ORIGINAL RESEARCH

Quantitative Electroencephalography After Pediatric Anterior Circulation Stroke

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Objective: Regional differences were investigated in quantitative EEG (QEEG) characteristics and associations of QEEG to hemodynamics after pediatric acute stroke.

Methods: Quantitative EEG was analyzed, including power in delta, theta, alpha, and beta bands, alpha–delta power ratio, total power, and spectral edge frequency from 11 children with unilateral, anterior circulation strokes during the first 24 hours of continuous EEG recording. Differences between injured and uninjured hemispheres were assessed using multivariate dynamic structural equations modeling. Dynamic structural equations modeling was applied to six children with hemorrhagic stroke undergoing arterial blood pressure, heart rate, and cerebral oximetry monitoring to investigate associations between hemodynamics with QEEG adjacent to anterior circulation regions.

Results: All patients with acute ischemic stroke ($n = 5$) had lower alpha and beta power and spectral edge frequency on injured compared with uninjured regions. This was not consistent after hemorrhagic stroke ($n = 6$). All hemorrhagic stroke patients demonstrated negative association of total power with arterial blood pressure within injured regions. No consistency was observed for direction or strength of association in other QEEG measures to arterial blood pressure nor were such consistent relationships observed for any QEEG measure studied in relation to heart rate or cerebral oximetry.

Conclusions: After pediatric anterior circulation acute ischemic stroke, reduced spectral edge frequency and alpha and beta power can be observed on injured as compared with noninjured regions. After pediatric anterior circulation hemorrhagic stroke, total power can be negatively associated with arterial blood pressure within injured regions. Larger studies are needed to understand conditions in which QEEG patterns manifest and relate to hemodynamics and brain penumbra.

Key Words: Quantitative electroencephalography, Pediatric stroke, Cerebral near-infrared spectroscopy, Arterial blood pressure, Dynamic structural equations modeling.

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Acute stroke occurs when there is insufficient cerebral blood flow to meet metabolic demand. This often comes in the form of arterial ischemic or hemorrhagic stroke (HS). The utility of EEG in monitoring for cerebral ischemia in adult neurocritical care is understood[1,2] with existing consensus recommendations, suggesting its use for identification of ischemia in patients at high risk.[3] When normal cerebral blood flow declines to approximately 25 to 35 $mL \cdot 100\ g^{-1} \cdot minute^{-1}$, continuous EEG loses faster frequencies. Similarly, as cerebral blood flow decreases lower to approximately 17 to 18 $mL \cdot 100\ g^{-1} \cdot minute^{-1}$, slower frequencies gradually increase.[1] Alpha variability and alpha–delta power ratio have been shown to be predictive of delayed cerebral ischemia in adults with aneurysmal subarachnoid hemorrhage.[4,5] Alpha activity can be assessed to provide information regarding the necessity for shunt insertion and detection of cerebral ischemia during carotid endarterectomies.[6] After adult stroke, alpha, beta, and delta power bands have been shown to be independent predictors of poststroke outcome.[7]

Although the dynamic nature of EEG makes it useful for detection of new-onset cerebral ischemia, its utility in the management of penumbral tissue after stroke occurs remains poorly understood. In this study, we aimed to investigate characteristics of quantitative EEG (QEEG) after pediatric ischemic and HS as well as their association with hemodynamics: mean arterial blood pressure (ABP), heart rate (HR), and cerebral oximetry (rSO_2).

METHODS

Patient Population

Children who were admitted to the Pediatric Intensive Care Unit at Phoenix Children's Hospital between January 2014 and January 2020 with acute ischemic stroke (AIS) or HS were evaluated if they

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All work was performed at the Barrow Neurological Institute at Phoenix Children's Hospital.

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TABLE 1. Demographics

Patient	Age (Years)	Race	Gender	Etiology	Vascular Distribution	% Artifact	ICP, Mean (mm Hg)	% Time With ICP >20 mm Hg	Vasoactive or Antihypertensive Medications Used	Neurosurgical Procedures
AIS-1	17	Hispanic	Male	Ischemic, dissection	Right MCA	16.4	N/A	N/A	None	None
AIS-2*	13	Caucasian	Female	Ischemic, dissection	Left MCA	33.5	N/A	N/A	None	None
AIS-3	10	Hispanic	Female	Ischemic, surgical	Right MCA	62.8	N/A	N/A	Norepinephrine	Aneurysm clipping removal and wrapping
AIS-4	10	Hispanic	Female	Ischemic, cardioembolic	Right MCA	3.7	N/A	N/A	Epinephrine, dobutamine, milrinone	Mechanical thrombectomy
AIS-5	12	Hispanic	Female	Ischemic, idiopathic	Left MCA	11.6	N/A	N/A	None	None
HS-1*	6	Mixed	Male	Hemorrhagic, ECMO	Right MCA	8.1	15.1	13.1	Epinephrine, milrinone	Decompressive craniectomy
HS-2*	10	Hispanic	Male	Hemorrhagic, AVM rupture	Left MCA	80.1	16.6	15.7	Epinephrine	Decompressive craniectomy
HS-3*	11	Hispanic	Female	Hemorrhagic, AVM rupture	Left MCA	52.1	11.1	1.1	Norepinephrine	Decompressive craniectomy
HS-4*	14	Caucasian	Male	Hemorrhagic, AVM rupture	Right MCA	48.9	9.1	3.7	Norepinephrine	Decompressive craniectomy
HS-5*	10	Caucasian	Male	Hemorrhagic, AVM rupture	Right MCA	17.8	5.8	0.0	Nicardipine	Decompressive craniectomy
HS-6*	1	Hispanic	Female	Hemorrhagic, aneurysm rupture	Right MCA	36.9	N/A	N/A	Nicardipine	None

*Patients who underwent hemodynamic monitoring.

AIS, arterial ischemic stroke; AVM, arteriovenous malformation; ECMO, extracorporeal membrane oxygenation; ICP, intracranial pressure; HS, hemorrhagic stroke; MCA, middle cerebral artery; mm Hg, millimeters of mercury; N/A, not applicable; %, percent.

underwent multimodal neurologic monitoring that included continuous EEG. Depending on clinical circumstances, multimodal neurologic monitoring could also include monitoring rSO_2, intracranial pressure, end-tidal carbon dioxide content, central venous pressure, and peripheral oxygen saturation. All patients underwent neuroprotective measures that included maintaining sodium levels >140 mmol/L, magnesium levels >2 mg/dL, normotension for age, and arterial carbon dioxide content between 35 and 45 mm Hg if intubated. Retrospective analysis from a prospectively collected clinical database was performed under approval of the Phoenix Children's Hospital Institutional Review Board (No: 17-142). This study limited its investigation to children who had acute stroke isolated to anterior circulation in one cerebral hemisphere.

Data Collection

Continuous EEG was captured using institutional standard clinical hardware (Xltek; Natus Medical, Pleasanton, CA) under the International 10 to 20 system with a sampling rate of 512 Hz, low-frequency filter of 1 Hz, and high-frequency filter at 70 Hz. Activity from the first 24 hours of recording was considered for this study. QEEG analysis was performed using Persyst Clinical Review (Persyst, Prescott, AZ). To account for heterogeneity in precise stroke locations within the corresponding vascular distribution, QEEG characteristics were averaged across electrodes over the anterior circulation that included injured and uninjured hemispheres. Left anterior circulation electrodes included Fp1-F7, F7-T7, Fp1-F3, and F3-C3. Right anterior circulation electrodes included Fp2-F8, F8-T8, Fp2-F4, and F4-C4. Bipolar montages were used during review. We investigated QEEG characteristics including alpha–delta power ratio,

TABLE 2. Proportion of Patients With Significantly Lower QEEG Values in the Injured Hemisphere Compared With the Uninjured

	AIS ($n = 5$)	HS ($n = 6$)
Delta	0.2	0.0
Theta	0.8	−0.3
Alpha	1.0	0.3
Beta	1.0	0.0
ADR	0.2	0.3
TP	−0.6	0.0
SEF	1.0	0.5

Negative proportions indicate the uninjured hemisphere had significantly lower QEEG values than the injured hemisphere. **Supplemental Digital Content 1** (see **Table**, http://links.lww.com/JCNP/A142) provides detailed results information.

ADR, alpha–delta power ratio; AIS, arterial ischemic stroke; HS, hemorrhagic stroke; QEEG, quantitative EEG; SEF, spectral edge frequency; TP, total power.

TABLE 3. Number of HS Patients With Significant Association Between Physiological Signals (ABP, rSO2, and HR) and QEEG

	HS (n = 6)											
	ABP				rSO2				HR			
Hemisphere	Injured		Uninjured		Injured		Uninjured		Injured		Uninjured	
Direction	+	−	+	−	+	−	+	−	+	−	+	−
Delta	5	0	3	3	3	3	3	3	4	2	3	3
Theta	5	1	4	1	2	4	2	4	4	2	3	3
Alpha	1	5	2	3	4	2	4	2	3	2	2	3
Beta	3	1	4	1	4	2	5	1	5	1	5	1
ADR	2	2	2	2	3	3	2	3	1	5	2	4
TP	0	6	2	4	3	3	4	2	1	4	2	3
Edge	1	4	3	3	1	5	3	3	3	2	2	3

ABP, mean arterial blood pressure; ADR, alpha–delta power ratio; HR, heart rate; HS, hemorrhagic stroke; QEEG, quantitative EEG; rSO2, regional cerebral oximetry; SEF, spectral edge frequency; TP, total power.

power in alpha (8–13 Hz), beta (13–20 Hz), delta (1–4 Hz), and theta (4–7 Hz) bands, and total power (TP) between 0 and 32 Hz. We also included spectral edge frequency (SEF) represented by the frequency below which 95% of the TP is located. Quantitative EEG values were computed using an 8-second window and produced a 0.125 Hz time series for each hemisphere. Recordings were reviewed by a board-certified epileptologist (B.L.A.), and patients with seizures in their first 24 hours were excluded.

When collected, rSO_2 data were collected using near-infrared spectroscopy (Covidien, Walpole, MA) with probe placement over the bifrontal scalp. Systemic ABP and HR were collected using radial arterial line and electrocardiogram monitoring, respectively, with data collected at 1 Hz. Continuous time stamped physiologic data from all monitoring devices were synchronized with EEG using a multimodal neurologic monitoring device (CNS200; Moberg ICU Solutions, Philadelphia, PA), with data stored within an institutional clinical database. Epochs of time with significant artifact in EEG, rSO_2, or ABP data were also excluded from data analysis.

Statistical Analyses

Dynamic structural equations modeling[8] was used to investigate time series differences between each QEEG characteristic on the injured and uninjured side of the brain for individual patients. Dynamic structural equations modeling produced estimated mean scores with corresponding 95% credible intervals based on Bayesian inference such that nonoverlapping intervals indicated statistically significant difference between the means. Multivariate dynamic structural equations modeling was used to estimate the strength and direction of linear associations of each QEEG characteristic within given brain regions to mean ABP, HR, and rSO_2 in HS patients who underwent monitoring that included all three hemodynamic elements. We investigated whether differences in direction and strength of associations existed within subjects on the injured and uninjured brain regions. The strength of associations was summarized using standardized coefficients and corresponding 95% credible intervals. Statistical significance was based on the interval not containing 0. Statistical analyses were performed using the statistical software packages R Studio Version 3.4.1 and Mplus 8.1 (Muthen and Muthen 1998–2018, Los Angeles, CA).

RESULTS

Demographics

Between January 2014 and January 2020, 134 children were admitted to the pediatric or cardiac intensive care units with acute

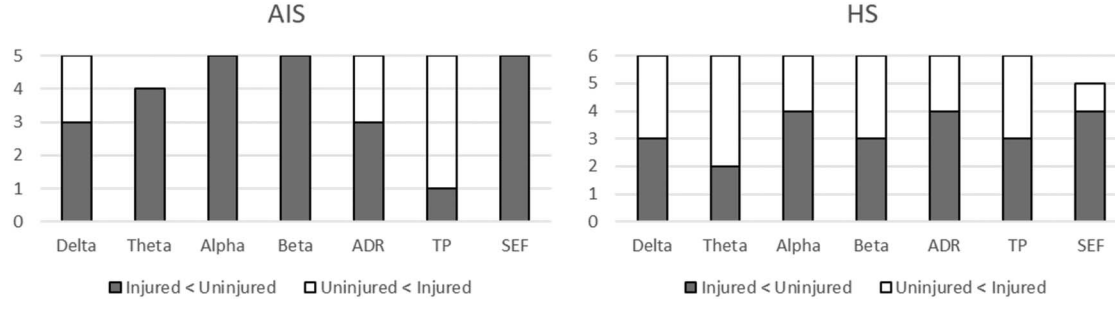

FIG. 1. Association of QEEG to injured and uninjured brain regions. Count of patients with significantly lower QEEG values in the injured hemisphere compared with the uninjured (gray bars) or significantly lower QEEG values in the uninjured hemisphere compared with the injured (white bars). **Supplemental Digital Content 1** (see **Table**, http://links.lww.com/JCNP/A142) provides detailed results. ADR, alpha–delta ratio; AIS, acute ischemic stroke; HS, hemorrhagic stroke; QEEG, quantitative EEG; SEF, spectral edge frequency; TP, total power.

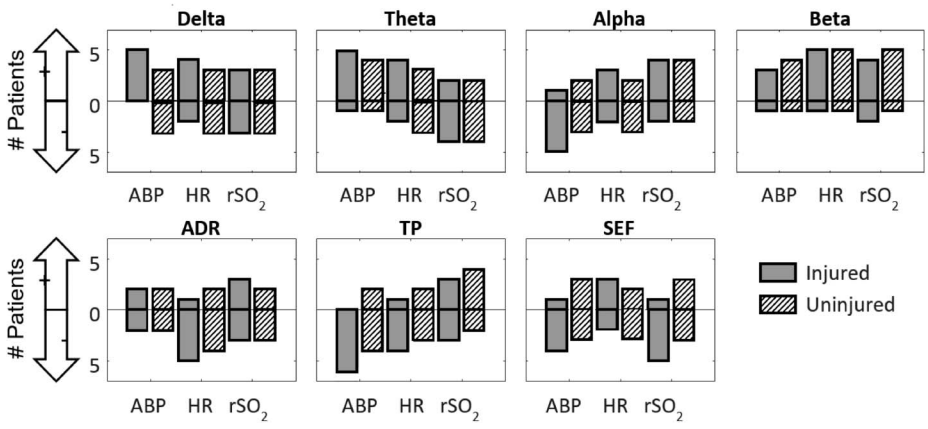

FIG. 2. Relationship of QEEG to hemodynamics. Number of HS patients with significant positive (+) or negative (−) associations between QEEG characteristics with hemodynamic measurements. QEEG on uninjured region is indicated by strips. All patients have a negative association between TP and ABP. **Supplemental Digital Content 2 to 7** (see **Tables** http://links.lww.com/JCNP/A143, http://links.lww.com/JCNP/A144, http://links.lww.com/JCNP/A145, http://links.lww.com/JCNP/A146, http://links.lww.com/JCNP/A147, http://links.lww.com/JCNP/A148) provide detailed information. ABP, mean arterial blood pressure; ADR, alpha–delta ratio; HR, heart rate; QEEG, quantitative EEG; rSO2, cerebral oximetry; SEF, spectral edge frequency; TP, total power.

stroke, of which 64 children (48%) presented with AIS and 70 (52%) were admitted with HS. Based on clinical need for advanced monitoring, 22 children with acute stroke (16%) underwent multimodal neurologic monitoring that included continuous EEG (Table 1). Among these 22 children, 11 (50%) had unilateral anterior circulation strokes without seizures within the first 24 hours of monitoring and were included for QEEG analysis (Table 1). Eight of 22 patients (36%) had AIS, of which six patients (75%) had a unilateral anterior circulation AIS, one patient (13%) had bilateral anterior circulation AIS, and one patient (13%) had a posterior circulation AIS. Two patients with AIS (25%) had seizures within the first 24 days of monitoring and were excluded from QEEG analysis, among which one patient (13%) had a unilateral anterior circulation AIS and one patient (13%) had bilateral anterior circulation AIS. This resulted in a total of five patients with AIS included for QEEG analysis. Fourteen of 22 patients (64%) who underwent multimodal neurologic monitoring had acute HS, among which eight patients (57%) had anterior circulation HS and six patients (43%) had posterior circulation HS. Of the eight patients with anterior circulation HS, 6 (75%) had unilateral anterior circulation HS and two had (25%) bilateral anterior circulation HS. No patients with HS who underwent multimodal neurologic monitoring had seizures within the first 24 hours of EEG monitoring. Of the 11 children with acute stroke who met criteria for inclusion in this analysis, there were six female and five male patients ranging in age from 1 to 17 years old (median 10.0, interquartile range 2.5). The etiologies in children with AIS included: idiopathic ($n = 1$), arterial dissection ($n = 3$), and cardioembolic stroke ($n = 1$). In children with HS, the etiologies included: arteriovenous malformation rupture ($n = 4$), aneurysmal subarachnoid hemorrhage ($n = 1$), and intracranial hemorrhage during extracorporeal membrane oxygenation ($n = 1$). Of the 11 patients, 7 (64%) had stroke within the right anterior circulation region and 4 (36%) had stroke within the left anterior circulation region. Seven of 11 patients (64%) underwent hemodynamic monitoring that included synchronized collection of HR, rSO_2, and invasive ABP data. Of those seven patients, one had AIS (14%) and six had HS (86%). Among the 11 patients who underwent QEEG analysis, percent artifact removed ranged from 3.7% to 80.1% (median 33.5, interquartile range 36.5). Five of six patients (83%) with HS required decompressive craniectomy before EEG monitoring and had concurrent intracranial pressure monitoring without evidence of significant intracranial hypertension. Two of five patients (40%) with AIS required neurosurgical interventions before EEG monitoring, and none required concurrent intracranial pressure monitoring. One patient (AIS-4) required mechanical thrombectomy of a right internal carotid artery clot before EEG monitoring. Another patient (AIS-3) underwent a right middle cerebral artery aneurysm clipping that resulted in stroke, requiring aneurysm clipping removal and wrapping before EEG monitoring. Seven of 11 patients (64%) required vasoactive or antihypertensive therapy during the day of recording, including use of norepinephrine, epinephrine, dobutamine, milrinone, or nicardipine. We did not observe worsening of ischemia or hemorrhagic transformation on repeat neuroimaging from our cohort.

QEEG Characteristics Across Hemispheres After AIS

Table 2 and Fig. 1 summarize the number of patients with significantly different QEEG characteristics between injured and uninjured hemispheres, and **Supplemental Digital Content 1** (see **Table**, http://links.lww.com/JCNP/A142) specifies further details. All five AIS patients (100%) had decreased alpha and beta power and SEF over the injured brain region as compared with the uninjured brain region. There was no consistency observed between differences in other QEEG characteristics within injured and uninjured brain regions.

QEEG Characteristics Across Hemispheres After HS

Table 2 and Fig. 1 also summarize the number of patients with significantly different QEEG characteristics between injured and uninjured anterior circulation brain regions, and **Supplemental Digital Content 1** (see **Table**, http://links.lww.com/JCNP/A142) specifies further details. There was no consistency observed between differences in QEEG characteristics within injured and uninjured brain regions.

Relationship of QEEG Characteristics to Hemodynamics

Table 3 and Fig. 2 summarize patients with significant associations between ABP, rSO_2, and HR with QEEG characteristics within injured or uninjured anterior circulation brain

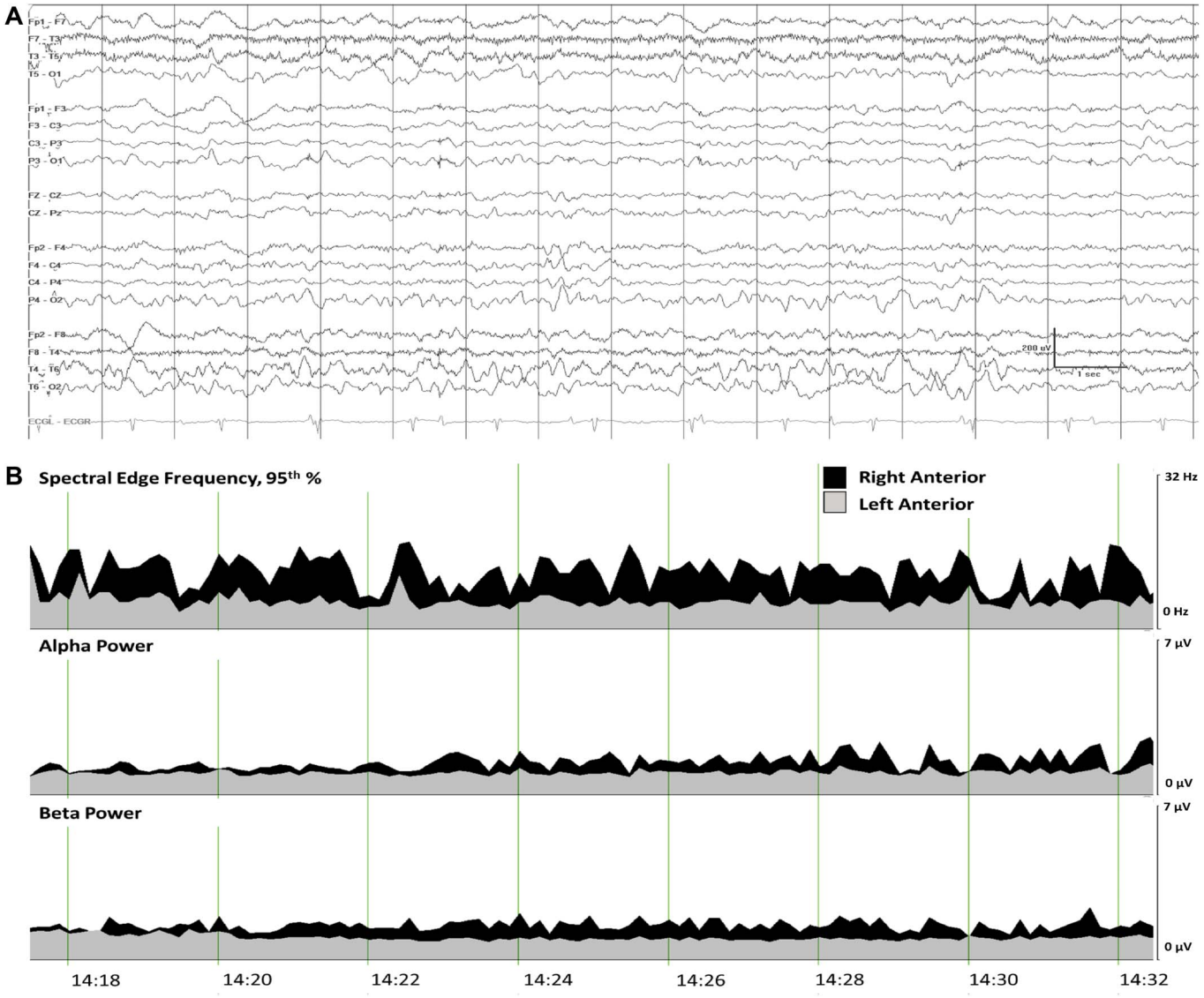

FIG. 3. Differences in QEEG parameters in relation to injured and noninjured brain regions. Example of raw EEG tracing (**A**) from patient, AIS-2 in addition to the corresponding QEEG trend (**B**) that demonstrates decreased spectral edge frequency, alpha power and beta power over the injured brain region (left anterior, gray) as compared with the uninjured brain region (right anterior, black). AIS, acute ischemic stroke; QEEG, quantitative EEG; Hz, hertz; µV, microvolts; %, percent.

regions in patients with HS, and further details are presented in **Supplemental Digital Content 2 to 7** (see **Tables**, http://links.lww.com/JCNP/A143, http://links.lww.com/JCNP/A144, http://links.lww.com/JCNP/A145, http://links.lww.com/JCNP/A146, http://links.lww.com/JCNP/A147, http://links.lww.com/JCNP/A148). All six patients had negative linear association between TP and ABP on injured brain regions. On uninjured regions, the direction of association was inconsistent (two positive and four negative cases). No patient had negative linear association between delta power and ABP on the injured brain region, with five of six patients (83%) carrying positive association of delta power and ABP, and with one of six patients (17%) with HS having no association. There were no other consistent patterns in direction or strength of association between TP, SEF, alpha–delta power ratio, or alpha, beta, or theta power to ABP. There were no consistent patterns in direction or strength of association of any QEEG characteristic studied to rSO_2 or HR.

DISCUSSION

This is the first study to investigate differences in QEEG characteristics between injured and uninjured brain regions as well as the relationships between QEEG and hemodynamics in children after stroke. Interestingly, we consistently observed that SEF and alpha and beta power were significantly decreased in injured brain regions as

compared with uninjured regions in children with acute AIS. However, this pattern was not seen in cases of HS. We found that lower ABP was associated with higher TP on injured regions in all children who underwent hemodynamic recordings, but no other consistent patterns between QEEG and hemodynamics were observed.

Much of the existing work investigating continuous EEG in stroke patients has been in adult populations where there is more stability of frequency power bands in relation to age.[1–7] Furthermore, existing work largely focused on how to use QEEG in the detection of new ischemia, as opposed to understanding how changes in continuous EEG characteristics relate to dynamics of ischemic penumbra with cerebral oximetry, HR, and ABP during management immediately after stroke occurrence. Very limited evidence exists for monitoring pressure augmentation in patients with ischemic penumbra using continuous EEG,[9] but QEEG characteristics have not been studied.

Although this study was exploratory in nature, the consistency of decreased SEF and alpha and beta power over stroke regions in children with AIS suggests that these QEEG characteristics may be useful for monitoring AIS in the at-risk regions of the brain in children undergoing extracorporeal membrane oxygenation[10] or congenital heart surgery. At times, these differences may be more apparent on QEEG trends than on the raw EEG tracing (Fig. 3). Our cohort, however, was limited to AIS patients with ages ranging between 10 and 17 years and further work is needed to investigate younger ages.

The consistent negative linear association of TP to ABP on injured brain regions of patients with HS may reflect a prominent rise in power below 1 Hz with reduced ABP, especially given that five of six patients demonstrated positive linear association of delta power (1–4 Hz) with ABP and no patients demonstrated negative associations within corresponding regions. Otherwise, there was lack of consistency in strength or direction of association for other QEEG measures in relation to ABP or in relation of any QEEG characteristic to HR or rSO_2. This lack of consistency challenges the assumption that after stroke occurs, changes in EEG frequency bands would shift similarly as in new-onset ischemia.[1] These findings raise questions as to what other factors may play a role in the relationship of QEEG to hemodynamics. Cortical versus subcortical injury characteristics may also be important, as subcortical pathologic conditions may lead to changes in specific frequencies.[11] Sizes of the infarct core and ischemic penumbra, sleep–wake cycling, and sedative medications used might also be major factors in understanding why specific relationships exist.[12] These potential differences raise new questions, and studies that incorporate advanced neuroimaging with perfusion and neurologic monitoring may be helpful in understanding how penumbra and infarct core in conjunction with the location may be relevant. Finally, the role of age at injury and the differences within the developing brain must also be considered and factored into these potential relationships.

Our study was limited by its retrospective nature, heterogeneity in stroke etiologies, as well as a very small sample size. Artifact can be substantial in relation to raw EEG and multimodal monitoring data, and while substantial efforts were made in data cleaning, some artifact may still be present. Substantial contributors to lengthy artifact epochs in our study included EEG sweat artifact, electrode popping, poor rSO_2 data quality, and arterial line removal. These events represent common occurrences in critical care and need to be accounted for when interpreting time series data involving QEEG and hemodynamics. We only reviewed QEEG characteristics in a bipolar montage, and similar dynamic structural equations modeling models using referential, and other montages may offer additional insight. The association of QEEG to rSO_2 is likely limited in patients for whom stroke location is distant from the near-infrared spectroscopy bifrontal scalp monitoring probes, and larger studies that assess distance of stroke from monitoring probes may be helpful. Because we only evaluated patients who required multimodal neurologic monitoring, our results are biased toward patients with severe strokes. Future work investigating QEEG changes either just before stroke occurrence or after mild strokes may demonstrate other findings that aid in a more comprehensive understanding of the relationship of QEEG to cerebrovascular physiology.

In conclusion, SEF and alpha and beta power were consistently lower in injured regions in patients with AIS and lower ABP was associated with higher TP within injured brain regions in patients with HS. These findings suggest that QEEG may be valuable for improving our understanding and potential monitoring of stroke in children. Larger studies that are standardized to age, stroke etiology, and that incorporate advanced neuroimaging may help better understand the relationship of QEEG characteristics to evolving hemodynamics after pediatric stroke.

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