Analysis of Neurodevelopmental Disorder in Special Child from EEG using Machine Learning: a Review

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Abstract: Angelman syndrome (AS) is a neurodevelopmental disorder caused by deletion on chromosome 15, uniparentaldisomy (UPD), imprinting defect, or UBE3A mutation. It is characterized by mental retardation with minimal speech and certain behavioral features. Assess the sleep quality and time in children with Angelman syndrome (AS) with sleep problems in using questionnaires and actigraphy, and in contrast to the Sleep settings in the typically developing (TD) children matched by age and gender. This study presents the largest objective set of sleep quality parameters in children with AS. Sleep quality is characterized by low efficiency and significant intra-and inter-individual variability, which requires further study. This paper, conducts the study about the Angelman syndrome (AS).

Keywords: Angelman syndrome, uniparentaldisomy, typically developing.

1. INTRODUCTION

Angelman syndrome is characterized by developmental delay, speech lack of motor impairment, epilepsy and a peculiar behavioral phenotype of happy demeanor (Dan 2008). It is caused by ube3a gene expression, the lack of which can lead to chromosome 15q11–q13-it's different disorders. Similar to chromosome 15q11-q13 disorders cause or Angelman syndrome, if they concern the chromosome inherited from the mother, or Prader-Willi syndrome (a clinically distinct condition, hypotonia, learning difficulties, obesity and hypogonadism) if they relate to chromosome paternal origin, illustrating the phenomenon of genomic imprinting. About 70% of patients, Angelman syndrome are due to a de novo 15q11-q13 micro deletion on the maternally inherited chromosome, which can be detected by fluorescence in situ hybridization (fish). Approximately 2-3% of patients inherit both copies of chromosome 15 father and no mother, i.e. paternal uniparental disomy. As a result, the functional copy of UBE3A from the mother is not inherited.These patients statistically show less severe phenotypes than deletion, larger head circumference, is less severe epilepsy and more words, but the final words is extremely limited. Another 3-5% of patients have the absence of a typical maternal pattern of DNA methylation. Phenotypically, they are indistinguishable from patients with uniparentaldisomy. There is a mutation in the maternal UBE3A gene in 5-10% of patients with high occurrence of private de novo mutations. Finally, no cytogenetic or molecular anomalies are found in up to 10% of typical cases. [1]

Compared to the surprisingly high-risk development of patients with Angelman syndrome, epilepsy can be analyzed for many conditions. In particular, early onset of intractable epilepsy in children with atypical abs and myoclonal seizures with a predisposition to seizure-free epileptic status is a common symptom. This may be due to a tendency to hyper synchronous neural activity, which may be associated with abnormal amino butyric acid (GABA)-mediated transmission due to the lack of ube3a expression or other factors. In recent years, there has been a growing understanding of the possibility of convulsive disorders in adult patients. [2]

Sleep is an important process for brain recovery and is important for maintaining cognitive function. Strong empirical evidence shows that sleep spindles promote learning, memory consolidation, declarative learning, motor skills, and plasticity that supports overall intellectual abilities [3].Cognitive functions associated with sleep spindles are also an important area of functional impairment in children with neurodevelopmental impairment (NDD). Clinically significant sleep disorders are common in children and adolescents with NDD. Among children with neurocognitive disorders, the effect of spindle formation of sleep disorders can be enhanced. As a result, NDD properties can increase both risk and vulnerability to abnormal sleep spindle formation[4].The purpose of this review is to analyze and integrate available data on the characteristics of uniform sleep in children with NDD and, if data are available, to examine the relationship between these characteristics and cognitive functions A better understanding of the relationship between sleep and NDD not only provides insight into pathophysiology and perhaps treatment of such disorders, but also provides improve our understanding of the relationship between sleep spindles and cognitive processes in children. It is likely that the characteristics of the sleep spindle represent a marker of brain development and functioning, as well as a Window into the underlying mechanisms that support cognitive activity.

1.1. Sleep Spindles in Developing Children

Sleep spindles represent an oscillatory electrical potential in the brain; they have a characteristic frequency of 11-16 Hz (usually 12-14 Hz in healthy adults) and last from one to several seconds [5]. In electroencephalography of the scalp (EEG), the spindle is often considered a sine wave in hieroglyphic morphology [6] crescendo-de-crescendo. Sleep spindles are characterized by their symmetry; synchrony between the hemispheres; the amplitude of peak-to-peak difference in the size of the spindle to reflect the voltage of the frequency is the number of waveforms per second density, the number of spindle bursts/min NREM sleep, and the last spindle of it. In

infants, the spindles sleep in the last few seconds duration, are in the high-alpha or low-beta frequency range, and the lack of synchrony in the front center, which is possible if not synchronized, is likely due to a lack of brain myelination in newborns. If the spindle is still asynchronous, the 2-year-old is considered anomalous [7]. Although sleep spindles in older children and adults are expressed diffusively throughout the head, as far as possible the central region and bilateral synchronicity and symmetry modes also sleep spindles can be divided into two different types based on their frequency and the expression in the field. Slow spindles (9 to<13Hz) [8] [9] occur maximally over the frontal regions, whereas fast spindles (>13-16Hz)domination over the central and parietal head regions, and usually precedes a slow spindles of hundreds of milliseconds. Slow spindles are typical of waxing and shrinking pattern, while fast spindles are mostly shrinking. This difference begins at about 2 years of age. Two residents of sleep spindles are thought to arise, and represent a variety of generators within the thalamus, a certain level of cortical involvement.

1.2. Children Sleep Spindle With Neurodevelopmental Disorder

Despite the high prevalence of NDD children's sleep problems and the wide range of cognitive deficits in this population, there is limited evidence regarding the function and function of sleep spindles. Some existing studies on the presumed association between sleep spindles and cognitive function have been done in children with intellectual disability, ASD, reading disability, and attention deficit hyperactivity disorder (ADHD), but the body of knowledge is often not always up-to-date, based on non-standard methods, but mainly on the basis of descriptive and correlated research interspersed, typical and even existing research has given us an initial view that sleep spindles and neurodevelopmental disorders or (in some studies), and intellectual performance.

1.3. Sleep Spindles In Children With Intellectual Disability

Intellectual disability is characterized by three characteristics: an intelligence quotient (IQ) of 75 or lower; significant limitations in adaptive behavior; and the onset of disability before the age of 18. In the past, the term “mental retardation" was used to describe this condition, but this term is no longer used. Common causes of mental retardation include genetic diseases, disorders of brain circulation during pregnancy, problems during childbirth, medical problems affecting brain health, and exposure to environmental toxins [10].

Sleep problems are more serious and more common in children with NDD than in children with normal development. Insomnia in normally developing infants is often based on behavior, whereas insomnia in children with Down's syndrome is often multifactorial, neurological, medical, and furthermore, insomnia in NDD tends to be chronic, and often lasts until adolescence or adulthood [12]. Quine showed that sleep problems remained in children with developmental disorders [13], Wiggs and Storz found that the average duration of sleep problems in these children was 7.13 years (SD4.04 years). [14]

Alarmingly high rates of sleep disorders were observed in children with intellectual disabilities: 86% - in children under the age of 6 years, 81% - between the ages of 6 and 11 years [13] and 77% - between the ages of 12 and 16 years [15].Nightespecially often there are difficulties in awakening and calming down affects more than half of children with NDD under the age of 16.The chronicity of these problems was illustrated by Quine[13], which found that half of children with NDD are sedentary problems and more than two-thirds of those with night wakefulnesswe still had problems 3 years later.ASD with children with sleep disorders is also very prevalent: a recent study reported that 40-85% of these children versus 20-40% of children developed normally[16-18].There are generally long-term sleep delays, including reported sleep disorders, anxiety and frequent sleep after waking at night, and reduced total sleep periods. With ASD aged 2-5 of fifty-3%of controls [19] of 32% compared with at least one sleep problem. ASD children often sleep more than 1 hour to take, a lot of people for 2 to 3 hours [20] can last night Wake you. Sleep problems with ASD tend to persist well past the middle stages of puberty. In the most common sleep problems under the age of 11-13,children with ASD, delayed sleep onset, frequent nighttime wakefulness and reduced sleep time[21].As for ADHD, 70% of children with this disorder were found to be mild to severe sleep problems[22]. The rate depends on ADHD subtypes, which in most prevalence are synthesized from sub [23] sleepiness, which can become more frequent in blunt subtypes [24]. In addition, both psychiatric coma and medications increase the prevalence of ADHD [23] sleep problems. Children and/or parents do not have the sleep problems inherent in ADHD, much more than bedtime resistance, difficulty in developing, sleep at night, difficulty in waking up in the morning, sleep breathing problems and healthy controls, but the most commonly reported problem is harder to sleep [25].

1.4. Intellectual Disability Children With Sleep Spindles And Intellectual Performance

In a series of studies [26] [27], shibakaki et al. Functional levels were classified according to the developmental index (DQs) of the participants. The Tumor questionnaire and the insane questionnaire for infants and young children were carried out, and they were divided into severe, moderate, and slight intellectually handicapped. They found that children with intellectual disabilities frequently have no sleep spindles, including children with intellectual disabilities that are associated with a range of disorders (eg, congenital brain dysplasia, hydrocephalus, Rubinstein-Taybi syndrome, Down syndrome, and chromosomal abnormalities), and these children had no sleep spindles of the shorter or no sleep spindles compared to those with a significant increase in brain waves and a higher incidence of longer sleep spindles [28].

2. LITERATURE REVIEW

Z. V. Okudan, Ç.d. Ozkara [29], the author provides an ongoing review of seizure factors and the management of reflex seizures. Reflex epilepsy (REs) is identified as epileptic seizures consistently arise to personal identification, goals, and specific triggers that can be this sexual stimulation, or the patient's own activity. Sub-type stimuli depending on the properties different. C. zkara, A. e. Gündüz, T. C¸kun [30], the clinical and electrophysiological features of temporal evolution presented by two siblings with mature onset

NCL and homozygous mutations in the CLN6 gene. This paper is a clinical and electrophysiological study results depicting the evolution of order medical records and electrophysiology data analyzed. The study of electrophysiology included multiformatics analysis of myocardial and cerebrospinal fluid reflexes as well as routine EEG and imaging EEG. R. S. Fisher [31], this review presents the newly developed International League against Epilepsy (ILAE) 2017 classification of seizure types. Classification allows for a new type of focal seizures and a few new generalized convulsions, and explains the terms that are used in anticonvulsant name.

D. C. Wallant, P. Maquet and C. Phillips [32], the sleep spindle is defined physically and topographically for the first time. From this General description, first extract the main characteristics to be detected and analyzed. There is an inexhaustible list of methods for automatic detection of spindles and a description of the main principles of their processing. E. Ferlazzo, C. Sueri, M. Elia, and T. D. Agostino [33], describes the unique patients of both as and trisomy and presents them with spontaneous and reflex drug resistant seizures. Yvonne M.Y. Hana, A. S. Chanba [34], this study examined the cortical connections of children with executive function and autism spectrum disorder (ASD) and examined the executive function exhibited by these children. M. Lowrie, C. Bessant, R. J. Harvey [35], the author study targeted feline auditory reflex seizures (FARS). An online questionnaire was created to capture information about cat owners suffering from FARS. The vet's medical history and contrast it's only been ninety-six cats were also included.

R.L. Moseley, R.J.F. Ypma [36], they observed that total brain function connectivity was highest in control, intermediate in sibling, and lowest in task and resting states in ASC. A visual processing and default mode network, such as a specific network to the local terminal phenotype effect, to be finalized and further added. The analysis is the first to show that the connection of total brain functional decline is the phenotype of the end of autism in adolescence, and therefore the association between ASC and the blue-green color of their kin. L. G. BieMertz and P. Thaulov [37] showed a marked decrease in developmental age in terms of visual, receptive, and expressive language when compared to those with UBE3A mutations and PUPD. Within all subgroups, expressive language performance was significantly reduced when compared to the receptive performance. S. E. Goldman, T. J. Bichell [38],Sleep anxiety is more common in children with Angelman syndrome, with 20-80% of individuals having a reduced sleep need and/ or abnormal sleep wake cycles. The effects of these sleep behaviors on parental sleep and stress is not known. Through standardized questionnaires, wrist actigraphy, and the use of polysomnography, they found that parents ' sleep behavior and parental stress levels with Angelman syndrome were significantly higher.

R. Coben, A. R. Clarke [39], autism spectrum disorder (ASD) is defined as a neurodevelopmental disorder with associated deficits in executive function, language, emotion, and social function. ASD is associated with pathophysiology in cerebral structural tissues. In the present study, we investigated the quantitative brain wave findings of two children diagnosed as autistic disorder compared with 20 controls, age, and EEG matched to gender, recorded at rest with ocular closure eye, and investigated the topographic differences in brain function using absolute, relative, total power, and inferred coherence within and between hemispheres. The literature survey shown that the Angelman syndrome (AS) was the neurodevelopmental disorder which made the overlap of the phenotype to be the features of intellectual disorder, speech and movement disorder, epilepsy, abnormal sleep, autism. Individuals with as display characteristic EEG patterns include high amplitude rhythm delta waves. This paper studied these EEG readouts of children with autism, motivated by a study of functional connectivity and sleep spindles.

3. DISCUSSION

The goal of this paper is to review and integrate the association (if possible) of cognitive function with available evidence of sleep spindle characteristics in NDD children. Before we try to integrate the findings across different obstacles, we should note the critical methodology limitations of existing work. Many of the studies reproduced have small sample sizes and even overestimated the impact size.

This becomes even more difficult if the researchers used data from the same participants in multiple publications.Another limitation of the sample is heterogeneous. First, clinical heterogeneity is unique to each of the NDD discussed. In addition, many studies have put together participants in terms of different developmental stages of puberty, whether to sleep, so they can suffer from developing heterogeneity. Heterogeneity can also be raised from other information levels and/or the inclusion of comorbid status participants. This kind of causes the results to other settings and circumstances that can be generalized about whether question. Other problems are difficult to directly compare the results can be a research of methodological differences. For example, in some studies, brainwave patterns were recorded during the full night of sleep, others were induced by chloral hydrate during the day, normal clinical electroencephalography or in the latter case, it was not possible to eliminate the medication effect, the time spent on stage 2NREM sleep was limited, and less sleep spindle was detected. These noted limitations may be an inherent challenge in the study of sleep spindles and cognition in children with NDD. Given the prevalence of disorders and their clinical nature, it can be difficult to obtain a larger, more homogeneous group. Both technologically and financially, it challenges the management, oftentimes the electrodes are difficult to tolerate, and you may dislike being in an unfamiliar environment, and cognitive testing, therefore, a practical problem poses a real barrier to the feasibility of large-scale, homogeneous studies that use objective measures of sleep and cognition. Our review consolidates existing data related to sleep spindle characteristics in children with NDD and seeks to correlate these differences with cognitive processes.

4. CONCLUSION

The study generated the largest objective data set of sleep quality parameters in children with AS. The quality of sleep was characterized by high variability, both among children as a sleep disorder reported by parents and at night for individual children. This variability is further underlined by the questionnaire report of parents over the seven-night period, and unlike those seen in children

with a typically developing on a weeknight, the change in total sleep duration is due to a lack of environmental and social constraints on children with AS.

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