Sleep Medicine 56 (2019) 41–46

Contents lists available at ScienceDirect

Sleep Medicine

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

Original Article

Sleep architecture in adolescents hospitalized during a suicidal crisis

Addo Boafo [a], Roseanne Armitage [b], Stephanie Greenham [a, c], Paniz Tavakoli [a], Alyson Dale [c], Ashley Nixon [c, d], Alexandre Lafrenière [d], Laura B. Ray [d], Joseph De Koninck [c, d], Rébecca Robillard [c, d, *]

[a] Children's Hospital of Eastern Ontario, Ottawa, ON, Canada
[b] Department of Psychiatry (Retired), University of Michigan, Ann Arbor, MI, USA
[c] School of Psychology, University of Ottawa, ON, Canada
[d] Sleep Research Unit, The Royal's Institute of Mental Health Research, Ottawa, ON, Canada

ARTICLE INFO

Article history:
Received 5 October 2018
Received in revised form
21 December 2018
Accepted 25 December 2018
Available online 9 January 2019

Keywords:
Adolescence
Suicide
Depression
Hospitalisation
Sleep
Polysomnography

ABSTRACT

Objective/background: Rates of suicide attempts in Canadian youths are concerning. Adolescence is a sensitive period for the emergence of both sleep and mood problems, two major risk factors for suicidality. This naturalistic study aimed to define the sleep profile of adolescents under the combined influence of suicidality, depression and pharmacotherapy during hospitalization for a suicidal crisis.
Patients/methods: Seventeen suicidal adolescents (15.0 + 1.2years, 82% females) with major depression were recruited from a Canadian pedopsychiatric inpatient unit. Seventeen non-depressed adolescents were retrospectively collated from another database (15.0 + 1.1years, 83% females). None of the participants had a history of sleep disorders or significant medical conditions.
Results: Compared to controls, suicidal adolescents had a longer sleep onset latency ($Z = -4.5$, $p < 0.001$), longer REM latency ($Z = -3.2$, $p = 0.001$), higher percentage of NREM1 sleep $t(33) = -2.6$, $p = 0.020$), and higher REM density ($Z = -2.8$, $p = 0.004$) than controls. Higher REM density correlated with higher CDI-II scores ($r = 0.55$, $p = 0.27$) A significant interaction indicated that the two groups had similar NREM3 percentages in the first two-thirds of the night, but that the suicidal group had significantly lower NREM3 percentage than the controls in the last third of the night ($F(2,66) = 3.4$, $p = 0.041$).
Conclusions: Significant sleep abnormalities were observed during hospitalization for a suicidal crisis in a sample of depressed and mostly medicated adolescents. This included sleep initiation and REM sleep latency abnormalities, shallower sleep and high REM density. Future studies should decipher the relative effects of depression, suicidality and medication on sleep. These findings stress the need to address sleep disturbances in the management of suicidality in adolescents.

1. Introduction

Suicide is the second leading cause of death in young Canadians [1], and rates of completed suicide have increased in children and adolescent females [2]. In fact, about 90% of the 350–400 yearly admissions of adolescent inpatients to the psychiatric unit of one of the major pediatric hospitals in Canada present with high risk suicide ideation and behaviour [3]. Anecdotally, many of those young inpatients report significant sleep problems, an observation which echoes reports of disturbed sleep in hospitalized adults [4,5]. Along the same lines, poor sleep quality during psychiatric hospitalization has been linked to worse clinical state in children [6]. The sleep profile of adolescents hospitalized for a suicidal crisis is likely to be influenced by a unique constellation of factors including not only the effects of suicidality and depression, but also the acute effects of sleeping in a new unfamiliar environment (often with strict bedtime/wake-up schedules, constant lights in adjacent corridors, and higher risks of noize), and the introduction of new psychotropic medications or changes in pre-existing medications. Although sleep may play a role in recovery processes during this critical period, little is known about the sleep profile of adolescents hospitalized during a suicidal crisis.

As part of the normal developmental changes from childhood to adolescence, there is a reduction in sleep duration mostly driven by delayed sleep onset times [7]; Ivo [8] and a shortening of rapid eye

* Corresponding author. The Royal's Institute of Mental Health Research, 1145 Carling Ave, Ottawa, Ontario K1Z 7K4, Canada. Fax: +1 613 761 3605.
E-mail address: Rebecca.Robillard@uottawa.ca (R. Robillard).

https://doi.org/10.1016/j.sleep.2018.12.018
1389-9457/

movement (REM) onset latency [9]. While SWS and slow wave activity (SWA; eg spectral power in the 1–4 Hz frequency band) are markedly high in children and teenagers, adolescence marks the start of a progressive decline towards adult amounts of SWS and SWA [9–11]. Changes in SWS and SWA across the night are reflective of homeostatic sleep regulation via which the sleep pressure accumulated during wakefulness dissipates across the next sleeping episode. A slower accumulation of homeostatic sleep pressure (as reflected by SWS/SWA dynamics) with similar rates of homeostatic sleep pressure dissipation [12,13] have been noted in adolescents. Importantly, there are indications that age-related changes may influence the sleep profile linked to mood disorders [14–19]. Compared to healthy individuals from the same age group, young (ie mean ages ranging between 15 and 20 years old) people with major depression often have more delayed and irregular sleep-wake schedules, a longer sleep onset latency, and more wake after sleep onset [15,20–22]. The increase in REM pressure typically seen in adults with depression is less prominent in adolescents [23], but some studies still reported short REM latencies and increased REM sleep (eg Ref. [24]. Alterations in SWS are also inconsistent: many studies failed to identify significant differences between adolescents with depression and healthy controls, but some reported increased SWS in depression (eg Ref. [25] and others noted lower SWA at the beginning of the night and irregular SWA dissipation across the night [26].

Despite the strong relationship between depression and suicidality, the effects of depression on sleep does not seem to fully explain the link between sleep disturbances and suicidality. Epidemiological work and meta-analyses concluded that, independently from depression severity, insomnia symptoms such as sleep initiation and maintenance difficulties, short sleep duration, and nightmares are all associated with increased risk of suicidal thoughts and behaviours in adults [27–29]. In adolescents, similar findings have been reported [30–37], but some studies indicated that both short and long self-reported sleep durations are linked to increased suicidality [38–41]. Furthermore, longitudinal studies in large community samples of adolescents revealed that complaints of sleep initiation or maintenance difficulties are predictive of subsequent suicidal ideation and attempts [42,43], strengthening the hypothesis of a causal role of sleep disturbances. While these important studies were based on subjective sleep reports, little evidence is currently available about how objective sleep measures relate to suicidality.

Two studies in adults with depression reported that, compared to people with low suicidality, those who did attempt suicide or had significant suicide ideation, had shorter REM latency, spent more time in REM sleep, had more phasic REM during the second REM period, and had less delta wave counts towards the end of the night in the fourth NREM period [44,45]. One study compared polysomnography data in 13 adolescents with major depression and suicidal ideation (eight of which had made a suicide attempt and the remaining five had reported a suicide plan), 14 depressed non-suicidal adolescents and 30 healthy controls [46]. While no significant sleep differences were found between the non-suicidal adolescents with depression and healthy controls, the subgroup with elevated suicidality had significantly longer sleep onset latency and mild trends for shortened REM latency, more REM sleep and increased REM density. Interestingly, they also found in the overall group with depression that SWS was significantly lower in those patients with complaints of insomnia and significantly higher in those with complaints of hypersomnia. Another study in 20 non-medicated adolescent outpatients with depression showed that higher scores on the suicidality item of the Hamilton Depression Rating Scale were significantly correlated with lower percentage of SWS, and higher total sleep time [47]. However, this was based on secondary exploratory analyses from the placebo condition of a pharmacological intervention study.

Overall, there is a need to expand knowledge about the sleep electroencephalography (EEG) profile of adolescents with suicidal ideation or behaviors, notably in clinical populations undergoing a full blown suicidal crisis. In typical cases, the sleep of these adolescents is most likely to be affected not only by suicidality, but also by depression, pharmacotherapy and the fact of sleeping in a psychiatric facility. The present naturalistic study aimed to define the sleep profile of adolescents under the combined influence of these factors during hospitalization for a suicidal crisis. It was anticipated that most adolescents with depression who are hospitalised for a suicidal crisis would be taking psychotropic medications. It was predicted that they would exhibit increased sleep onset latency, abnormal SWS dynamics across the night, and that their overall SWS would vary according to subjective sleep duration in the week preceding the study. It was however postulated that they would have few REM sleep abnormalities due to the suppressing effects of antidepressant medications.

2. Methods

2.1. Participants

Sample characteristics are reported in Table 1. Seventeen adolescents (12–17 years old) with a diagnosis of major depression (based on – criteria from the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)) who were admitted for acute risk of suicide (ie, suicidal group) were recruited from the Inpatient Psychiatric Unit of the Children's Hospital of Eastern Ontario (CHEO), one of the major pediatric hospitals in Canada. All participants from the suicidal group had reported a plan to kill themselves with the full intention of dying, 16/17 had made a suicide attempt. At the time of admission, suicidal risk was judged to be too high for these adolescent to safely be in the community. Eighty-eight percent (n = 15) of these patients started new psychotropic medications or underwent significant changes in medication dosage following admission (ie a few days before the study). Ninety-four percent (n = 16) of these patients were taking at least one psychotropic medication at the time of polysomnography (Table 1). Three participants started antidepressant medication less than 2 weeks prior to polysomnography (across the entire sample, antidepressants or mood stabilizers were initiated 1–300 days before polysomnography). A sample of 17 age- and sex-matched

Table 1
Sample characteristics.

	Control	Suicidal
Sex Distribution (n (%) Females)	14 (82.4)	14 (82.4)
Age (years; mean(SD))	14.9 (1.1)	15.0 (1.2)
Medication Intake (n (%))		
Antidepressants	0 (0%)	15 (88.2%)
SSRI	0 (0%)	13 (76.5%)
SNRI	0 (0%)	1 (5.9%)
SARI	0 (0%)	5 (29.4%)
Mood Stabilizer	0 (0%)	1 (5.9%)
Melatonin	0 (0%)	4 (23.5%)
Stimulants	0 (0%)	1 (5.9%)
Atypical Antipsychotic	0 (0%)	4(23.5%)

n(%) Females: Number and percentage of females. Medication Intake n(%): Number (percentage of total sample) of participants taking at least one subtype of medications. SSRI: Selective serotonin reuptake inhibitor (Fluoxetine (n = 4), Sertraline (n = 5), Escitalopram (n = 4), Citalopram (n = 1)); SNRI: Serotonin-norepinephrine reuptake inhibitor (Venlafaxine (n = 1)); SARI: Serotonin antagonist and reuptake inhibitor (Trazodone (n = 5)); Mood Stabilizer (Lithium Carbonate (n = 1)); Stimulants (Methylphenidate (n = 2)); Atypical Antipsychotic (Quetiapine (n = 1), Aripiprazole (n = 3)).

non-depressed adolescents (ie, control group) was retrospectively collated from an existing database from the University of Michigan (UM) Sleep & Chronophysiology Laboratory. All participants from the control group were asymptomatic on the Children's Depression Rating Scale-Revised (total score < 65) [48] and on the Suicide Ideation Questionnaire-Junior (total score < 31), had no personal or first-degree family history of psychopathology, no personal history of sleep disorders, and no lifetime diagnosis of anorexia or bulimia, or substance abuse in the last six months. None of them was taking psychotropic medications. Across both groups, none of the participants: were taking benzodiazepines, sedatives or hypnotics, sustained a head injury or a loss of consciousness for more than 5 minutes, or had a history of a significant medical condition. All participants (and their parents for those younger than 16 years of age) provided written informed consent. This study was approved by the Research Ethics Board of the Children's Hospital of Eastern Ontario.

2.2. Procedures

2.2.1. Clinical assessment

The admitting psychiatrist or psychologist documented medication intake at the time of the study and administered the Children's Depression Inventory-II (CDI-II). The CDI-II is a 28-item self-report inventory that assesses the presence and severity of depressive symptoms in children aged 7–17 years [49]. Each item is rated on a 3-point scale ranging from 0 (none) to 2 (definite) for the previous two-week period. A clinical cut-off score is established with T-scores of 65 or above based on sex and age specific norms. The CDI-II has shown good internal consistency ($\alpha = 0.73$-0.91.). Participants were also asked to fill out the Suicide Behaviors Questionnaire-Revised (SBQ-R), which has four items each tapping a different dimension of suicidality: lifetime suicide ideation and/or suicide attempt; frequency of suicidal ideation over the past 12 months; threat of suicide attempt; and self-reported likelihood of suicidal behaviour in the future. The total SBQ-R score ranges from 3 to 18. In adult psychiatric inpatients the cut-off score is 8 with sensitivity of 80% and specificity of 91% [50]. Participants also filled out a retrospective sleep log to document estimated sleep-wake schedules and subjective sleep quality and quantity for the weekdays preceding the study (excluding week-ends). Sleep log, CDI-II and SBQ-R data was missing from one participant from the suicidal group.

2.2.2. Polysomnography

All participants underwent two polysomnographic recordings according to their individual habitual sleep-wake schedules: the first one was used as an adaptation night, and the second was used for the final analyses. For the suicidal group, the polysomnography data were acquired in the patient's private room inside the Inpatient Psychiatric Unit at CHEO using the MediPalm 22-Channel polysomnography amplifier (Braebon Medical, Ottawa, ON). Since in this ward, all bedroom doors have an indoor window, nighttime checks did not interfere with sleep. For the control group, polysomnography data were acquired in the UM Sleep & Chronophysiology Laboratory using the Astro-Med Grass P511 A/C amplifier (Natus, Pleasantan, CA) set at a sensitivity of 5 (50 μV, 0.5 s calibration), corresponding to a gain of 50,000. The low- and high-bandpass filters were set at 0.3 and 30 Hz. A 60-Hz notch filter was used to attenuate electrical noise.

Electroencephalographic (EEG) data (F3, F4, C3, C4, P3, P4, O1 and O2), ground and reference channels, right and left electrooculograms (EOG), two chin and leg electromyograms (EMG), and two electrocardiogram (ECG) channels were collected. On the adaptation night, respiration was monitored with an airflow cannulae (pressure transducer) or a nasal-oral thermistor, as well as chest and abdomen plethysmography. One single registered sleep technologist manually scored sleep stages for all participants across both groups according to guidelines established by the American Academy of Sleep Medicine [51] on Stellate Harmonie (Natus, Pleasantan, CA) analyses software. The sleep variables of interest obtained from polysomnography were: total sleep time, sleep onset latency, REM sleep latency, wake after sleep onset (WASO), and percentage of time spent in Stage 1 (N1) sleep, Stage 2 (N2), slow wave sleep (N3), and rapid eye movement (REM) sleep. Total sleep time was calculated as the total time spent asleep between “lights off” and “lights on” tags. WASO was defined as epochs following sleep onset that were scored as wake. Sleep stage percentages were calculated as the percentage of time between “light off” and “lights on” scored as N1, N2, N3, and REM divided by TST. Rapid eye movements were automatically scored on EOG channels during REM sleep and using Stellate Harmonie (Natus, Pleasantan, CA) analyses software. Rapid eye movement density was calculated as the total number of rapid eye movements per minute of REM sleep.

Statistical analysis

Outlying values (above or below 2 standard deviations from the mean) were replaced by curtailing. Across all variables, no more than one data point was outlying. The normality of data distribution within each group was assessed with the Shapiro–Wilk test. Independent sample t-tests were used for group comparisons for all normally distributed variables, and Mann–Whitney tests were used for variables which were not normally distributed. In order to assess group differences along the course of SWS dynamics across the night, percentages of N3 sleep were submitted to a mixed ANOVA with one independent factor (Suicidal versus Control group) and one repeated measure (time: first, second, and third thirds of the sleep period). Within the suicidal group, two-tailed Pearson correlations were conducted to evaluate potential correlations between: i) SWS (in the first, second, and third thirds of the sleep period) and subjective sleep duration in the week preceding the study, and ii) all polysomnography variables and scores on the SBQ-R and the CDI-II.

3. Results

3.1. Depression severity and suicidality in the suicidal group

Within the 16 participants from the suicidal group who had valid clinical data, depressive symptoms severity on the CDI-II ranged from 11 to 43 (mean ± SD: 29.0 ± 10.7). Twenty-five percent of the sample had CDI-II scores within the “high average” or “elevated” severity ranges, and 69% had scores in the highest severity “very elevated” range. All participants endorsed significant suicidal thoughts and behaviors on the Suicide Behaviors Questionnaire-Revised (SBQ-R total score $\geq$ 8). More specifically, SBQ-R total scores ranged from 10 to 18 (mean ± SD: 15.6 ± 2.1). One participant reported having thought about killing himself three or four times in the past year, and all others reported that they had these thoughts five or more times in the past year (ie highest frequency rating on the SBQ-R). Eighty-one percent reported that they were likely to attempt suicide someday.

3.2. Retrospective subjective sleep estimates in the suicidal group

Of the suicidal group, 6% (n = 1) reported having “good” sleep in the week preceding the study, 50% (n = 8) reported “fair” sleep and 44% (n = 7) reported “poor” sleep. Thirty-one percent of the sample (n = 5) reported a habitual bedtime between 1:00AM and 3:30AM.

Subjective estimates of habitual sleep onset latency ranged between 3 and 240 minutes (mean ± SD: 75.8 ± 74.7 minutes), and estimates of habitual sleep duration ranged between 2.5 and 10.5 hours (mean ± SD: 6.1 ± 2.2 hours). Overall, 75% (n = 12) participants reported subjective sleep difficulties as reflected by either a habitual sleep onset latency greater than 30 minutes and/or an atypically short (<6.5 hours) or long (>9.5 hours) sleep duration.

3.3. Objective sleep measures

Polysomnography sleep architecture variables for each group are reported in Table 2. Significant group differences were found for sleep onset latency (Z = −4.5, p < 0.001), REM latency (Z = −3.2, p = 0.001), REM density (Z = −2.8, p = 0.004), and the percentage of N1 sleep (t(33) = −2.6, p = 0.020). Specifically, compared to the control group, the suicidal group took 46 minutes longer to fall asleep and 70 minutes longer to reach REM sleep. Their REM density was nearly twice as high as that of the control group. Also, the percentage of N1 sleep was slightly, but significantly, higher in the suicidal group than in the control group. There was no other significant group difference in total sleep time, WASO, or all night N2, N3 and REM sleep.

Within the depression group, higher REM density correlated significantly with higher CDI-II scores (r = 0.55, p = 0.27; Fig. 1), but not with SBQ-R scores. No other polysomnography variable was significantly correlated with SBQ-R or CDI-II scores. Shorter subjective habitual sleep duration correlated significantly with higher percentage of N3 sleep in the first third of the sleep period (r = −0.53, p = 0.035; Fig. 2). No significant correlation was found.

3.4. SWS dynamics across the night

A significant group by time interaction was found for the percentage of N3 sleep (F(2,66) = 3.4, p = 0.041; Fig. 3). The two groups had similar N3 percentage in the first two thirds of the night (p ≥ 0.411), but the suicidal group had significantly lower N3 percentage than the control group in the last third of the night (p = 0.008).

4. Discussion

Based on objective polysomnography recorded directly inside a Canadian inpatient pedopsychiatric unit, the present findings provide a unique window on sleep and severe suicidality in hospitalized adolescents. To the best of our knowledge, this is the first

Table 2
Polysomnography variables in the control and suicidal group.

	Control	Suicidal
Sleep Architecture		
Sleep Latency (min) **	11.5 (7.1)	58.7 (46.0)
Total Sleep Time (min)	445.8 (30.9)	451.7 (37.2)
REM Latency (min) **	109.4 (32.1)	182.8 (65.1)
REM density (REM/minute)*	5.1 (4.2)	9.9 (5.5)
Wake After Sleep Onset	15.0 (10.0)	20.5 (22.2)
Absolute Sleep Stages Durations (min)		
N1	9.0 (3.1)	16.0 (10.5)
N2	220.3 (34.3)	234.4 (42.2)
N3	125.6 (24.0)	114.1 (47.8)
REM	90.8 (13.9)	85.6 (35.5)
Relative Sleep Stages Durations (%)		
N1*	2.0 (0.7)	3.5 (2.2)
N2	49.3 (6.3)	51.6 (9.4)
N3	28.0 (5.0)	25.7 (11.8)
REM	20.4 (3.3)	18.7 (7.2)

*p > 0.050, **p ≥ 0.001.

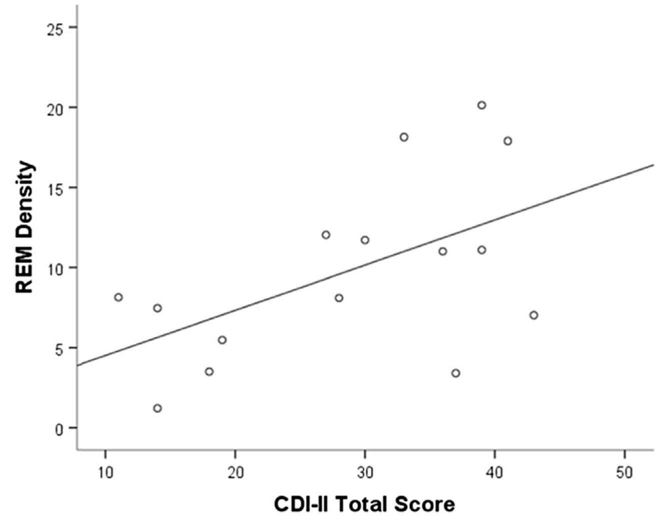

Fig. 1. Correlation between depression severity and REM Density in the suicidal group. REM Density: Average number of rapid eye movements per minute. CDI-II: Child Depression Inventory II.

naturalistic study evaluating the combined influence of major depression, suicidality, and acute medication changes in young people sleeping in a psychiatric ward.

Subjective sleep estimates in the week preceding the study (ie the days surrounding psychiatric admission) varied widely within this sample of suicidal adolescents. Over a quarter of this sample reported delayed sleep schedules and three quarters reported serious sleep initiation difficulties and/or abnormally short or long sleep duration (mostly short durations). These high rates of subjective sleep disturbances are consistent with previous findings in suicidal people (eg Refs. [27–29,38]).

In terms of objective sleep measures during hospitalization, our finding of long sleep onset latency and increased REM density is aligned with results from the previous polysomnography study in suicidal adolescents [46]. Conversely to some past reports in adults [44,45] and adolescents [46], we observed a longer REM latency. While this cannot be ascertained with the design of the present

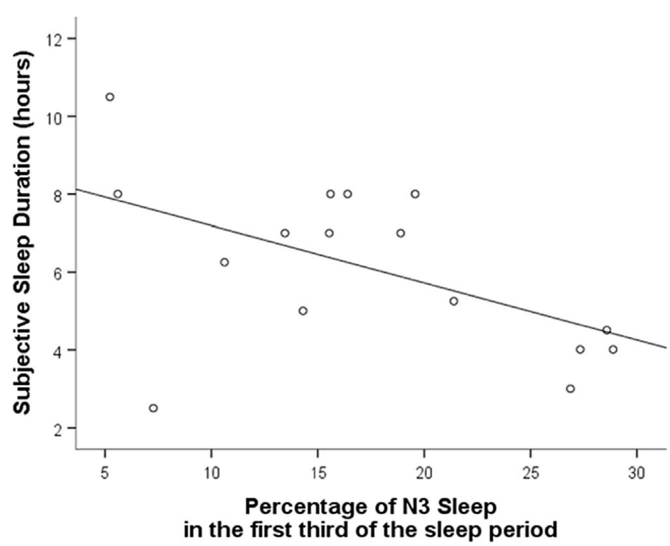

Fig. 2. Correlation between subjective sleep duration during the week preceding polysomnography and the proportion of Slow Wave Sleep in the Suicidal group. N3 Sleep: Non-REM 3 (Slow Wave) Sleep.

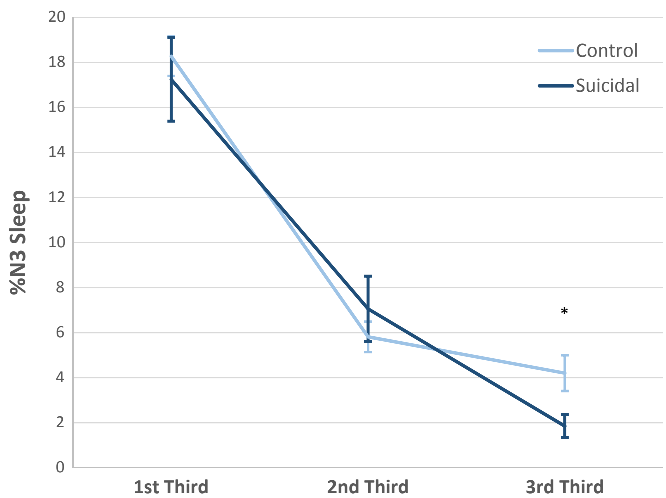

Fig. 3. Changes in slow wave sleep across the night.

study, this could potentially be linked to the REM suppressing effects of antidepressant medications and/or to the phase shifting effects of an imposed early sleep schedule in adolescents who may have been going to sleep later before being admitted to the inpatient unit. Our new observation of a slightly higher proportion of N1 sleep in the suicidal group is suggestive of shallower sleep. This interpretation may also be consistent with the reduction in deep SWS towards the end of the night, a finding similar to that reported in suicidal adults [44,45]. This is also reminiscent of previous reports that worse suicidality is associated with lower SWS [47]. In the present study, a large proportion of our sample reported short sleep durations in the week preceding the study. Shorter sleep duration before the study was associated with high levels of SWS in the first third of the night during hospitalization. While this remains to be further investigated, higher proportions of SWS early in the night during the recovery period after a suicidal crisis may be reflective of a homeostatic sleep response proportional to the degree of sleep loss occurring in the acute phase of a suicidal crisis. While more severe depressive symptoms were associated with higher REM density, the lack of significant correlation between the severity of suicidal symptoms and sleep variables could possibly result from a ceiling effect inherent to the nature of our clinical sample since all participants were highly suicidal.

The link between sleep disturbances and suicidality has previously been proposed to result from common pathophysiological mechanisms, like elevations in proinflammatory cytokines, alterations in the HPA axis and reductions in serotonergic tone [52–55]. Furthermore, the emotional (eg increased lability and negative ruminations), cognitive (eg impulsivity) and behavioral (eg disinhibition) effects of sleep loss are likely to worsen depressive states and restrict psychological coping resources [28]. Of note, abnormalities in factors known to influence SWS, SWA and homeostatic sleep regulation have been found in the frontal cortex of suicide victims: higher serotonin affinity and binding sites, higher corticotropin-releasing hormone levels and signs of $GABA_A$ dysregulation (eg Ref. [56]. Adolescence is a critical neurodevelopmental period for the maturation of the frontal cortex. This period also sees major changes in growth hormone synthesis (notably during sleep), a factor notably related to suicidality in adolescents with depression [57]. Further investigations of SWS dynamics across the night and their potential interaction with neuroendocrine, cognitive, and inflammatory markers may provide valuable information about some of the pathophysiological sleep mechanisms linked to suicidality during adolescence.

Several limitations need to be acknowledged. This study was based on a small sample size and both groups were recruited from different sites. Although polysomnography protocols were similar across both sites and all sleep data from both groups was scored by the same sleep technologist, the equipment used for data collection did differ between the suicidal and control group. The main analyses were based on a single night of polysomnography. Considering the high night-to-night variability commonly seen in youths with depression, and since sleep is likely to undergo progressive changes across the hospitalization and recovery periods, it may be useful to monitor sleep over multiple nights. Subjective sleep data collected retrospectively could have been altered by memory bias. While this study specifically aimed to investigate the combination of suicidality, depression, pharmacotherapy and psychiatric hospitalisation (ie a typical combination in most instances of severe suicidal crises), further work is required to decipher the relative influence of these different factors. Some factors may have additive and/or interactive influence with each other. Notably, some attention should be given to the potential interactions between prior medication history and acute medication changes during hospitalization.

In brief, the present findings provide an ecological estimate of how suicidal youths, most of whom also have depression and are taking psychotropic medications, sleep when hospitalised in a typical Canadian psychiatric ward. This highlights the importance of taking active measures to protect the sleep of inpatients, who, in addition to sleeping in a foreign environment away from their family during a period of crisis, are also often exposed to increased nocturnal noises and lights. Considering the strong evidence supporting the predictive value of sleep disturbances for subsequent mental health and suicidality, there is a strong need for a better understanding of the relative impact of the different factors which can worsen sleep and for developing effective sleep interventions in youths with high suicidal risk. Beyond pharmacotherapy, known for its limited long-term efficacy, several sleep-promoting strategies could be implemented during hospitalization.

Funding

Part of this work was supported by a postdoctoral fellowship from the Fonds de la recherche en santé du Québec (FRQS) allocated to RR.

Acknowledgements

The authors wish to thank the participants and their families who generously gave their time and efforts during a difficult period, as well as Professors Roger Godbout (Centre de recherche de l'Institut universitaire en santé mentale de Montréal) and Kenneth Campbell (School of Psychology, Univeristy of Ottawa) for their assistance with study development.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2018.12.018.

References

[1] Public healthy agency of Canada analysis of statistics Canada mortality data. 2012 [Ottawa].

[2] Skinner R, McFaull S. Suicide among children and adolescents in Canada: Trends and sex differences, 1980-2008. CMAJ 2012;184(9):1029–34. https://doi.org/10.1503/cmaj.111867.
[3] Greenham SL, Bisnaire L. An outcome evaluation of an inpatient crisis stabilization and assessment program for youth. Resid Treat Child Youth 2008;25(2):123–43.
[4] Lee CY, Low LP, Twinn S. Older men's experiences of sleep in the hospital. J Clin Nurs 2007;16(2):336–43.
[5] Lei Z, Qiongjing Y, Qiuli W, et al. Sleep quality and sleep disturbing factors of inpatients in a Chinese general hospital. J Clin Nurs 2009;18(17):2521–9.
[6] Sadeh A, McGuire JP, Sachs H, et al. Sleep and psychological characteristics of children on a psychiatric inpatient unit. J Am Acad Child Adolesc Psychiatr 1995;34(6):813–9.
[7] Gradisar M, Gardner G, Dohnt H. Recent worldwide sleep patterns and problems during adolescence: A review and meta-analysis of age, region, and sleep. Sleep Med 2011;12(2):110–8.
[8] Iglowstein I, Jenni OG, Molinari L, et al. Sleep duration from infancy to adolescence: reference values and generational trends. Pediatrics 2003;111(2):302–7. https://doi.org/10.1542/peds.111.2.302.
[9] Gaudreau H, Carrier J, Montplaisir J. Age-related modifications of NREM sleep EEG: From childhood to middle age. J Sleep Res 2001;10(3):165–72. https://doi.org/10.1046/j.1365-2869.2001.00252.x.
[10] Jenni OG, Carskadon MA. Spectral analysis of the sleep electroencephalogram during adolescence. Sleep 2004;27(4):774–83. https://doi.org/10.1093/sleep/27.4.774.
[11] Ohayon MM, Carskadon MA, Guilleminault C, et al. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. Sleep 2004;27(7):1255–73.
[12] Jenni OG, Achermann P, Carskadon MA. Homeostatic sleep regulation in adolescents. Sleep 2005a;28(11):1446–54. https://doi.org/10.1093/sleep/28.11.1446.
[13] Taylor DJ, Jenni OG, Acebo C, et al. Sleep tendency during extended wakefulness: Insights into adolescent sleep regulation and behavior. J Sleep Res 2005;14(3):239–44. https://doi.org/10.1111/j.1365-2869.2005.00467.x.
[14] Armitage R, Hoffmann R, Trivedi M, et al. Slow-wave activity in NREM sleep: Sex and age effects in depressed outpatients and healthy controls. Psychiatr Res 2000;95(3):201–13. https://doi.org/10.1016/S0165-1781(00)00178-5.
[15] Goetz RR, Puig-Antich J, Ryan N, et al. Electroencephalographic sleep of adolescents with major depression and normal controls. Arch Gen Psychiatr 1987;44(1):61–8. https://doi.org/10.1001/archpsyc.1987.01800130069009.
[16] Lauer CJ, Riemann D, Wiegand M, et al. From early to late adulthood changes in EEG sleep of depressed patients and healthy volunteers. Biol Psychiatr 1991;29(10):979–93. https://doi.org/10.1016/0006-3223(91)90355-P.
[17] Robillard R, Naismith SL, Smith KL, et al. Sleep-wake cycle in young and older persons with a lifetime history of mood disorders. PLoS One 2014;9(2):399–402. https://doi.org/10.1371/journal.pone.0087763.
[18] Surridge-David M, Maclean A, Coulter ME, et al. Mood change following an acute delay of sleep. Psychiatr Res 1987;22(2):149–58. Retrieved from, https://journals-scholarsportal-info.proxy.bib.uottawa.ca/pdf/01651781/v22i0002/149_mcfaados.xml.
[19] Wendt A. Social theory of international politics. Camb Stud Int Relat 1999;67(4):429. 67, https://doi.org/10.1017/CBO9780511612183.
[20] Appelboom-Fondu J, Kerkhofs M, Mendlewicz J. Depression in adolescents and young adults – polysomnographic and neuroendocrine aspects. J Affect Disord 1988;14(1):35–40. https://doi.org/10.1016/0165-0327(88)90069-9.
[21] Robillard R, Hermens DF, Naismith SL, et al. Ambulatory sleep-wake patterns and variability in young people with emerging mental disorders. J Psychiatr Neurosci 2015;40(1):28–37. https://doi.org/10.1503/jpn.130247.
[22] Robillard R, Naismith SL, Rogers NL, et al. Delayed sleep phase in young people with unipolar or bipolar affective disorders. J Affect Disord 2013;145(2):260–3. https://doi.org/10.1016/j.jad.2012.06.006.
[23] Baglioni C, Nanovska S, Regen W, et al. Sleep and mental disorders: A meta-analysis of polysomnographic research. Psychol Bull 2016;142(9):969–90.
[24] Emslie GJ, Rush AJ, Weinberg WA, et al. Sleep EEG features of adolescents with major depression. Biol Psychiatr 1994;36(9):573–81. https://doi.org/10.1016/0006-3223(94)90067-1.
[25] Tesler N, Gerstenberg M, Franscini M, et al. Increased frontal sleep slow wave activity in adolescents with major depression. Neuroimage: Clin 2016;10:250–6.
[26] Lopez J, Hoffmann R, Emslie G, et al. Sex differences in slow-wave electroencephalographic activity (SWA) in adolescent depression. Ment Illness 2012;4(1):4. https://doi.org/10.4081/mi.2012.e4.
[27] Goodwin RD, Marusic A. Association between short sleep and suicidal ideation and suicide attempt among adults in the general population. Sleep 2008;31(8):1097–101. https://doi.org/10.1027/0227-5910/a000104.
[28] Pigeon WR, Pinquart M, Conner K. Meta-analysis of sleep disturbance and suicidal thoughts and behaviors. J Clin Psychiatr 2012;73(9). https://doi.org/10.4088/JCP.11r07586.
[29] Wojnar M, Ilgen MA, Wojnar J, et al. Sleep problems and suicidality in the National comorbidity survey replication. J Psychiatr Res 2009;43(5):526–31. https://doi.org/10.1016/j.jpsychires.2008.07.006.
[30] Bailly D, Bailly-Lambin I, Querleu D, et al. Le sommeil des adolescents et ses troubles une enquête en milieu scolaire. L'Encéphale 2004;30(4):352–9. https://doi.org/10.1016/S0013-7006(04)95447-4.
[31] Barbe RP, Williamson DE, Bridge JA, et al. Clinical differences between suicidal and nonsuicidal depressed children and adolescents. J Clin Psychiatr 2005;66(4):492–8. https://doi.org/10.4088/JCP.v66n0412.
[32] Becker SP, Dvorsky MR, Holdaway AS, et al. Sleep problems and suicidal behaviors in college students. J Psychiatr Res 2018;99:122–8.
[33] Choquet M, Menke H. Suicidal thoughts during early adolescence: prevalence, associated troubles and help-seeking behavior. Acta Psychiatr Scand 1990;81(2):170–7. https://doi.org/10.1111/j.1600-0447.1990.tb06474.x.
[34] Liu X. Sleep and adolescent suicidal behavior. Sleep 2004;27(7):1351–8. https://doi.org/10.1093/sleep/27.7.1351.
[35] McKnight-Eily LR, Eaton DK, Lowry R, et al. Relationships between hours of sleep and health-risk behaviors in US adolescent students. Prev Med 2011;53(4–5):271–3. https://doi.org/10.1016/j.ypmed.2011.06.020.
[36] Nrugham L, Larsson B, Sund AM. Specific depressive symptoms and disorders as associates and predictors of suicidal acts across adolescence. J Affect Disord 2008;111(1):83–93. https://doi.org/10.1016/j.jad.2008.02.010.
[37] Sarchiapone M, Mandelli L, Carli V, et al. Hours of sleep in adolescents and its association with anxiety, emotional concerns, and suicidal ideation. Sleep Med 2014;15(2):248–54. https://doi.org/10.1016/j.sleep.2013.11.780.
[38] Chiu HY, Lee HC, Chen PY, et al. Associations between sleep duration and suicidality in adolescents: A systematic review and dose–response meta-analysis. Sleep Med Rev 2018 Dec;42:119–26.
[39] Fitzgerald CT, Messias E, Buysse DJ. Teen sleep and suicidality: Results from the youth risk behavior surveys of 2007 and 2009. J Clin Sleep Med 2011;7(4):351–6. https://doi.org/10.5664/JCSM.1188.
[40] Goldstein TR, Bridge JA, Brent DA. Sleep disturbance preceding completed suicide in adolescents. J Consult Clin Psychol 2008;76(1):84–91. https://doi.org/10.1037/0022-006X.76.1.84.
[41] Lee YJ, Cho S-J, Cho IH, et al. Insufficient Sleep and Suicidality in Adolescents. Sleep 2012;35(4):455–60. https://doi.org/10.5665/sleep.1722.
[42] Roane BM, Taylor DJ. Adolescent insomnia as a risk factor for early adult depression and substance abuse. Sleep 2008;31(10):1351–6. https://doi.org/10.5665/sleep/31.10.1351.
[43] Wong MM, Brower KJ. The prospective relationship between sleep problems and suicidal behavior in the National longitudinal study of adolescent health. J Psychiatr Res 2012;46(7):953–9. https://doi.org/10.1016/j.jpsychires.2012.04.008.
[44] Agargun MY, Cartwright R. REM sleep, dream variables and suicidality in depressed patients. Psychiatr Res 2003;119(1–2):33–9. https://doi.org/10.1016/S0165-1781(03)00111-2.
[45] Sabo E, Reynolds CF, Kupfer DJ, et al. Sleep, depression, and suicide. Psychiatr Res 1991;36(3):265–77. https://doi.org/10.1016/0165-1781(91)90025-K.
[46] Dahl RE, Puig-Antich J, Ryan ND, et al. EEG sleep in adolescents with major depression: the role of suicidality and inpatient status. J Affect Disord 1990;19(1):63–75. https://doi.org/10.1016/0165-0327(90)90010-6.
[47] McCracken JT, Poland RE, Lutchmansingh P, et al. Sleep electroencephalographic abnormalities in adolescent depressives: Effects of scopolamine. Biol Psychiatr 1997;42(7):577–84. https://doi.org/10.1016/S0006-3223(96)00431-3.
[48] Poznanski E, Freeman L, Mokros H. Children's depression rating scale-revised (September 1984). Psychopharmacol Bull 1985;21:979–89. Retrieved from, https://ci.nii.ac.jp/naid/10012180635/.
[49] Kovacs M. Children's depression inventory (CDI2): technical manual. North Tonawanda, NY: Multi-Health Systems, Inc; 2011.
[50] Osman A, Bagge CL, Gutierrez PM, et al. The Suicidal Behaviors Questionnaire-Revised (SBQ-R): validation with clinical and nonclinical samples. Assessment 2001;8(4):443–54.
[51] Silber MH, Ancoli-Israel S, Bonnet MH, et al. The visual scoring of sleep in adults. J Clin Sleep Med 2007;3(2):22.
[52] Dahl RE, Ryan ND, Puig-Antich J, et al. 24-Hour cortisol measures in adolescents with major depression: A controlled study. Biol Psychiatr 1991;30(1):25–36. https://doi.org/10.1016/0006-3223(91)90067-V.
[53] Janelidze S, Mattei D, Westrin Å, et al. Cytokine levels in the blood may distinguish suicide attempters from depressed patients. Brain Behav Immun 2011;25(2):335–9. https://doi.org/10.1016/j.bbi.2010.10.010.
[54] Mathew SJ, Coplan JD, Goetz RR, et al. Differentiating depressed adolescent 24 h cortisol secretion in light of their adult clinical outcome. Neuropsychopharmacology 2003;28(7):1336–43. https://doi.org/10.1038/sj.npp.1300184.
[55] Perlis M, Gehrman P, Pigeon WR, et al. Neurobiologic mechanisms in chronic insomnia. Sleep Med Clin 2009 Dec;4(4):549–58.
[56] Merali Z, Du L, Hrdina P, et al. Dysregulation in the suicide brain: mRNA expression of corticotropin-releasing hormone receptors and GABA(A) receptor subunits in frontal cortical brain region. J Neurosci: Off J Soc Neurosci 2004;24(6):1478–85. https://doi.org/10.1523/JNEUROSCI.4734-03.2004.
[57] Ryan ND, Puig-Antich J, Rabinovich H, et al. Growth hormone response to desmethylimipramine in depressed and suicidal adolescents. J Affect Disord 1988;15(3):323–37. https://doi.org/10.1016/0165-0327(88)90029-8.