Sleep Medicine 38 (2017) 24–30

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Sleep and slow-wave activity in depressed adolescent boys: a preliminary study

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ARTICLE INFO

Article history:
Received 23 December 2016
Received in revised form
22 June 2017
Accepted 23 June 2017
Available online 25 July 2017

Keywords:
Sleep
Depression
Adolescence
Slow-wave activity
Slow-wave activity dissipation
Polysomnography

ABSTRACT

Objective: Adolescence is a vulnerable period of life that is characterized by increasing incidence of depression. Sleep disturbance is one of the diagnostic symptoms of depressive disorder. Adolescence is also characterized by dramatic maturational changes in sleep and its regulation. The goal of this study was to assess sleep macroarchitecture and slow-wave activity (SWA) in depressed adolescent boys.
Methods: Eight non-medicated adolescent boys meeting the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria for depressive disorder and 10 age-matched healthy controls (average age 16.0 years) underwent polysomnography in their home environment for two consecutive nights. Sleep macroarchitecture, SWA, and SWA dissipation were assessed in all subjects.
Results: Depressed boys showed a flattened pattern of SWA dissipation through the night. SWA power was lower during the first non-rapid eye movement (NREM) episode in the frontal derivation and higher during the third NREM episode in the central derivation in the group of depressed boys as compared to healthy boys. The SWA dissipation pattern correlated with the severity of depressive symptoms, and the correlation was strongest in the frontal derivation. In addition, total sleep time was shorter in patients as compared to the control group, but no other differences were found in the macroarchitecture of sleep.
Conclusion: Depression in adolescent boys is characterized by more evenly distributed SWA through the night as compared to healthy subjects, and we showed for the first time that this pattern of SWA distribution is associated with severity of depressive symptoms. These findings suggest that homeostatic regulation of sleep may be impaired in adolescent depression.

1. Introduction

The incidence of both depressive disorder and insomnia increases rapidly across adolescence [1–5]. According to epidemiological research, the prevalence of major depressive disorder (MDD) among adolescents ranges from 5 to 12% [1,6–8], and the prevalence of insomnia is about 11–13% [4,9]. Depression during adolescence frequently recurs or persists into adulthood, which, in addition to causing considerable human suffering, induces a substantial economic burden for society [3,10–12].

Sleep disturbance is one of the diagnostic symptoms of depressive disorder. In depressed adults, sleep is characterized by a range of changes in sleep structure [13], which can be divided into three groups [14]: impaired sleep continuity (prolonged sleep latency, increased number of night awakenings), rapid eye movement (REM) sleep disinhibition (shortened REM sleep latency, elevated REM sleep frequency and duration), and non-rapid eye movement (NREM) sleep changes (decreased NREM sleep duration and reduced slow-wave activity (SWA)). Moreover, different REM and NREM sleep parameters have been previously shown to correlate with the severity of depressive symptoms in adults [15].

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http://dx.doi.org/10.1016/j.sleep.2017.06.029
1389-9457/

Depressed adolescents frequently complain about their sleep [16,17], but the results from objective polysomnographic studies remain controversial. Some polysomnographic studies in depressed adolescents have shown the same sleep alterations which are consistently found in depressed adults [18–23], but others have failed to demonstrate any abnormalities in sleep architecture [24–26]. The differential findings can be explained by, for example, small sample sizes and the heterogeneity of studied samples in terms of gender, age/pubertal status, and the variable clinical features/symptoms of depression (such as inpatient/outpatient status, imposed vs free sleep schedules, comorbid psychiatric disorders, medication use).

Adolescence is characterized by dramatic maturational changes in sleep and its regulation [27–29]. The most remarkable change in sleep architecture during typical adolescence is the reduction in the amount of SWS (by up to 40%) and SWA (by up to 60%) [28,30–34]. The changes in sleep occur during adolescence go hand in hand with major maturation of the psychosocial and physical processes, including a massive reorganization of the brain neuronal networks (synaptic pruning) [27]. It has even been suggested that the observed decline in SWA across adolescence can be explained by a reduction in synaptic density [35,36]. Thus, sleep, and particularly SWA, and brain maturation during adolescence are tightly interconnected.

To date, the literature regarding SWA changes in adolescent depression is scarce. Currently, there are only a few studies regarding SWA abnormalities in NREM sleep of depressed adolescents. In one mixed-gender study a lower delta power in the first NREM sleep episode and an irregular SWA dissipation through the night in depressed adolescents boys compared to healthy subjects have been observed [20]. In the only study using a homogeneous sample in terms of age, gender and medication use, a lower delta amplitude and power has been shown in depressed adolescent girls as compared to healthy girls [37]. Recently, it has also been shown that the topographical pattern of SWA distribution in depressed adolescents is characterized by increased SWA over the frontal cortex compared to healthy controls [38]. However, currently there are no studies that measure SWA abnormalities using a homogeneous sample of depressed boys. Moreover, none of the previous studies have looked at the association between severity of depressive symptoms and SWA dissipation.

Therefore, the aim of our study was to examine sleep macroarchitecture, SWA power, and SWA dissipation and their relationship with depression severity in a sample of non-medicated depressed adolescent boys as compared to a healthy control group. Furthermore, as an additional marker of sleep homeostasis, we calculated the rise rate of SWA during the initial stage of sleep, which has been shown to be associated with homeostatic sleep pressure [39].

2. Materials and methods

2.1. Participants

A total of 20 (10 patients and 10 healthy controls) non-medicated adolescent boys aged between 14 and 17 years were recruited for a research project focusing on adolescent depression, sleep, and brain maturation (the ADSLEEP project). Patients suffering from depressive and/or sleep symptoms were recruited from the Helsinki University Central Hospital Department of Adolescent Psychiatry outpatient units, and healthy controls were recruited via advertisements in a newspaper for the hospital staff. Exclusion criteria for all participants consisted of mental retardation, insufficient knowledge of the Finnish language, current use of medication, age over 17.5 or under 14.5 years, chronic somatic illness, substance abuse/dependence, principal Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) diagnosis other than depressive/sleep disorder.

All subjects underwent detailed clinical and psychiatric evaluation. All adolescents were free of psychotropic and other medication during the study, and the presence of somatic conditions and structural brain pathologies were ruled out based on brain magnetic resonance imaging (MRI) and blood samples. One patient was excluded from the analyses presented in this paper because he was diagnosed with a circadian rhythm sleep disorder only.

Nine patients were diagnosed with depressive disorder according to the DSM-IV. Diagnostic assessment was performed with the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime version (K-SADS-PL) by one of the authors (A.S.U.) [40]. Depression symptom severity was further evaluated with two different scales: the self-administered, 21-item Beck Depression Inventory (BDI-21) [41], and the Hamilton Depression Rating Scale (HDRS), administered by one of the authors (A.S.U.) [42]. Insomnia symptoms were assessed by the Athens Insomnia Scale (AIS) [43]. Polysomnographic data were not available for one patient due to drop out, leaving eight patients and 10 control subjects in the analyses of the current study.

The study protocol conformed to the Declaration of Helsinki and was approved by the ethics committee of the Helsinki University Central Hospital. Written informed consent for study participation was received both from the participants and their parents or legal guardians.

2.2. Sleep recording and scoring

Polysomnographic recordings were conducted in subjects' home environments with ambulatory recording devices (Embla, Flaga Hf. Medical devices; electroencephalogram (EEG) positions according to the International 10–20 system; derivations F4-M1, C4-M1, O2-M1 and backup derivations F3-M2, C3-M2, and O1-M2; sampling rate 200 Hz) for two consecutive nights. EEG, electrooculogram, and electromyogram were recorded according to standard criteria and the whole recording period was manually scored for sleep stages in 30-s epochs by a certified sleep technician blinded to the patient/control status of the subjects. Night 1 served as an adaptation night and night 2 has been used for the sleep and power spectral analyses presented in this paper.

Total sleep time (TST), sleep efficiency (time asleep relative to sleep period, which was calculated as time from sleep onset through last epoch of sleep), SWS and REM sleep latencies (time from sleep onset until first SWS or REM sleep episode, correspondingly), and number of awakening episodes were calculated from the scored data.

NREM periods used for power spectral analysis were determined as a succession of sleep stages 1, 2, and 3 with a duration of 15 min or more and terminated by REM sleep or wakefulness of least 5 min. No minimum REM sleep duration was required for the first REM sleep episode. Only the first three episodes of NREM sleep were included in the power spectral analysis, because all the subjects had at least three NREM sleep episodes during the night.

2.3. Power spectral analysis

The EEG (sampling rate 200 Hz) was subjected to spectral analysis off-line using a fast Fourier transform (FFT) routine with the help of Spike 2 software (version 8.07 CED, Cambridge). Power spectra (Hanning window) from central C4-M1 and frontal F4-M1 channels were computed, using FFT size of 512 Hz giving a resolution of 0.39 Hz. The spectral power was averaged in 30-s epochs to be of identical length with the stage-score epoch length. Power