = 0.05$. For visualization, results were presented using box plots with overlaid individual subject values. Significant pairwise differences were annotated directly on figures using standard notation (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$) (Table 2).

DAR: Left side: Elevated DAR in the left electrode sites in the PFS group compared to non-PFS and healthy control was found. DAR values at two out of the three left frontal electrode sites were statistically significantly different. Fp1, $H(2) = 3.39$, $p = 0.18$, $\eta^2 = 0.17$; F3, $H(2) = 7.85$, $p = 0.02$, $\eta^2 = 0.73$. DAR at F3 was significantly higher in PFS than non-PFS ($p = 0.007$) and healthy control ($p = 0.05$), while non-PFS and healthy control did not differ ($p = 0.36$). F7, $H(2) = 5.30$, $p = 0.07$, $\eta^2 = 0.16$. DAR at F7 was significantly higher in PFS than non-PFS ($p = 0.02$), but not healthy control ($p = 0.28$). Non-PFS was not significantly different than healthy control ($p = 0.18$). (Table 2 Fig. 2a-c).

DAR: Right side: DAR analysis revealed consistent elevated DAR in the right electrode sites in the PFS group compared to non-PFS and healthy control. Fp2: $H(2) = 7.21$, $p = 0.03$, $\eta^2 = 0.40$. DAR at Fp2 was significantly higher in PFS than non-PFS ($p = 0.015$) and healthy control ($p = 0.03$), but not between non-PFS and healthy control ($p = 0.64$). F4: $H(2) = 7.84$, $p = 0.02$, $\eta^2 = 0.48$. DAR at F4 was significantly higher in PFS than in non-PFS ($p = 0.007$) and healthy control ($p = 0.05$), but no significant difference in DAR was found between non-PFS and healthy control ($p = 0.36$). And, at F8: $H(2) = 7.47$, $p = 0.02$, $\eta^2 = 0.43$. DAR at F8 was significantly higher in PFS than non-PFS ($p = 0.010$) and healthy control ($p = 0.04$), but no significant difference in DAR was found between non-PFS and healthy control ($p = 0.49$) (Table 2 and Fig. 2d-f).

Table 2. Descriptive statistics for DAR across electrode sites for each group								
			PFS	Non-PFS	Control	P-value	Effect Size (η^2)	
Left								
	Fp1	Mean $\pm$ SD	1.06 $\pm$ 0.22	0.80 $\pm$ 0.12	0.90 $\pm$ 0.17	$p = 0.18$	$\eta^2=0.17$	
		Median (min-max)	0.96 (0.94–1.40)	0.82 (0.67–0.92)	0.95 (0.67–1.06)			
	F3	Mean $\pm$ SD	1.06 $\pm$ 0.04	0.40 $\pm$ 0.06	0.70 $\pm$ 0.20	$p < 0.02^*$	$\eta^2=0.73$	
		Median (min-max)	1.05 (1.0-1.11)	0.42 (0.33–0.44)	0.77 (0.42–0.84)			
	F7	Mean $\pm$ SD	1.2 $\pm$ 0.04	0.54 $\pm$ 0.29	0.90 $\pm$ 0.39	$p = 0.07$	$\eta^2=0.16$	
		Median (min-max)	1.20 (1.16–1.26)	0.44 (0.32–0.88)	0.93 (0.39–1.33)			
Right								
	Fp2	Mean $\pm$ SD	1.30 $\pm$ 0.03	0.83 $\pm$ 0.08	0.66 $\pm$ 0.16	$p = 0.03^*$	$\eta^2=0.40$	
		Median (min-max)	1.29 (1.25–1.32)	0.87 (0.74–0.89)	0.71 (0.44–0.81)			
	F4	Mean $\pm$ SD	1.06 $\pm$ 0.06	0.42 $\pm$ 0.08	0.66 $\pm$ 0.16	$p = 0.02^*$	$\eta^2=0.48$	
		Median (min-max)	1.03 (1.03–1.15)	0.46 (0.33–0.48)	0.71 (0.44–0.81)			
	Kruskall–Wallis test with significance set at $p=0.05$							
	Pairwise comparisons using Dunn–Šidák							
Epsilon squared: 0.01=small effect; 0.08= medium effect; ≥ 0.26 = large effect								

Table 2. Descriptive statistics for DAR across electrode sites for each group							
		PFS	Non-PFS	Control	P-value	Effect Size (η^2)	
F8	Mean $\pm$ SD	1.22 $\pm$ 0.05	0.61 $\pm$ 0.15	0.73 $\pm$ 0.09	$p = 0.02^*$	$\eta^2=0.43$	
	Median (min-max)	1.22 (1.15–1.27)	0.62 (0.45–0.75)	0.74 (0.61–0.82)			
Kruskall–Wallis test with significance set at $p=0.05$							
Pairwise comparisons using Dunn–Šidák							
Epsilon squared: 0.01=small effect; 0.08= medium effect; ≥ 0.26 = large effect							

Behavioral Ataxia Analysis

Scale of the Assessment and Rating of Ataxia (SARA)

Higher scores on the SARA, indicating more severe symptoms, were found in the PFS group compared to the non-PFS group (Table 3). Areas of highest severity in the PFS group included balance, gait and kinetic scores. Only two domains were statistically significantly different between the groups, right alternating hand ($p = 0.03$) and total alternating hand ($p = 0.04$). However, it should be noted, the non-PFS group had a mean of 0 for both of these domains. Although the PFS group had consistently higher mean scores on the SARA, reflecting more severe symptoms of ataxia than the non-PFS group, the difference did not reach statistical significance. However, there was a large effect for both the kinetic subtest ($\eta_p^2=0.33$) and the total SARA score ($\eta_p^2=0.23$) (Table 3).

Table 3

Descriptive statistics for the Scale of the Assessment and Rating of Ataxia (SARA) across the PFS and Non-PFS groups.

	PFS (n = 6)	Non-PFS (n = 5)	Multivariate Analysis	Effect Size (η_p^2)
Gait	M = 3.0, SD = 2.44	M = 1.66, SD = 2.08	$p = 0.48$	$\eta_p^2=0.10$
Balance	M = 4.5, SD = 4.35	M = 2.3, SD = 2.30	$p = 0.47$	$\eta_p^2=0.10$
Kinetic	M = 5.0, SD = 4.02	M = 1.16, SD = 0.76	$p = 0.17$	$\eta_p^2=0.33^{**}$
TOTAL (40)	M = 9.5, SD = 8.25	M = 3.5, SD = 1.80	$p = 0.28$	$\eta_p^2=0.23^{**}$
<i>R=Right side, L=Left side and T=Total</i>				

Multivariate Analysis of Variance (MANOVA) with significance level set at 0.05*.

Partial eta squared: 0.1, small effect; 0.6, medium effect; 0.14, large effect**.

Perceptual Speech Assessment

While those in the PFS group demonstrated a greater extent of perceptual deficits in respiratory, resonance, rate, prosody, pitch, loudness, voice and overall speech, results from a multivariate analysis of variance (MANOVA) revealed no significant differences between PFS and non-PFS groups. However, a large effect size was detected for rate ($\eta_p^2=0.24$), prosody ($\eta_p^2=0.21$) and overall speech ($\eta_p^2=0.30$). Rate and prosody were rated as most perceptually detectible impairments. No perceptual impairments were identified in the areas of articulation and fluency in the PFS group (Table 4).

Table 4
Descriptive statistics for the perceptual speech assessment across the PFS and Non-PFS groups.

	PFS	Non-PFS	Multivariate Analysis	Effect Size (η_p^2)
Respiratory	M = 0.75, SD = 1.5	M = 0.0	$p = 0.43$	$\eta_p^2=0.12$
Resonance	M = 1.75, SD = 3.5	M = 0.0	$p = 0.43$	$\eta_p^2=0.12$
Articulation	M = 0.0	M = 0.0	n/a	n/a
Rate	M = 12.75, SD = 17.36	M = 0.0	$p = 0.26$	$\eta_p^2=0.24^{**}$
Prosody	M = 11.0, SD = 16.04	M = 0.0	$p = 0.29$	$\eta_p^2=0.21^{**}$
Pitch	M = 1.25, SD = 2.5	M = 0.0	$p = 0.43$	$\eta_p^2=0.12$
Loudness	M = 1.25, SD = 2.5	M = 0.0	$p = 0.43$	$\eta_p^2=0.12$
Fluency	M = 0.0	M = 0.0	n/a	n/a
Voice	M = 2.25, SD = 4.5	M = 0.0	$p = 0.43$	$\eta_p^2=0.12$
Overall	M = 3.75, SD = 4.34	M = 0.0	$p = 0.20$	$\eta_p^2=0.30^{**}$

Perceptual assessment utilized a 100mm visual analog scale to rate severity across speech domains.

Multivariate Analysis of Variance (MANOVA) with significance level set at 0.05*.

Partial eta squared: 0.1, small effect; 0.6, medium effect; 0.14, large effect**.

Correlation between DAR and Ataxia Severity

A Spearman's Rho correlation was used to determine the relationship between DAR values and ataxia severity, specifically rate, prosody and overall speech from the perceptual speech assessment and kinetic, balance, gait and total score on the SARA (Table 5). While DAR at F7 was strongly and significantly correlated with all ataxia severity across all areas, DAR at F3 and Fp2 were correlated with ataxia severity across four out of six areas. As DAR value increased, severity of ataxia symptoms increased.

Table 5

Correlation between delta alpha ratio (DAR) at each electrode and ataxia symptom severity in all participants with a history of posterior fossa tumor resection (n = 7)

		Speech Rate	Speech Prosody	Speech Overall	SARA Kinetic	SARA Balance	SARA Gait	SARA Total
DAR in Left	Fp1	$r_s = 0.27$ $p = 0.28$	$r_s = 0.27$ $p = 0.28$	$r_s = 0.35$ $p = 0.21$	$r_s = 0.40$ $p = 0.18$	$r_s = 0.17$ $p = 0.35$	$r_s = 0.24$ $p = 0.30$	$r_s = 0.31$ $p = 0.25$
	F3	$r_s = 0.75$ $p = 0.002^*$	$r_s = 0.75$ $p = 0.024^*$	$r_s = 0.80$ $p = 0.015^*$	$r_s = 0.84$ $p = 0.008^*$	$r_s = 0.51$ $p = 0.09$	$r_s = 0.61$ $p = 0.07$	$r_s = 0.67$ $p = 0.05^*$
	F7	$r_s = 0.80$ $p = 0.015^*$	$r_s = 0.80$ $p = 0.015^*$	$r_s = 0.75$ $p = 0.02^*$	$r_s = 0.77$ $p = 0.02^*$	$r_s = 0.71$ $p = 0.03^*$	$r_s = 0.76$ $p = 0.02^*$	$r_s = 0.77$ $p = 0.02^*$
DAR in Right	Fp2	$r_s = 0.75$ $p = 0.02^*$	$r_s = 0.75$ $p = 0.024^*$	$r_s = 0.80$ $p = 0.015^*$	$r_s = 0.81$ $p = 0.013^*$	$r_s = 0.47$ $p = 0.14$	$r_s = 0.45$ $p = 0.15$	$r_s = 0.45$ $p = 0.15$
	F4	$r_s = 0.58$ $p = 0.08$	$r_s = 0.58$ $p = 0.08$	$r_s = 0.65$ $p = 0.05^*$	$r_s = 0.66$ $p = 0.05^*$	$r_s = 0.48$ $p = 0.13$	$r_s = 0.56$ $p = 0.09$	$r_s = 0.63$ $p = 0.06$
	F8	$r_s = 0.13$ $p = 0.38$	$r_s = 0.13$ $p = 0.58$	$r_s = 0.18$ $p = 0.35$	$r_s = 0.45$ $p = 0.15$	$r_s = 0.05$ $p = 0.45$	$r_s = 0.13$ $p = 0.39$	$r_s = 0.13$ $p = 0.39$
Spearman's Rho Correlation: $*p < 0.05$								

Discussion

This pilot study aimed to compare DAR values at left and right frontal electrode sites between individuals who had a posterior fossa tumor resection and acquired PFS, did not acquire PFS and healthy controls. Results revealed the PFS group to have higher DAR values across all electrode sites compared to the other groups, some differences reaching statistical significance and strong effect sizes suggesting DAR may be a useful measure for differential diagnosis of PFS.

We hypothesized the PFS group would demonstrate higher DAR values across all frontal electrode sites which was upheld with statistically significantly higher DAR present in all electrode sites (F3, F7, Fp2, F4 and F8) except Fp1. Findings revealed individuals with PFS have higher amount of slow wave oscillations compared with fast wave oscillations, a well-established marker for cerebral abnormalities most commonly reported in ischemia [15], [30] but also reported in network disruption [30]. This finding is consistent with that reported in other populations including acute ischemic stroke [20], [23], subacute stroke [31] and mild traumatic brain injury (review) [25].

We also hypothesized DAR in the left frontal electrode sites would be significantly higher than the right frontal electrode sites due to less volume in fronto-cerebellar fibers in the left more than the right cortex in children with PFS compared to healthy peers [12]. This hypothesis was not upheld. Higher DAR was found in both right and left side electrode sites. Information regarding tumor location was gathered post hoc and revealed all participants (PFS and Non-PFS) had midline tumor location that filled the 4th ventricle, which may explain these findings. One prediction for acquiring PFS is surgical injury to superior cerebellar peduncle, where 90–100% of the fronto-cerebellar fiber (FCF) pathways travel [12]. While all participants had midline tumors, those who acquired PFS may have had a surgical injury to the superior cerebellar peduncle while those who did not acquire PFS may have had the superior cerebellar peduncle spared.

Finally, we hypothesized there would be a significant relationship between DAR and ataxia severity scores. This hypothesis was upheld. DAR in F7 was found to have consistently strong, positive and statistically significant correlation with all ataxia severity scores including balance, kinetic, gait and total scores on the SARA as well as prosody, rate and overall speech suggesting increased DAR in the left inferior frontal / anterior temporal cortex to be most closely associated with more severe ataxia symptoms across gross motor, fine motor and speech domains. While DAR values in the left and right dorsolateral prefrontal cortex (F3 and F4) were associated with ataxia severity, right prefrontal cortex (Fp2) was also correlated with ataxia severity in some, but not all domains tested. Although, increases in delta oscillations have been associated with motor impairment following acute brain injury [23], [28], to our knowledge, this is the first study to identify the relationship between DAR and ataxia symptoms.

Diaschisis

Diaschisis, or more specifically, connection diaschisis, is defined as the changes of structural and function connectivity between brain areas distant to the lesion due to deafferentation, reduced cerebral blood flow and metabolism [32], [33]. Both delta and alpha oscillations are generated by pyramidal cells that are sensitive to reduced oxygen [8]. As such, EEG changes have been correlated with changes in cerebral blood flow [17], [19], specifically, increased delta and decreased alpha has been associated with hypoperfusion [19], [34]. In general, reperfusion metabolic recovery has been associated with functional recovery [33]. Although, more recent work found reduction in DAR as a response to return of blood flow, but it was not always associated with restored function or tissue preservation [35]. Miller and colleagues (2010) found decreased cerebral blood flow in the right frontal cortex and reduced blood volume in the left frontal region in children with PFS compared with controls [11]. Relatedly, deafferentation visualized by a reduction in fronto-cerebellar fibers (FCF) potentially leading to cerebellocortical diaschisis in PFS has been reported [12]. The increased DAR values observed in the PFS group in this study may be a reflection of reduced cerebral blood flow and/or metabolism in the left and right frontal cortical regions and/or deafferentation, both of which are signs of diaschisis.

DAR in Stroke and Brain Injury Recovery

While EEG oscillations have not been studied in the recovery of PFS to date, they have been shown to predict motor recovery in stroke [36]. Specifically, high DAR has been associated with poorer motor outcome, specifically in delayed cerebral ischemia following acquired brain injury [24] and mild traumatic brain injury [28]. Finnegan and colleagues (2004) measured diffusion and perfusion through MRI and frequency oscillations using EEG in the acute stage following ischemic stroke and reported the delta change index in the acute stage following stroke correlated with lesion volume at 15 hours post symptom onset and 30-day NIH Stroke Scale (NIHSS) [37] and DAR at 49 days post symptom onset was significantly correlated with 30-day NIHSS [38]. In another study, these authors determined DAR to have 100% specificity for the absence of and 100% sensitivity for the presence of an ischemic stroke lesion compared to healthy controls [23]. Authors suggested DAR may reflect the integrity of the penumbra, which may inform future studies.

Furthermore, DAR was correlated with the National Institutes of Health Stroke Scale (NIHSS) and modified Rankin Scale suggesting DAR may be a meaningful biomarker for stroke recovery [26]. DAR was significantly correlated with scores on the functional independence measure (FIM) and functional assessment measure (FAM), with the increase of DAR being associated with poorer performance on FIM/FAM scale in individuals with acute ischemic stroke [27]. While, no correlation was found between DAR and scores on the Fugl Meyer Scale in chronic stroke [39] suggesting DAR may be predictive of functional motor recovery in acute and subacute phases of stroke recovery but less predictive in chronic stroke. Elevated DAR values have also been found across cortical regions in acquired brain injury that are associated with scores on FIM/FAM functional outcome measures [28]. In a meta-analysis, DAR was found to be interpreted as a 1) reflection of global network dysfunction, 2) biomarker for remote functional depression rather than just focal damage and 3) phenomenon that is correlated with poorer motor outcomes [40]. All of which can inform neurophysiologic mechanisms underlying PFS.

Limitations

The target sample of this study was 8 participants in each group, however due to relatively limited database, geographic location of individuals in the database and funding timeline, seventeen participants were enrolled and completed the study. Although seventeen participants completed the study and despite the EEG being collected by experienced researchers in a Faraday room with minimal electrode impedance, EEG recordings from six participants were eliminated from the analysis as they did not have 2 minutes of clean data within the recordings. Data was collected at one time point which may not be reflective of the variability of EEG oscillations. In addition, the individuals' activity prior to the EEG recording was not controlled. Both of which offer opportunities for investigation.

Future Directions

Findings from this work provides a foundation for future research including the inclusion of neuroimaging to confirm if DAR is truly a reflection of diaschisis in the frontal regions identified in this study, which would provide a roadmap to a potential site for non-invasive brain stimulation. Future work might also compare DAR across multiple time-points throughout the recovery process to elucidate the

neurophysiology of recovery. Finally, a larger study comparing tumor location and duration since surgery would also provide valuable insight into the utility of DAR; all which could point to DAR as a biomarker for PFS in the future.

Conclusion

This exploratory study identified DAR as a potential sensitive neurophysiologic marker for differential diagnosis of PFS that may also be associated with ataxia severity. These findings may be an indirect reflection of the integrity of the dento-thalam-cortical pathway and/or cerebello-cortical diaschisis that have been implicated in PFS. Further, findings from this exploratory pilot can contribute to the diagnosis of PFS, provide a benchmark for recovery and help identify therapeutic options that promote neuroplasticity, such as non-invasive brain stimulation paired with behavioral rehabilitation.

Declarations

Consent to participate

Informed consent and assent were obtained from parents and participants under the age of 18 years. All participants over 18 years were able to consent for themselves.

Ethics approval

This study was approved by the University of Minnesota Institutional Review Board.

Author Contribution

S.S.G, Y.L., C.D., E.O. and C.S. wrote the main manuscript textS.S.G., Y.L., C.S. prepared figuresAll authors reviewed the manuscript

Acknowledgement

The authors thank the Masonic Institute for the Developing Brain (MIDB) and the Center for Neurobehavioral Development (CNBD) at the University of Minnesota for providing the research space, equipment, and infrastructure support necessary to conduct this study.

Data Availability

The raw data that support the findings of this study are available from the corresponding author, but restrictions apply to the availability of these data. The data are, however, available upon request.

Funding

Funding for this study was supported by an internal research grant from the University of Minnesota Duluth.

References

1. Catsman-Berrevoets CE, Aarsen FK. The spectrum of neurobehavioural deficits in the Posterior Fossa Syndrome in children after cerebellar tumour surgery. *Cortex*. Jul. 2010;46(7):933–46. 10.1016/j.cortex.2009.10.007.
2. Jabarkheel R et al. Aug., Molecular correlates of cerebellar mutism syndrome in medulloblastoma, *Neuro-Oncology*, p. noz158, 2019, 10.1093/neuonc/noz158
3. Pollack IF, Polinko P, Albright LA, Towbin R, Fitz C. Mutism and Pseudobulbar Symptoms after Resection of Posterior Fossa Tumors in Children, *Neurosurgery*, vol. 37, no. 5, pp. 885–892, Nov. 1995, 10.1227/00006123-199511000-00006
4. Wells EM et al. Apr., Postoperative cerebellar mutism syndrome following treatment of medulloblastoma: neuroradiographic features and origin: Clinical article, *PED*, vol. 5, no. 4, pp. 329–334, 2010, 10.3171/2009.11.PEDS09131
5. Gudrunardottir T, et al. Consensus paper on post-operative pediatric cerebellar mutism syndrome: the Iceland Delphi results. *Childs Nerv Syst*. Jul. 2016;32(7):1195–203. 10.1007/s00381-016-3093-3.
6. De Smet HJ, Baillieux H, Catsman-Berrevoets C, De Deyn PP, Mariën P, Paquier PF. Postoperative motor speech production in children with the syndrome of ‘cerebellar’ mutism and subsequent dysarthria: A critical review of the literature, *European Journal of Paediatric Neurology*, vol. 11, no. 4, pp. 193–207, Jul. 2007, 10.1016/j.ejpn.2007.01.007
7. De Smet HJ et al. Nov., Long-term cognitive deficits following posterior fossa tumor resection: A neuropsychological and functional neuroimaging follow-up study., *Neuropsychology*, vol. 23, no. 6, pp. 694–704, 2009, 10.1037/a0016106
8. Palmer SL, et al. Neurocognitive outcome 12 months following cerebellar mutism syndrome in pediatric patients with medulloblastoma. *Neurooncology*. Aug. 2010;094:noq. 10.1093/neuonc/noq094.
9. Schreiber JE et al. Nov., Posterior fossa syndrome and long-term neuropsychological outcomes among children treated for medulloblastoma on a multi-institutional, prospective study, *Neuro-Oncology*, vol. 19, no. 12, pp. 1673–1682, 2017, 10.1093/neuonc/nox135
10. Hartley H, et al. Ataxia and mobility in children following surgical resection of posterior fossa tumour: A longitudinal cohort study. *Childs Nerv Syst*. Sep. 2021;37(9):2831–8. 10.1007/s00381-021-05246-0.
11. Miller NG et al. Feb., Cerebellocerebral Diaschisis Is the Likely Mechanism of Postsurgical Posterior Fossa Syndrome in Pediatric Patients with Midline Cerebellar Tumors, *AJNR Am J Neuroradiol*, vol.

- 31, no. 2, pp. 288–294, 2010, 10.3174/ajnr.A1821
12. Soelva V, et al. Fronto-cerebellar fiber tractography in pediatric patients following posterior fossa tumor surgery. *Childs Nerv Syst.* Apr. 2013;29(4):597–607. 10.1007/s00381-012-1973-8.
 13. Murri L, Gori S, Massetani R, Bonanni E, Marcella F, Milani S. Evaluation of acute ischemic stroke using quantitative EEG: A comparison with conventional EEG and CT scan, *Neurophysiologie Clinique/Clinical Neurophysiology*, vol. 28, no. 3, pp. 249–257, Jun. 1998, 10.1016/S0987-7053(98)80115-9
 14. Yin X, Chen Y, Song X, Xing Y. The Prognostic Value of Quantitative Electroencephalography Combined with Transcranial Doppler in Patients with Ischemic Stroke in Neurological Intensive Care Units. *Neuropsychiatry.* 2018;08(03). 10.4172/Neuropsychiatry.1000433.
 15. Nuwer MR, Jordan SE, Ahn SS. Evaluation of stroke using EEG frequency analysis and topographic mapping, *Neurology*, vol. 37, no. 7, pp. 1153–1153, Jul. 1987, 10.1212/WNL.37.7.1153
 16. Jordan KG. Emergency EEG and Continuous EEG Monitoring in Acute Ischemic Stroke. *J Clin Neurophysiol.* Sep. 2004;21(5):341–52. 10.1097/01.WNP.0000145005.59766.D2.
 17. Sharbrough FW, Messick JM, Sundt TM. Correlation of Continuous Electroencephalograms With Cerebral Blood Flow Measurements During Carotid Endarterectomy. *Stroke.* Jul. 1973;4(4):674–83. 10.1161/01.STR.4.4.674.
 18. Nagata K, Tagawa K, Hiroi S, Shishido F, Uemura K. Electroencephalographic correlates of blood flow and oxygen metabolism provided by positron emission tomography in patients with cerebral infarction. *Electroencephalogr Clin Neurophysiol.* Jan. 1989;72(1):16–30. 10.1016/0013-4694(89)90027-8.
 19. O’Gorman RL et al. Jul., Coupling Between Resting Cerebral Perfusion and EEG, *Brain Topogr*, vol. 26, no. 3, pp. 442–457, 2013, 10.1007/s10548-012-0265-7
 20. Machado C et al. Jul., Assessing Acute Middle Cerebral Artery Ischemic Stroke by Quantitative Electric Tomography, *Clin EEG Neurosci*, vol. 35, no. 3, pp. 116–124, 2004, 10.1177/155005940403500303
 21. Finnigan S, Van Putten MJAM. EEG in ischaemic stroke: Quantitative EEG can uniquely inform (sub-)acute prognoses and clinical management. *Clin Neurophysiol.* Jan. 2013;124(1):10–9. 10.1016/j.clinph.2012.07.003.
 22. Greve S, et al. Age-specific distributions of total power, frequency bands, and alpha/delta ratio in quantitative EEG. *Intensive Care Med – Paediatr Neonatal.* Oct. 2025;3(1):39. 10.1007/s44253-025-00092-3.
 23. Finnigan S, Wong A, Read S. Defining abnormal slow EEG activity in acute ischaemic stroke: Delta/alpha ratio as an optimal QEEG index. *Clin Neurophysiol.* Feb. 2016;127(2):1452–9. 10.1016/j.clinph.2015.07.014.
 24. Claassen J, et al. Quantitative continuous EEG for detecting delayed cerebral ischemia in patients with poor-grade subarachnoid hemorrhage. *Clin Neurophysiol.* Dec. 2004;115(12):2699–710. 10.1016/j.clinph.2004.06.017.

25. Rapp PE, et al. Traumatic Brain Injury Detection Using Electrophysiological Methods. *Front Hum Neurosci*. Feb. 2015;9. 10.3389/fnhum.2015.00011.
26. Ajčević M, et al. Hyper-acute EEG alterations predict functional and morphological outcomes in thrombolysis-treated ischemic stroke: a wireless EEG study. *Med Biol Eng Comput*. Jan. 2021;59(1):121–9. 10.1007/s11517-020-02280-z.
27. Schleiger E, Sheikh N, Rowland T, Wong A, Read S, Finnigan S. Frontal EEG delta/alpha ratio and screening for post-stroke cognitive deficits: The power of four electrodes. *Int J Psychophysiol*. Oct. 2014;94(1):19–24. 10.1016/j.ijpsycho.2014.06.012.
28. Leon-Carrion J, Martin-Rodriguez JF, Damas-Lopez J, Barroso JM, Martin Y, Dominguez-Morales MR. Delta–alpha ratio correlates with level of recovery after neurorehabilitation in patients with acquired brain injury, *Clinical Neurophysiology*, vol. 120, no. 6, pp. 1039–1045, Jun. 2009, 10.1016/j.clinph.2009.01.021
29. Arai Y, Okanishi T, Oguri M, Kanai S, Fujimoto A, Maegaki Y. Power and connectivity changes on electroencephalogram in postoperative cerebellar mutism, *Brain and Development*, vol. 44, no. 10, pp. 759–764, Nov. 2022, 10.1016/j.braindev.2022.06.006
30. Baang HY et al. Mar., The Utility of Quantitative EEG in Detecting Delayed Cerebral Ischemia After Aneurysmal Subarachnoid Hemorrhage, *Journal of Clinical Neurophysiology*, vol. 39, no. 3, pp. 207–215, 2022, 10.1097/WNP.0000000000000754
31. Fanciullacci C, et al. Delta Power Is Higher and More Symmetrical in Ischemic Stroke Patients with Cortical Involvement. *Front Hum Neurosci*. Jul. 2017;11:385. 10.3389/fnhum.2017.00385.
32. Carrera E, Tononi G. Diaschisis: past, present, future. *Brain*. Sep. 2014;137(9):2408–22. 10.1093/brain/awu101.
33. Meyer JS, Obara K, Muramatsu K. Diaschisis. *Neurol Res*. Dec. 1993;15(6):362–6. 10.1080/01616412.1993.11740164.
34. Foreman B, Claassen J. Quantitative EEG for the detection of brain ischemia. *Crit Care*. 2012;16(2):216. 10.1186/cc11230.
35. Schleiger E, Wong A, Read S, Coulthard A, Finnigan S. Improved cerebral pathophysiology immediately following thrombectomy in acute ischaemic stroke: Monitoring via quantitative EEG. *Clin Neurophysiol*. Aug. 2016;127(8):2832–3. 10.1016/j.clinph.2016.06.001.
36. Cassidy JM, et al. Low-Frequency Oscillations Are a Biomarker of Injury and Recovery After Stroke. *Stroke*. May 2020;51(5):1442–50. 10.1161/STROKEAHA.120.028932.
37. Finnigan SP, et al. Correlation of Quantitative EEG in Acute Ischemic Stroke With 30-Day NIHSS Score: Comparison With Diffusion and Perfusion MRI. *Stroke*. Apr. 2004;35(4):899–903. 10.1161/01.STR.0000122622.73916.d2.
38. Finnigan SP, Walsh M, Rose SE, Chalk JB. Quantitative EEG indices of sub-acute ischaemic stroke correlate with clinical outcomes, *Clinical Neurophysiology*, vol. 118, no. 11, pp. 2525–2532, Nov. 2007, 10.1016/j.clinph.2007.07.021

39. Saes M, Meskers CGM, Daffertshofer A, De Munck JC, Kwakkel G, Van Wegen EEH. How does upper extremity Fugl-Meyer motor score relate to resting-state EEG in chronic stroke? A power spectral density analysis. Clin Neurophysiol. May 2019;130(5):856–62. 10.1016/j.clinph.2019.01.007.

40. Sood I, et al. Quantitative electroencephalography to assess post-stroke functional disability: A systematic review and meta-analysis. J Stroke Cerebrovasc Dis. Dec. 2024;33:108032. 10.1016/j.jstrokecerebrovasdis.2024.108032.

Figures

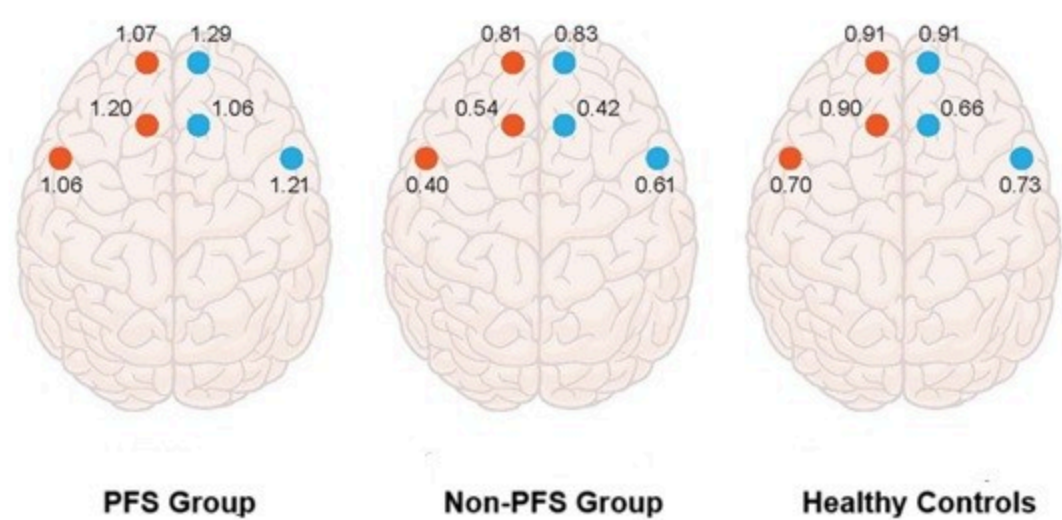


Figure 1

Median delta/alpha ratio (DAR) values at each electrode site. DAR values in the frontopolar (Fp1-Fp2), frontal (F3-F7) and anterior temporal (F4-F8) across all groups, those status post posterior fossa tumor resection that acquired posterior fossa syndrome (PFS Group), did not acquire PFS (Non-PFS Group) and healthy controls

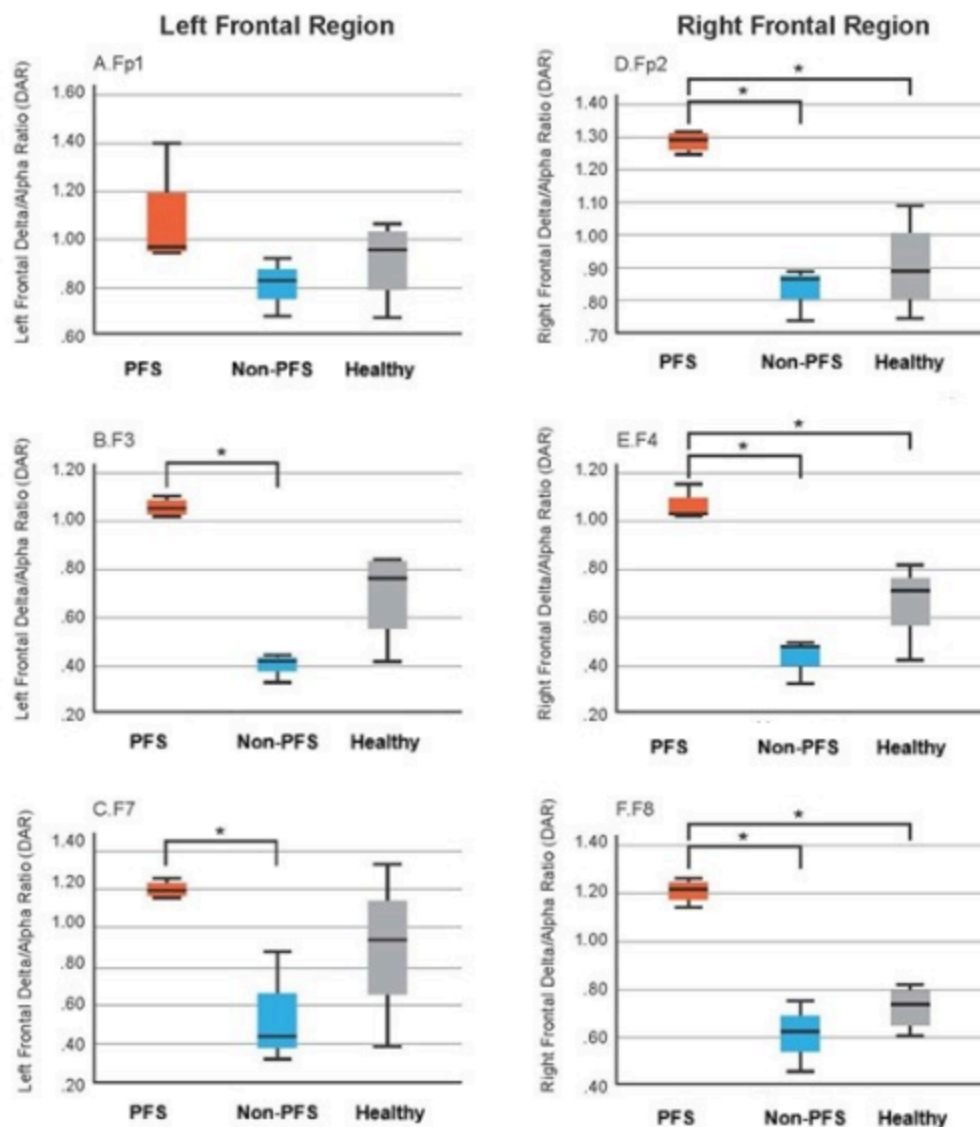


Figure 2

DAR values in the frontopolar (Fp1-Fp2), frontal (F3-F7) and anterior temporal (F4-F8) across all groups, those status post posterior fossa tumor resection that acquired posterior fossa syndrome (PFS Group), did not acquire PFS (Non-PFS Group) and healthy control (Control Group)