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School performance in children at the time of new-onset seizures and at long-term follow-up: A retrospective cohort study

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Abstract

Objective: School-performance difficulties (SPD) are common in children with epilepsy. The objectives of this study were to determine if the rate of SPD in children with seizures change from seizure-onset to follow-up and differ from children with psychiatric disorders.

Methods: School-aged children who required an initial electroencephalography (EEG) test in 2016 were reviewed and separated into two groups based on the presence or absence of seizures. Developmental delay and SPD were compared between groups at initial assessment and SPD was assessed after 2–4 years of follow-up. Analysis was also performed on a sub-set of patients with psychiatric disorders.

Results: At baseline, the rate of SPD was similar between the seizure (n = 146) and non-seizure (n = 332) groups [26% vs. 27%]. At follow-up, the seizure (n = 119) group had a significantly higher rate of SPD than the non-seizure (n = 215) group (54% vs. 43%). There was no difference in the rate of SPD between the seizure (n = 119) and psychiatric (n = 69) groups at baseline (31% vs. 43%) or follow-up (54% vs. 55%).

Conclusion: Over time, children with recurrent seizures experience more SPD than children without seizures, but similar SPD to children with psychiatric disorders.

Keywords

Seizures, epilepsy, children, psychiatric disorder, school performance

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Introduction

Irrespective of the level of intellect or seizure remission,[1] children with epilepsy experience more difficulties with school performance and make less academic progress compared to healthy children.[2–6] School-performance difficulties (SPD) in children with seizures are probably related to the neurobiological, cognitive, psychological, and social consequences of seizure recurrence.[7] The effect of various seizure-related variables, including use of anti-epileptic drugs (AEDs), and seizure frequency has not been fully established. For example, while some studies have found that increased seizure frequency is negatively related to school performance,[8,9] others have found no relationship.[10–12] In addition, data are lacking on the timing of SPD in children with epilepsy and on how SPD may change from seizure onset to long-term follow-up.[6,13]

Children with psychiatric disorders are also at risk of SPD.[14–17] Indeed, in spite of limited longitudinal population-based studies in children with mental health disorders, a correlation has been described between early childhood mental health disorders, school-performance and later educational attainment.[16,17] Due to the multifactorial nature of psychiatric disorders and the number of factors impacting educational performance, the relation between the two is complex.[18] Although several studies have assessed SPD and compared data from children with epilepsy to those with other chronic diseases (including asthma and diabetes),[2–6] there is a paucity of data comparing SPD from children with seizures to children with psychiatric disorders.

The main objective of this retrospective cohort study was to compare the rate of SPD in children with recent-onset seizures with that in children without seizures but who also required an investigative electroencephalography (EEG) test and determine if the rate changed over time (2–4 years). A second objective was to examine if there was a difference in the rate of SPD between children with seizures and children with psychiatric disorders.

Methods

Setting

This retrospective cohort study was conducted at British Columbia Children's Hospital (BCCH), which is the only tertiary-care centre in British Columbia, serving a population of 5 million people. The reporting of this study conforms to STROBE guidelines.[19] The study was approved by the BCCH and Women's Research Ethics Board (H21-00887) and written/verbal consent was not required because it was a retrospective study.

Study population

The BCCH database was searched for all patients who had an initial EEG test, in 2016 and were of school-age (4–19 years). Patients were included in the study if adequate clinical data, including information about school performance, were available from medical records and the BCCH database. There were no specific exclusion criteria.

It is routine practice at BCCH that children who require an EEG test, have their results and clinical data, including developmental skills and school performance history recorded at each EEG visit on a standardized form by a technologist who enters the data into the BCCH database. Most patients do not have routine neuropsychological assessments taken.

EEGs were ordered for various clinical indications, including clarification of paroxysmal events or assessment of altered level of consciousness. All patients were

required to be sleep deprived for the EEGs and they were recorded for 25–45 minutes using Biologic or Natus machines, according to the international 10–20 system with sampling frequency of 256 Hz and using 0.5–70 Hz filters. Hyperventilation and photic stimulation were routinely performed, unless they were not feasible due to patient cooperation or for contraindications (e.g., stroke history or severe pulmonary/cardiac disease).

Developmental delay was defined as a delay in meeting at least one developmental milestone; these included, gross motor, fine motor, expressive language and receptive language skills. School performance was assessed by a series of questions put to the parents/guardians on the degree of educational support required by the child. The severity of SPD was graded according to the following scale: A = average grade; B = learning assistance required; C = below grade level or in a special class; D = attends a special school or is integrated with a 1:1 aide. Children with grades B, C or D were considered to have SPD. Although, this was an arbitrary classification, we are of the opinion that it is based on practical information and has clinical significance.

Data collection

Data were extracted from the BCCH database and medical records. Information on school performance, developmental skills, clinical data (i.e., age at the time of EEG, history of seizures, number of seizures (from initial EEG to follow-up), diagnosis of epilepsy, seizure aetiology) were recorded.[20] Seizure type and aetiology were classified according to the 2017 International League Against Epilepsy.[21] In patients with seizures, the presence or absence of epileptiform discharges (including focal, multi-focal or generalized spike wave discharges) on the initial EEG were documented. In patients without seizures, the reason for the EEG, and other relevant diagnoses, were documented. Patients were included in the psychiatric diagnoses group if they had been diagnosed with a psychiatric disorder by a psychiatrist or paediatrician according to the DSM-V criteria at baseline or during the follow-up period. In patients with seizures and 2–4 years of follow-up data, the total number of seizures and AEDs administered during the follow-up period were documented.

Statistical analyses

Children were separated into two groups: seizures (i.e., those with new-onset clinical seizures) and non-seizures (i.e., required an EEG but did not have clinical seizures). Developmental delay and SPD were compared between the groups at the time of the initial EEG. In addition, SPD data were compared between the two groups for patients with 2–4 years of follow-up data after the initial EEG. An additional analysis was performed comparing data from the seizure group with a sub-set of children no seizures and a concomitant psychiatric illness with 2–4 years of follow-up data. SPD data were compared between groups at the time of the initial EEG and at follow-up. Statistical analysis was performed using R statistical software (R Core team 2021, Vienna, Austria.). Descriptive statistics were used to characterize the cohort.

In patients with seizures, a logistic regression model controlling for SPD status at baseline was used to determine if clinical factors influenced SPD at follow-up, including the number of seizures, number of AEDs, age at initial EEG and seizure aetiology. Logistic regression models were used to compare developmental delay and SPD in patients with and without seizures at baseline (i.e., initial EEG assessment). Odds ratios (OR) with 95% confidence intervals (CI) were

determined. In a sub-set analysis, patients with seizures were compared to patients with psychiatric disorders also using logistic regression models.

Results

Of 1140 children that had an initial EEG test in 2016, 509 were of school-age (4–19 years) and reviewed for the study. Of these 509 children, clinical information, including SPD data, were available for 478 patients of which, 146 (31%) were included in the seizure group and 332 (69%) in the non-seizure group (Table 1). In those children with seizures, aetiologies included: structural (i.e., tumour [n = 20], genetic [n = 9], infectious [n = 1], autoimmune [n = 3], unknown [55]), self-limited focal epilepsy (n = 16), juvenile myoclonic epilepsy (n = 25) and seizures due to hyponatremia (n = 17). Median ages at the time of the initial EEG for the seizure and non-seizure groups were 9 and 10 years, respectively.

At the time of the initial EEG, the proportion of children with developmental delay was similar between the groups: (22% vs 23%) and the SPD rate was also similar between group (26% vs 27%. Additionally, SPD graded by the severity scale was also similar between the groups (Table 1).

Adequate clinical data from the 2–4 year follow-up, were available for 119 (82%) children with recurrent seizures and 215 (65%) patients classified as no seizures (Table 1). Of the 119 children with seizures, 85 had 'other identified' aetiology for epilepsy (structural [genetic, infectious, metabolic, autoimmune, unknown]), 21 had self-limited epilepsy and 13 had genetic-generalized epilepsy. Patients without seizures had chronic psychiatric, movement disorders, migraines or systemic disorders that led them to have a long-term follow-up

Table 1. Developmental delay and school performance data from seizure and non-seizure groups who required an EEG test.

	Initial EEG assessment				Follow-up (2–4 years)			
	Seizure group n = 146	Non-seizure group n = 332	Odds ratio: 95% CI	Statistical significance	Seizure group n = 119	Non-seizure group n = 215	Odds ratio: 95% CI	Statistical significance
Developmental delay*								
No	111 (78)	248 (77)	0.99:	*ns*	na	na	–	–
Yes	32 (22)	76 (23)	0.62, 1.56		na	na		
SPD								
Yes	38 (26)	91 (27)	0.93:	*ns*	64 (54)	92 (43)	2.61:	$P < 0.005$
No	108 (74)	241 (73)	0.59, 1.44		53 (46)	123 (57)	1.41, 4.90	
SPD grade								
A	108 (74)	241 (73)	0.97:	*ns*	54 (45)	127 (59)	2.98:	$P < 0.005$
B	20 (14)	57 (17)	0.62, 1.49		36 (30)	52 (24)	1.75, 5.09	
C	5 (3)	10 (3)			8 (7)	12 (6)		
D	13 (9)	24 (7)			21 (18)	24 (11)		

Values are shown as *n* (%).

*Developmental delay was defined as a delay in meeting at least one developmental milestone (i.e., gross motor, fine motor, expressive language and receptive language skills).

Abbreviations: EEG, electroencephalography; SPD: School performance difficulties; CI, confidence intervals; *ns*, not statistically significant; na not assessed.

SPD grades: A = average grade; B = learning assistance required; C = below grade level or in a special class; D = attends a special school or is integrated with a 1:1 aide.

at a tertiary care centre. The SPD rate at the 2–4 year follow-up was statistically significantly greater in the seizure group compared with the non-seizure group (54% vs 47%; $P < 0.005$). Severity grades of SPD were also significantly different between groups with more children in the seizure group requiring assistance compared with the non-seizure group ($P < 0.005$) (Table 1). Furthermore, closer inspection of data from patients with SPD at follow-up, showed that more patients in the seizure group (23%) compared with the non-seizure group (11%) had a deterioration in SPD from baseline (Table 2).

With regard to the aetiology, there were fewer patients in the 'other identified aetiologies' group (19%) compared with the 'self-limited focal epilepsy' group (29%) and the 'genetic-generalised epilepsy' group (39%) who had no SPD at the initial assessment but had difficulties at follow up (Table 3).

Logistic regression analysis of data from patients with seizures at follow-up (n = 119) showed that neither age at initial EEG nor aetiology were predictive of SPD (Table 4). However, a high number (>30) of seizures ($P < 0.03$), high number of AEDs ($P < 0.01$) and presence of epileptiform abnormalities ($P < 0.04$) were significantly associated with SPD.

Adequate clinical data at follow-up were available for 69 patients with psychiatric diagnoses and no seizures. Of these patients, 22 (32%) had more than one psychiatric diagnosis. Overall, 38 (55%) children had anxiety disorders, 12 (17%) depressive disorders, 17 (25%) somatic symptom related disorders, 6 (9%) schizophrenia and other psychotic disorders, 6 (9%) disruptive, impulse-control and conduct disorders, 10 (14%) obsessive-compulsive and related disorders and 6 (9%) had other specified of unspecified mental disorders. In addition, there was one child (1%) in each group that had: patient feeding and eating related disorders; personality disorder (schizoaffective); bipolar and related disorders; trauma and stressor-related disorder (post-traumatic stress disorder).

No significant differences in the rate of SPD were observed between the seizure group and the psychiatric disorder group at the time of the initial EEG (37/119 [31%] vs. 30/69 [43%]) or at 2–4 year follow-up (64/119 [54%] vs. 38/69 [57%]).

Discussion

In this study, we found that children with new-onset seizures have a similar incidence and severity of SPD to children without seizures. However, over time, children with recurrent seizures or epilepsy, had a higher rate of SPD than children without seizures. Studies that have investigated the onset of SPD in children with epilepsy have found variable results. For example, similar

Table 2. Changes in school performance difficulties from initial EEG assessment to follow-up (2–4 years) for patients who had follow-up data.

School performance difficulties	Seizure group n = 119	Non-seizure group n = 215
Baseline (present) → Follow-up (absent)	0 (0)	4 (2)
Baseline (absent) → Follow-up (present)	27 (23)	24 (11)
Baseline (absent) → Follow-up (absent)	55 (46)	119 (55)
Baseline (present) → Follow-up (present)	37 (31)	68 (32)

Values are shown as *n* (%).
Abbreviations: EEG, electroencephalography.

Table 3. Changes in school performance difficulties from initial EEG assessment to follow-up (2–4 years) based on aetiology of patients with seizures (n = 119).

School performance difficulties	Self-limited epilepsy n = 21	Genetic-generalized epilepsy n = 13	'Other identified' seizure aetiology* n= 85
Baseline (absent) → Follow-up (present)	6 (29)	5 (39)	16 (19)
Baseline (absent) → Follow-up (absent)	11 (52)	7 (54)	37 (44)
Baseline (present) → Follow-up (present)	4 (19)	1 (8)	32 (38)

Values are shown as *n* (%).
Abbreviations: EEG, electroencephalography.
*Structural (genetic, infectious, metabolic, autoimmune, unknown).

Table 4. Logistic regression analysis for school performance difficulties at follow up among patients with seizures (n = 119).

Term	Odds ratio (95% CI)	Statistical significance
Number of seizures*: 1	Reference	_
Number of seizures: ≤5	4.78 (1.04, 34.63)	*ns*
Number of seizures: 6–30	5.41 (1.07, 41.06)	*ns*
Number of seizures: >30	6.95 (1.46, 51.63)	$P = 0.03$
Number of AEDs	1.71 (1.18, 2.86)	$P = 0.01$
Epileptiform abnormalities**: no	Reference	_
Epileptiform abnormalities**: yes	0.36 (0.14, 0.93)	$P = 0.04$
Age at initial EEG	0.94 (0.83, 1.06)	*ns*
Aetiology: Combined***	Reference	
Aetiology: SLE	1.26 (0.38, 3.94)	*ns*
Aetiology: GGE	1.65 (0.43, 5.98)	*ns*

Abbreviations: EEG, electroencephalography, CI, confidence intervals; AEDs, anti-epileptic drugs; SLE: self-limited epilepsy; GGE: genetic generalized epilepsy; *ns*, not statistically significant.
*Total number of seizures during follow-up period.
** This includes focal, multi-focal or generalized spike wave discharges on initial EEG.
***Structural (genetic, infectious, metabolic, autoimmune and unknown aetiologies).

to our findings, one study found no difference in academic achievement between children with new-onset seizures and controls with asthma at baseline.[6] However, by 24 months, the children with recurrent seizures experienced a decline in academic performance. Another study found no difference in academic achievement at baseline comparing children with new-onset seizures with sibling controls, although there were early differences in neuropsychological functioning.[22] However, a study that followed a cohort of children with new-onset seizures for five years found that special education assistance was required in 23% of children which started prior to seizure diagnosis, 5% between first seizure and diagnosis, and 30% after diagnosis.[23]

Factors that lead to SPD are multifactorial, and may involve an interplay of neurological, neuropsychological, age, AEDs, social and seizure variables. Interestingly, studies investigating early onset of seizures, frequent or persistent seizures, abnormal EEGs, as risk factors for academic underachievement have been inconsistent.[3,6,11,18,24–26] While we found that the age of seizure-onset was not

predictive of SPD, some studies have reported seizure-onset at a young age was associated with high rates of academic underachievement.[3,18,27,28] However, others have found no relationships between age at onset of seizures and neurocognitive scores.[11,29] In our study, we found that SPD at follow-up was associated with a high number of AEDs. Although it has been established that certain AEDs, including phenobarbital, benzodiazepines, and topiramate are associated with adverse cognitive effects, studies are limited on the effects of newer AEDs on cognitive function.[30–32] Interestingly, one study found that there was no difference in performance on cognitive and behavioural measures between children with new-onset seizures on short term treatment with AEDs (six months) and children with recently diagnosed diabetes mellitus.[33] The aetiology of the seizures is also important. Indeed, identified aetiology and developmental epileptic encephalopathy have been shown to be predictors of impaired adaptation and academic underachievement.[13,23,34] We observed that children with self-limited or genetic generalized epilepsies with normal school performance at baseline were more likely to develop SPD over time compared with 'other identified' aetiology. However, logistic regression analysis of these data controlling for SPD at baseline and using 'other identified' aetiology as a reference group did not find an effect of aetiology on SPD. Nevertheless, logistic regression analysis showed that the presence of epileptiform abnormalities was significantly associated with SPD.

Although previous studies have reported that children with epilepsy have more SPD than children with other chronic diseases,[2–6] comparisons to children with psychiatric diagnoses are limited. We observed that patients with seizures and psychiatric disorders (without seizures), had similar incidence and severity of SPD at baseline and over time. We were not able to stratify our psychiatric patients based on the type of disorder because of too few patients. However, the type of disorder may have a role on SPD. For instance, a study in 800 children followed from age 6–18 years externalizing (i.e., over-activity, poor impulse control, noncompliance and aggression) but not internalizing problems (i.e., anxiety, sadness, social withdrawal and fearfulness) predicted poor academic performance.[35] In addition in a cohort study of 400 children, investigators found that internalizing and externalizing problems from 6–8 years of age strongly diminished the chance of accomplishing a high-school degree.[36]

Limitations of our study included its retrospective design and the fact that standardized neuropsychological evaluations were not available for most patients. In addition, we used an arbitrary scale to assess school performance which may have lacked precision, but we believe it has practical value and clinical significance. Further studies will be required to test the robustness of our grading system. However, a strength of our study is that school performance and developmental skills were systematically obtained for all patients that required an EEG test at our centre and so we had access to a substantial database. Future, prospective studies in patients with new-onset seizures and psychiatric disorders, stratified by diagnosis subtype with detailed educational data are warranted to delineate precise school performance in this population.

In conclusion, we found that children with new-onset seizures had a similar rate of SPD compared with children without seizures. However, over time, children with recurrent seizures were at risk for more SPD than children without seizures. In a relatively small number of patients, we observed that children with seizures and those with chronic psychiatric disorders had similar rates of SPD. These findings

highlight the importance of regular assessments of school performance in both populations to facilitate early interventions. Prompt communication and collaboration between clinicians, families and teachers may enable these children to reach their optimal academic potential. Moreover, there is a significant need to develop effective interventions for children in these vulnerable populations who are facing difficulties in school performance.

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Declaration of Conflicting Interests

The authors declare that there are no conflicts of interest.

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