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Prevalence of Epileptiform Discharges in Healthy 11 and 12-Year-Old Children

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Abstract

We sought to determine the prevalence of interictal epileptiform discharges (IEDs) in healthy 11 and 12-year-old children. Sixth grade students with no history of seizure, or neurologic or psychiatric disease were enrolled in a longitudinal physical activity intervention study. Per study protocol, each student had two EEG recordings approximately 6 months apart. Epileptiform discharges were present in 4 (2.9%) of 140 students: centrottemporal in three and generalized in one. In three children the discharges were still present six months later. None of the children had developed seizures a minimum of one year after the second EEG. These results are consistent with those of two landmark European studies performed nearly a half century ago, before the modern era of digital EEG. Healthy 11 and 12-year-old children with no history of seizure may have centrottemporal or generalized epileptiform discharges on EEG, which can persist for at least 6 months. Based on both our results and those of the two prior European studies, such discharges, if found incidentally in otherwise healthy children in this age group, should not prompt further evaluation or treatment.

Keywords

EEG; pediatric; centrottemporal; generalized spike-wave

1. Introduction

Adolescents may have an EEG study for indications other than high suspicion of seizure. In such instances, interictal epileptiform discharges (IEDs) may be an incidental benign finding that is mistakenly interpreted as suggestive or even diagnostic of epilepsy. Knowing the pre-test probability of IEDs in healthy adolescents with no history of seizure would aid determining the clinical significance of such discharges in EEG studies of adolescent patients with low suspicion for seizure or epilepsy.

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The prevalence of abnormal EEG findings in carefully screened healthy children is difficult to determine. By definition such children are unlikely to undergo an EEG for clinical indications. EEGs obtained in research laboratories are rarely recorded with standard clinical EEG parameters or electrodes, and being research recordings are not reviewed for clinical abnormalities. Furthermore, subjects in EEG-related research are usually adults.

We recorded EEGs for research purposes in 140 healthy 6th grade students with no history of seizure, or neurologic or psychiatric disease. Each subject had two EEGs, approximately 6 months apart, recorded with a clinical EEG machine and standard International 10-20 system electrodes.

2. Methods

2.1 Subjects

Subjects consisted of an ethnically diverse sample of 140 adolescents at a public middle school in Southern California. Sixth grade students were recruited through school in four consecutive years (cohorts 1 - 4) as part of a prospective, longitudinal physical activity intervention study [1]. Exclusion criteria included left-handedness, playing on a sports team, inability to exercise, and history of head injury, neurological disease, seizure, asthma, and depression (a score that corresponded to moderate or severe depression as assessed using the Beck Depression Inventory [2] (cohort 1) or the Child Depression Inventory [3] (cohorts 2-4)). The main purpose of the EEG recordings was to test hypotheses in the biological psychology literature regarding associations between hemispheric asymmetry of frontal EEG power in the alpha band and specific personality traits. Subjects and a parent or legal guardian provided written assent and consent, respectively. The study was approved by the University of California Institutional Review Board.

2.2 EEG Data Acquisition and Review

EEGs were obtained with a *micro*EEG (BioSignal Group Corp., Brooklyn, NY), a miniature wireless EEG device, using all standard 10-20 system electrodes (except A1 and A2) embedded in a flexible nylon electrode cap (Electro-Cap International, Inc.) [4-6]. The sampling rate was 250 Hz and all electrode impedances were under 45 k Ω , an acceptable range for clinical recordings with the *micro*EEG device [4]. The EEG studies were reviewed with Persyst Insight II software (Persyst, Solano Beach, CA). The EEG recordings took place in a classroom that was turned into a clinical laboratory for the purposes of the parent physical activity intervention study. Each EEG consisted of four 4-minute segments alternating between eyes open and eyes closed, with 15 - 45 seconds for the subject to stretch and review instructions in between each segment, for a total duration of about 18 minutes. For the eyes-closed condition, subjects were instructed to sit comfortably and think about something interesting to help stay awake, as they were supposed to remain fully awake throughout the study. Each subject had two EEG studies approximately six months apart (EEG1, EEG2). All EEGs were reviewed by a board-certified clinical neurophysiologist (ACG).

2.3 Subjects with abnormal EEG studies

Per study protocol, a letter along with a clinical EEG report was sent to the parents of each subject with an abnormal EEG, encouraging them to have their child evaluated by a pediatric neurologist. These four subjects were followed by research staff for a minimum of one year after the second EEG.

3. Results

Subjects were 50% male and 50% Latino, 27% Caucasian non-Hispanic, 13% African-American and 10% Asian. Useful EEG data were not available for 13 EEG1 studies (hair styles incompatible with using an electrode cap [n=7], technically compromised studies [n=6]), and 15 EEG2 studies (hair styles incompatible with using an electrode cap [n=6], technically compromised studies [n=5], subject moved out of school district [n=3], subject removed from study [n=1]).

Mean subject age at first EEG was 11.5y (SD 0.41) and at second EEG was 12.0y (SD 0.40). Although subjects were instructed to remain awake, a substantial fraction had EEG patterns consistent with intermittent drowsiness. None of the subjects entered stage 2 sleep.

3.1 Abnormal EEG studies

Four subjects (2.9%) had abnormal EEG findings. Other than the abnormalities, all 8 EEGs included normal wake and drowsy patterns for age.

S1. 11.5 year-old boy. Occasional 0.5 – 2 second bursts of generalized ~4Hz spike or polyspike-wave discharges (Fig. 1a), increasing significantly in drowsiness. Occasional 2 – 5 second runs of occipital intermittent rhythmic delta activity (OIRDA, Fig. 1b).

S2. 11.1 year-old boy. Frequent right centrottemporal epileptiform discharges.

S3. 11.7 year-old girl. Occasional, independent, left and right centrottemporal epileptiform discharges, increased during drowsiness.

S4. 11.6 year-old girl. Occasional, up to 300 μ V (peak-to-peak) left centrottemporal epileptiform discharges, usually occurring in ~2 second runs of 5 – 6 discharges, increased in drowsiness (Fig. 2).

The abnormalities were present in both EEGs for all four subjects except S3, whose second EEG was normal. None of the subjects had had a seizure or were started on antiepileptic medication a minimum of one year after the second EEG.

4. Discussion

We found epileptiform discharges in 2.9% of healthy 11 and 12-year-old students with no history of seizure, or neurologic or psychiatric disease. The discharges were centrottemporal in three children (75%) and generalized in one (25%). The EEG studies were otherwise normal, i.e. there was no independent pathologic slowing. The discharges were present in follow-up EEGs performed 6 months later, except in one child with centrottemporal

discharges whose second EEG was normal. None of the children had developed seizures a minimum of one year after the second EEG.

Two landmark studies - both prior to the modern era of digital EEG - examined EEG findings in a large number of children screened to be free of neurologic or psychiatric disease or symptoms [7, 8]. Eeg-Olofsson et al. recorded 8-channel EEGs on 743 Scandinavian children aged 1 to 15 years [7]. The recordings included wake and sleep, as well as hyperventilation and intermittent photic stimulation. Definite IEDs were reported in 14 children (1.9%), and equivocal “spike-like” activity in an additional 0.5%. In 9 of the 14 children with definite IEDs the discharges were central or centrottemporal, and of the 104 11 and 12-year-old children, 3 (2.9%) had IEDs, all in the central region.

Cavazutti et al. recorded EEGs in 3,726 Italian children aged 6 - 13 years [8]. Studies were obtained in the awake state and included hyperventilation. The number of EEG electrodes and EEG duration were not specified. IEDs were reported in 3.5% of the entire group, as well as in 3.5% (n=8) of the 228 11-year-old children and 0.5% (n=1) of the 214 12-year-old children, for a combined total of 2.0% of 442 11 and 12-year-old children. In four of these nine children the discharges were “Rolandic-parietal” or mid-temporal, and in the remaining five they were generalized. Follow-up studies over a 9 year period revealed spontaneous disappearance of the EEG abnormalities in most subjects, with only seven of the 131 children with IEDs developing epileptic seizures, all of the generalized type (absence, myoclonic, generalized tonic-clonic).

Our data are remarkably consistent with those of these two studies performed 45 and 35 years ago [7, 8]. Taken together, ours and the earlier studies indicate that about 3% of healthy 11 and 12-year-old adolescents with no history of seizure or neurologic disease have incidental Rolandic or generalized IEDs. We presume that such IEDs are an inherited trait without phenotypic expression in this age group. Thus, in a patient in this age range *without* a history suggestive of generalized or Rolandic seizures and an associated age-related epilepsy syndrome, generalized and centrottemporal IEDs are likely to be an incidental and benign finding that warrants neither further diagnostic studies (e.g. brain imaging) nor an epilepsy diagnosis. However, in patients with such a history, the presence of such discharges should be interpreted as consistent with and supportive of the presumptive epilepsy syndrome diagnosis. Perhaps equally important, focal IEDs that are *not Rolandic* (i.e. maximal surface negativity in the region defined by electrodes C3/C4, T3/T4, and P3/P4) are extremely rare in healthy young adolescents, and if found incidentally should prompt an appropriate diagnostic evaluation, including at a minimum a comprehensive history and physical examination.

The impact of IEDs on cognition is controversial [9-11]. Generalized IEDs have been shown to transiently impair specific cognitive functions in some people with epilepsy (e.g. Krestel et al. [12]), while centrottemporal IEDs have been associated with cognitive deficits [10,13,14]. A very recent EEG-fMRI study demonstrated that in children with Rolandic epilepsy, centrottemporal IEDs disrupt the functional brain networks responsible for language, behavior, and cognition, and that both verbal and performance IQ correlated with IED-induced changes in dynamic functional connectivity in specific brain regions [14]. The

authors concluded that centrottemporal IEDs should be suppressed to reduce the risk of neuropsychological impairment. We do not think such an approach is justified in children *without* epilepsy and incidentally discovered generalized or centrottemporal IEDs because the effect of IEDs on cognition in people without epilepsy is unknown, in nearly all instances the IEDs are age-limited [8], and there is no medication that reliably suppresses IEDs, particularly focal IEDs, without its own cognitive side effects.

This study had at least two weaknesses. First, although large from a practical standpoint, the number of subjects was relatively small to measure and categorize a finding with an expected prevalence of only 3%. Second, the true prevalence of children with IEDs was probably underestimated due to the short duration of the EEG recordings (~ 18 minutes), the absence of stage 2 NREM sleep in all recordings, and the absence of drowsiness in most recordings. These limitations were an unavoidable consequence of the EEG studies being obtained with a protocol determined by the overriding research endeavor [1].

5. Conclusions

Approximately 3% of 11 and 12-year-old with no history of seizure, neurologic disease, or neurologic symptoms will have centrottemporal or generalized IEDs on a routine EEG study. Our data, combined with those of Eeg-Olofsson et al. and Cavazutti et al., suggest that such discharges are highly likely to resolve spontaneously, are unlikely to indicate increased risk of future seizures, and should be treated as a benign finding. Conversely, focal IEDs outside of the centrottemporal region are extremely rare in healthy children in this age group and warrant an appropriate diagnostic evaluation.

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References

1. Schneider M. Process evaluation and proximal impact of an affect-based exercise intervention among adolescents. *Transl Behav Med.* 2014; 4:190–200. [PubMed: 24904703]
2. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry.* 1961; 4:561–71. [PubMed: 13688369]
3. Kovacs M. The Children's Depression, Inventory (CDI). *Psychopharmacol Bull.* 1985; 21:995–8. [PubMed: 4089116]
4. Omurtag A, Baki SG, Chari G, Cracco RQ, Zehtabchi S, Fenton AA, et al. Technical and clinical analysis of microEEG: a miniature wireless EEG device designed to record high-quality EEG in the emergency department. *Int J Emerg Med.* 2012; 5:35. [PubMed: 23006616]
5. Grant AC, Abdel-Baki SG, Omurtag A, Sinert R, Chari G, Malhotra S, et al. Diagnostic accuracy of microEEG: a miniature, wireless EEG device. *Epilepsy Behav.* 2014; 34:81–5. [PubMed: 24727466]
6. Zehtabchi S, Abdel Baki SG, Omurtag A, Sinert R, Chari G, Roodsari GS, et al. Effect of microEEG on clinical management and outcomes of emergency department patients with altered mental status: a randomized controlled trial. *Acad Emerg Med.* 2014; 21:283–91. [PubMed: 24628753]

7. Eeg-Olofsson O, Petersen I, Sellden U. The development of the electroencephalogram in normal children from the age of 1 through 15 years. Paroxysmal activity. *Neuropadiatrie*. 1971; 2:375–404. [PubMed: 5171489]
8. Cavazzuti GB, Cappella L, Nalin A. Longitudinal study of epileptiform EEG patterns in normal children. *Epilepsia*. 1980; 21:43–55. [PubMed: 6766393]
9. Binnie CD. Cognitive impairment during epileptiform discharges: is it ever justifiable to treat the EEG? *Lancet Neurol*. 2003; 2:725–30. [PubMed: 14636777]
10. Nicolai J, Kasteleijn-Nolst Trenite D. Interictal discharges and cognition. *Epilepsy Behav*. 2011; 22:134–6. [PubMed: 21767995]
11. Vannest J, Tenney JR, Gelineau-Morel R, Maloney T, Glauser TA. Cognitive and behavioral outcomes in benign childhood epilepsy with centrotemporal spikes. *Epilepsy Behav*. 2015; 45:85–91. [PubMed: 25775975]
12. Krestel HE, Nirkko A, von Allmen A, Liechti C, Wettstein J, Mosbacher A, et al. Spike-triggered reaction-time EEG as a possible assessment tool for driving ability. *Epilepsia*. 2011; 52:e126–9. [PubMed: 21883181]
13. Wolff M, Weiskopf N, Serra E, Preissl H, Birbaumer N, Kraegeloh-Mann I. Benign partial epilepsy in childhood: selective cognitive deficits are related to the location of focal spikes determined by combined EEG/MEG. *Epilepsia*. 2005; 46:1661–7. [PubMed: 16190940]
14. Xiao F, An D, Lei D, Li L, Chen S, Wu X, et al. Real-time effects of centrotemporal spikes on cognition in rolandic epilepsy: An EEG-fMRI study. *Neurology*. 2016; 86:544–51. [PubMed: 26747882]

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Figure 1. Subject 1. a) 1 second burst of generalized 3-4Hz spike-wave. B) Two second interval of OIRDA.

Figure 2.
Subject 4. Four second burst of left centrotemporal IEDs.