

with VWI enhancement. **Results: Case 1.** 4-year old boy with sickle cell anemia, who developed encephalopathy during a hemolytic crisis. MR-VWI revealed bilateral extracranial internal carotid enhanced narrowing, deemed a secondary vasculopathy, with resolution upon follow-up.

Case 2. 16-year old male presented with left middle cerebral artery (MCA) infarction. VWI revealed left internal carotid terminus and proximal MCA enhancement. Conventional angiography showed abnormalities in mesenteric and hepatic arteries. Stability sustained on anticoagulation and immunosuppressive therapy.

Case 3. 10-year old girl, developed bilateral MCA infarctions with enhanced extracranial segments of both ICAs, and narrow PCAs, consistent with Moyamoya vasculopathy. Improved on combined immunosuppressive and anticoagulation therapy.

Case 4. 13-year old boy had an episode of right facial weakness, with a normal neurological exam; with enhancement and narrowing in the left extracranial ICA, likely an intramural hematoma from dissection. He responded to dual anticoagulation therapy. **Conclusions:** In conclusion, these cases illustrate similarities in vessel wall imaging abnormalities under different clinical contexts, with practical utility in longitudinal follow-up and prognostication.

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Cerebral Sinovenous Thrombosis in Preterm Infants

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Background: Neonatal cerebral sinovenous thrombosis (CSVT) can lead to severe brain injury and long-term neurodevelopmental impairments. Previous studies of neonatal CSVT have mainly included term infants. In this study, we examined the clinical and radiological features, treatment and outcome of CSVT in preterm infants. **Methods:** This was a retrospective cohort study of preterm infants born <37 weeks with radiologically confirmed CSVT. All MRI/MRV and CT/CTV scans were re-reviewed. Clinical and radiological data were analysed using descriptive statistics, ANOVA and chi-square tests. **Results:** A total of 26 preterm infants with CSVT were included. Of these, 65% were late preterm, 27% very preterm and 8% extreme preterm. Most were symptomatic (seizures 50%, abnormal exam 50%). Radiological features included transverse sinus (85%) and sagittal sinus thrombosis (42%), intraventricular hemorrhage (42%) and venous infarction (19%). Most preterm infants with CSVT (69%) were treated with anticoagulation. Anticoagulation was not associated with new or worsening intracranial hemorrhage. Outcome at follow-up ranged from no impairment (39%), mild impairment (19%), severe impairment (19%) and death (23%). **Conclusions:** Preterm infants with CSVT are often symptomatic and present with a distinct pattern of brain injury. Anticoagulation treatment of preterm CSVT appeared to be safe. Further studies and treatment guidelines for preterm CSVT are needed.

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Pediatric acute ischemic stroke protocols

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Background: Approximately 1,000 children present with AIS annually in North America. Most suffer from long-term disability. Childhood AIS is diagnosed after a median of 23 hours post-symptom onset, limiting thrombolytic treatment options that may improve outcomes. Pediatric stroke protocols decrease time to diagnosis. AIS treatment is not uniform across Canada, nor are pediatric stroke protocols standardized. **Methods:** We contacted neurologists at all 16 Canadian pediatric hospitals regarding their AIS management. **Results:** Response rate was 100%. Seven centers have an AIS protocol and two have a protocol under development. Seven centers do not have a protocol – two redirect patients to adult neurology, and five use a case-by-case approach for management. Analysis of the seven AIS protocols reveals differences: 1) IV-tPA dosage: age-dependent 0.75-0.9 mg/kg (n=1) versus age-independent 0.9 mg/kg (n=6), with maximum doses 75 mg (n=1) or 90 mg (n=6); 2) IV-tPA lower age cut-off: 2 years (n=4) versus 3, 4 or 10 years (n=1); 3) IV-tPA exclusion criteria: PedNIHSS score <4 (n=3), <5 (n=1), or <6 (n=3); 4) Pre-treatment neuroimaging: CT (n=3) versus MRI (n=4); 5) Intra-arterial tPA use (n=3). **Conclusions:** The seven Canadian pediatric AIS protocols show prominent differences. We plan a teleconference discussing a Canadian pediatric AIS consensus approach.

OTHER CHILD NEUROLOGY

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Curriculum mapping can facilitate transition to Competence by Design

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Background: Curriculum maps outline the content of an educational program identifying links between targeted outcomes, educational opportunities, and assessments. The transition to Competence by Design (CBD) in Canadian specialty residency programs requires thoughtful reorganization of educational programming. A curriculum map may assist with understanding the existing curriculum and thereby facilitate planning for CBD. **Methods:** A map of the pediatric neurology residency curriculum at the University of Calgary was constructed by linking objectives with related learning activities and assessments. Qualitative line-by-line analysis was then conducted to identify gaps in the existing curriculum. The map was used as a framework to plot CBD outcomes and curricular structure as these were established. **Results:** Generating the traditional curriculum map was time-consuming, requiring 48 hours. Careful review identified several objectives that did not link to formal learning activities or assessments. Many such gaps were recognized to link to non-clinical activities. Using the scaffold of the traditional curriculum reduced the

time required for mapping the planned CBD curriculum to 4 hours. **Conclusions:** The creation of a curriculum map prior to transition to CBD improved understanding of the existing curriculum and will facilitate transition to CBD. Ongoing evaluation of the fit of our predicted CBD map will support effective implementation.

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Emergency Department Use in Children with Cerebral Palsy: A Data Linkage Study

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Background: Improved understanding of factors predictive of emergency department (ED) visits in children with cerebral palsy (CP) can help optimize healthcare use. We sought to identify the pattern of ED consultations in these children. **Methods:** Data from the *Registre de paralysie cérébrale du Québec* and provincial administrative databases were linked. The CP cohort was comprised of children born between 1999 and 2002. Data pertaining to ED presentations between 1999 and 2012 were obtained. Relative risks were calculated to identify factors associated with increased ED visits. Peers without CP were selected from administrative databases and matched in a 20:1 ratio. Chi-square tests and Student's T-tests were used to compare the two cohorts. **Results:** 301 children with CP and 6040 peer controls were selected. Ninety-two percent (92%) of the CP cohort had at least one ED visit, compared to 74% amongst controls. Children with CP had an increased risk of high ED use compared to peers (RR 1.40 95% CI 1.30-1.52). Factors predictive of high ED use were comorbid epilepsy, severe motor impairment and low socioeconomic status. **Conclusions:** Children with CP have a higher need for urgent health assessments than their peers, resulting in increased use of ED services. System factors and barriers should be investigated.

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Case series: Clinical and genetic spectrum of SCN8A-related disorders in British Columbia

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Background: Children with pathogenic variations in *SCN8A* can present with early infantile epileptic encephalopathy-13, benign familial infantile seizures-5 or intellectual disability alone without epilepsy. In this case series, we discuss six children with variants in *SCN8A* managed at BC Children's Hospital. **Methods:** We describe clinical and genetic results on six individuals with *SCN8A* variants identified via clinical or research next-generation sequencing. Functional consequences of two *SCN8A* variants were assessed using electrophysiological analyses in transfected cells. **Results:** Clinical findings ranged from normal development with well-controlled epilepsy to significant developmental delay with treatment-resistant epilepsy. Phenotypes and genotypes in our cohort are described in the table below. Functional analysis supported gain-of-function in P2 and loss-of-function in P4. **Conclusions:** Our cohort expands the clinical and genotypic spectrum of *SCN8A*-related disorders. We establish functional evidence for two missense variants in *SCN8A*, including LoF variant in a patient with intellectual disability, and autism spectrum disorder without seizures.

Table for P.120

Patients	Age/Sex	Development	Age of seizure onset	Epilepsy type	Current antiseizure medication	Seizure frequency	Gene variant/ Function	Inheritance
P1	14y/F	Profound GDD	5m	Infantile spasms, LGS, hyperkinetic movements	Clobazam	Daily	c.1238C>A (p.Ala413Asp)	De novo
P2	6y/F	Normal	3-7m	Focal epilepsy	Carbamazepine	Seizure free	c.5630A>G (p.Asn1877Ser)/GoF	Paternal
P3	4y/F	Normal	12m	Focal epilepsy	Clobazam, topiramate	Seizure free	c.4447G>A (p.Glu1483Lys)	De novo
P4	6y/F	GDD, autism	3y - EEG abnormality only	-	Sodium valproate (discontinued)	No clinical seizure	c.971G>A (p.Cys324Tyr)/LoF, VUS in KCNQ3	De novo
P5	7y/M	GDD	5m	Generalized seizures	Ethosuximide, acetazolamide	Daily	c.773C>T (p.Thr258Ile)	De novo
P6	19y/F	Normal	10y	Focal epilepsy	Carbamazepine	Seizure free	c.986A>G (p.Asp329Gly)	De novo

Abbreviations: *Father with similar history, y Years, m Months, GDD Global developmental delay, LGS Lennox-Gastaut syndrome, VUS Variant of unknown significance, LoF Loss-of-function, GoF Gain-of-function, EEG Electroencephalogram, F - Female, M - Male, CBD - Cannabidiol