Published in final edited form as:
Sleep Med. 2010 August ; 11(7): 622–627. doi:10.1016/j.sleep.2009.11.018.

APPROACHES TO THE ASSESSMENT OF AROUSALS AND SLEEP DISTURBANCE IN CHILDREN

Shalini Paruthi, MD[1] and **Ronald D. Chervin, MD, MS**[2]

[1]Pediatric Sleep and Research Center and Department of Pediatrics, Saint Louis University, St. Louis, Missouri

[2]Sleep Disorders Center and Department of Neurology, University of Michigan, Ann Arbor, Michigan

Abstract

Childhood arousals, awakenings, and sleep disturbances during the night are common problems for both patients and their families. Additionally, inadequate sleep may contribute to daytime sleepiness, behavioral problems, and other important consequences of pediatric sleep disorders. Arousals, awakenings, and sleep disturbances can be quantified by routine polysomnography, and arousal scoring is generally performed as part of the standard polysomnogram. Here we review current approaches to quantification of arousals and sleep disturbances and examine outcomes that have been associated with these measures. Initial data suggest that computer-assisted identification of nonvisible arousals, cyclic alternating patterns, or respiratory cycle-related EEG changes may complement what can be accomplished by human scorers. Focus on contiguous bouts of sleep or specific sleep stages may prove similarly useful. Incorporation of autonomic arousal measures—such as heart rate variability, pulse transit time, or peripheral arterial tone—into standard reports may additionally capture subtle sleep fragmentation.

Keywords

child; pediatric; sleep; arousals; sleep disturbance; spectral analysis; respiratory cycle related EEG changes; RCREC; heart rate variability; pulse transit time; peripheral arterial tonometry; movements

Introduction

Childhood arousals, awakenings, and sleep disturbances during the night are common problems for both, patients and their families. Additionally, inadequate sleep may contribute to daytime sleepiness, behavioral problems, and other important consequences of pediatric sleep disorders. Arousals, awakenings, and sleep disturbances can be quantified by routine polysomnography, and arousal scoring is generally performed as part of the standard polysomnogram. The concepts of sleep consolidation versus fragmentation are intrinsic to most clinicians' views of normal versus disordered sleep; yet data are sparse on the clinical meaning of arousals and other forms of disruption in normal sleep cycles. This manuscript will review current

Address for Correspondence: Shalini Paruthi, MD Saint Louis University 1465 S. Grand Blvd. Glennon Hall 2715 St. Louis, MO 63104 Phone: 314-268-6439 Fax: 314-268-2798 sparuthi@slu.edu.

approaches to quantification of arousals and sleep disturbances and examine outcomes that have been associated with these measures.

Assessment of arousals and sleep disturbances in standard polysomnograms

History

The American Sleep Disorders Association's (ASDA) precursor to the American Academy of Sleep Medicine (AASM) provided consensus criteria in 1992 for the scoring of arousals in adults. An arousal required an increase in electroencephalogram (EEG) frequency lasting at least three seconds in duration.[1] If the shift occurred during rapid eye movement (REM) sleep, increased submental muscle tone was also required.[1] This definition was replaced in 2007 by recommendations for scoring arousals in The AASM Manual for the Scoring of Sleep and Associated Events (AASM Scoring Manual).[2] Briefly, the AASM Scoring Manual endorses scoring of arousals during all stages of sleep if an abrupt shift of EEG frequency occurs, including alpha, theta or frequencies greater than 16 Hertz (but not spindles). The disruption should last at least three seconds, with at least 10 seconds of preceding stable sleep. Scoring arousals during REM sleep requires additional increases in submental electromyogram (EMG) lasting at least one second. Further guidance is provided to use information from the occipital and central EEG leads. Use of additional recorded information, including respiratory events and other EEG channels, can improve arousal scoring. These scoring rules apply to adults and children, between which no distinction is made. The AASM Scoring Manual arousal recommendation builds on the foundation of the ASDA arousal definition, adds guidelines to score from frontal leads, and further defines the length of required EMG increases during REM sleep.[2]

Sleep disturbance is discussed under the AASM Scoring Manual section on major body movements. Movement and muscle artifact can obscure the EEG for more than half an epoch, making it difficult to determine the sleep stage. The epoch should be scored as wake if alpha rhythm is present for any part of the epoch (even <15 seconds), or if the preceding or following epoch is scoreable as wake. Otherwise, the epoch is scored as the same stage as the following epoch. [2]

Furthermore, the AASM Pediatric Task Force published a separate review paper covering scoring of sleep and arousals in infants and children in 2007, summarizing evidence used to support the terminology and rules in the AASM Scoring Manual.[3] Their considerations and conclusions, which complement the AASM Scoring Manual, will also be reviewed briefly.

Length of Arousal

Debate exists about the minimum length of arousals (1-, 2-, or 3-seconds) scored in pediatric polysomnography. Some investigators have attempted to use shorter arousal duration criteria in an attempt to increase sensitivity.[1] In 1992, the ASDA guidelines[4] stated that a 3-second duration criterion was chosen for methodological rather than physiological reason to improve interscorer identification and agreement on cortical arousals. The 2007 AASM Scoring Manual also recommends using a duration of 3 seconds. Multiple duration criteria were tested by two experienced sleep practitioners who reviewed 36 pediatric polysomnograms (from 20 children with sleep disordered breathing and 16 normal children) using the ASDA 3-second criteria and modified duration criteria of 1 and 2 seconds.[5] Excellent interscorer agreement was found using the 3-second duration criteria (intraclass correlation coefficient [ICC] 0.90). Only poor to fair interscorer agreement was noted with the modified 1-second criteria (ICC 0.35) and modified 2-second criteria (ICC 0.42). Furthermore, short arousals accounted for only 15% of all arousals

in children with obstructive sleep apnea and 8.5% of all arousals in normal children, suggesting that the scoring of shorter arousals may have limited clinical value.[5]

Clinical utility of arousal scoring and sleep staging in children

In order to quantify sleep fragmentation or sleep disruption, clinicians often turn to the arousal index routinely scored and calculated on polysomnography reports. However, further differentiation of arousals as spontaneous, respiratory-related, periodic limb movement-related, or technician-induced is typically not performed. Part of the challenge is the absence of accepted criteria to define the required proximity of an arousal to the termination of an associated apneic event or to a technician disturbance. The 2007 AASM Scoring Manual now requires that < 0.5 seconds separate a periodic leg movement from an arousal considered to be associated with the movement, regardless of which comes first.[2]

Pediatric polysomnograms from children with and without obstructive sleep apnea showed the former to have significantly more total arousals from sleep.[6] Yet, the arousal index was considerably lower than the apnea index in the children with severe OSA. As the OSA worsened through the night, particularly during the latter third of the night due to the increase in rapid eye movement (REM) sleep, the respiratory arousal index also increased. In contrast, the spontaneous arousal index (total, Non-REM [NREM} or REM sleep) did not differ significantly as the night progressed.[6] Increased arousals with sleep apnea were also found in a larger study of 559 children (345 children with obstructive sleep apnea or mild sleep disordered breathing and 214 control children) aged 2-15 years.[7] These results are not unexpected, as arousals are used in part to define an element of the apnea / hypopnea index (AHI), specifically the hypopneas.

Clinical utility of the arousal index is somewhat limited, however, given the large variability in pediatric normal values reported by many investigators. Normal reported values range from 5±2 to 10.8±4.2 for children,[3] and the AASM Pediatric Task force has recommended an arousal index of < 14 per hour of sleep as normal for a prepubertal child in a sleep laboratory, taking first-night effect into account.[3] Used without other polysomnographic variables, the arousal index alone does not provide as much clinical utility as many clinicians would hope. Clinicians can often provide patients with an estimate of sleep fragmentation in more understandable terms. After interventions to treat sleep disordered breathing (SDB), clinicians may be able to use other polysomnographic variables in combination with EEG arousals to estimate treatment effect.[8]

However, investigators also have raised questions about the direct clinical utility of conventional sleep staging in the assessment of childhood sleep disorders. For example, sleep staging in children with and without obstructive sleep apnea may not show significant differences. [6,9]

Placement of electrodes

The AASM Scoring Manual recommends placement of EEG leads over the frontal, central, and occipital regions to optimize recordings in infants, children, and adults.[2] Central electrodes referenced to the contralateral ear detected 96% of arousals in pediatric polysomnograms, with only 88% of the same arousals concurrently identified over the frontal electrodes.[10] Only 2% of arousals appeared solely on the frontal leads.[11] In contrast, K complexes that may often represent internally or externally induced brain activations (if not accompanied by faster frequency that can be recognized as arousals[11,12]) are maximal over the pre-frontal and frontal regions.[2] The addition of the frontal leads to conventional polysomnography also allows scorers to distinguish more reliably between spindles and cortical arousals. Frontal sleep spindles are more prominent than centroparietal spindles in young children, though typically

frontal spindles abruptly decrease in EEG power and occurrence beginning at age 13 years, whereas centroparietal spindles persist unchanged.[2]

Once the recording has been obtained, multiple options exist for scoring of sleep stages and arousals. Digital acquisitions of signals followed by human scoring has become standard practice. In an effort to save time and costs, computerized scoring systems have been developed, based mainly on the Rechtschaffen and Kales sleep staging manual.[13] In 2007, the AASM Digital Task Force published a review of computerized scoring and assessed issues such as computer-to-human staging concordance.[14] Certain waveforms carry higher reliability for detection, including spindles and alpha waves, by computerized scoring systems. The task force members noted artifacts had yet to be adequately addressed; this has clinical implications as many large movements trigger arousals or changes in sleep stages.[15] Though progress has been made on the design of computerized scoring systems, they have generally been based on the previous staging manual, and may need to be updated and validated against the new AASM Scoring Manual rules for sleep stages and arousals. At present, visual scoring remains the standard practice.

To improve the ability to score arousals, the AASM Arousal Task Force stresses the importance of scorer training and experience.[15] Periodic directed training, utilizing gold-standard sleep recordings is recommended.

Alternative Approaches to the assessment of arousals and sleep disruption

Given that standard arousal and sleep stage analyses are not always as clinically informative as might be hoped, several alternative approaches have been developed to characterize or classify sleep and its disruption. Some of these approaches are outlined below, though at this time, none have yet become standard of practice in most sleep laboratories.

Sleep Pressure Score

As mentioned earlier, arousals can be subdivided into the following: spontaneous, respiratory-related, periodic limb movement-related, or technician-induced. An increased total arousal index and increased respiratory arousal index, but decreased spontaneous arousal index are found in children with sleep disordered breathing.[7] If compensatory mechanisms aim to maintain sleep homeostasis, the spontaneous arousal index should decrease in response to an increase in respiratory related arousals. The reciprocal relationships of the apnea/hypopnea index, respiratory arousal, spontaneous arousal and total arousal are described by the sleep pressure score (SPS) formula:

$$\text{sleep pressure score} = \frac{\text{respiratory arousal index}}{\text{total arousal index}} \star \frac{1 - \text{spontaneous arousal index}}{\text{total arousal index}}$$

The resulting value may reflect increases in sleep pressure (sleepiness) that occur in the context of chronic sleep disruption by SDB and may provide a sensitive and independent correlate of cognitive and behavioral morbidity in children who snore. [7]

Investigators tested this hypothesis on 199 five- to seven-year-old children (49 with high SPS and 150 with low SPS).[7] The cutoff for the SPS correlated to an AHI of approximately 7 and marked the point of increased sleep pressure. Children with higher SPS had relative neurocognitive deficits, particularly in areas of memory, language and visuospatial subtests. A trend was noted toward more hyperactive behavior in children with higher sleep pressure scores.[16]

Cyclic alternating pattern

Cyclic alternating pattern (CAP) is a spontaneous pattern during NREM sleep in which transient EEG patterns believed to represent brain arousal or activation are separated by generally somewhat longer intervals of background EEG activity. Cyclic alternating pattern can now be used as a systematized method to quantify and classify arousals and NREM sleep instability. The large majority (87%) of AASM-defined arousals are captured within CAP sequences by the A2 and A3 subtypes of cortical activations.[17] The CAP scoring also identifies A1 activations that are comprised solely of slow wave EEG activity rather than faster frequencies that form all or part of the A3 and A2 forms, respectively.[18] Therefore, CAP in comparison to standard arousal scoring captures additional elements of sleep stage instability, for example, during progression to deeper sleep stages or unsuccessful threats to sleep continuity. Please see an accompanying article on CAP in this issue for further discussion.

Non-visible cortical arousals detected by computer analysis

Spectral Analysis—As children, in comparison to adults, are less likely to exhibit visible EEG cortical arousal during or following apneic respiratory events,[19] many investigators have searched for less obvious electronic signs of sleep disruption in association with respiratory disturbances. Dynamic changes in EEG spectra were demonstrated during obstructive sleep apnea among 8 children aged 2-8 years.[20] The investigators found 28% of isolated obstructive sleep apneas occurred without arousal or artifact. Spectral power analysis showed that the mean EEG delta power decreased during the respiratory event and then increased immediately following the respiratory event to exceed baseline levels. Conversely, theta power increased during the respiratory events and decreased following the events. No significant changes were noted in the alpha, sigma and beta frequencies. The delta frequency power changes that did occur were not correlated with severity of hypoxia. Although these findings did not persist when 4 or more obstructive events occurred in clusters, results were suggestive overall that delta and theta frequency power changes could represent subtle arousal. [21]

Respiratory Cycle Related EEG Changes (RCREC)—In assessing children for SDB, sleep fragmentation has almost always been analyzed—by human scoring or rarely computer analysis—strictly in association with apneic events. However, SDB patients who undergo esophageal pressure monitoring often demonstrate increased work of breathing during the many breaths that occur between discrete, scoreable apneic events. Considerable literature now shows that standard polysomnographic measures, including arousals, often fail to predict neurobehavioral morbidity which is considered one of the main outcomes of childhood SDB. In an effort to assess possible contributions from continuously labored breathing, during the majority of a typical night not occupied by apneas or hypopneas, a computer algorithm was developed to detect any EEG changes that might occur on a breath-to-breath basis during non-apneic sleep. The algorithm was first tested in a child who had SDB before adenotonsillectomy and was cured by the procedure.[21] Highly statistically significant changes in EEG power could be documented, in concert with the average respiratory cycle, in this child. A subsequent analysis of data from this patient, as well as 9 other children with a wide range of SDB severity or no SDB showed that children with SDB tended to have more prominent respiratory cycle-related EEG changes (RCREC) than children without SDB.[22] Furthermore, after adenotonsillectomy, the RCREC in children who had SDB tended to improve. Subsequent studies in adults studied for possible SDB suggested that RCREC, particularly in the sigma frequency range, predicted sleepiness as measured by Multiple Sleep Latency Tests even after the apnea/hypopnea index was taken into account.[23] A study of more than 100 children, scheduled for adenotonsillectomy or else surgical care unrelated to the upper airway, showed that parent-rated, subjective sleepiness was predicted by sigma RCREC, again even after taking the apnea/hypopnea index into account.[24] An additional study in adults showed that sigma RCREC, in particular, tracks to some extent within individual patients the changes that occur

over the night in esophageal pressures.[25] Taken together, existing data suggest that RCREC may represent inspiratory microarousals that are subtle but numerous and consequential during sleep.

Sleep Dynamics—Beyond standard arousal and sleep staging, the length of particular sleep stage bouts may also be informative.[9] After adenotonsillectomy in 36 children with obstructive sleep apnea syndrome, no significant change was noted in the cumulative or median consecutive sleep stage duration, but the median uninterrupted slow wave sleep period showed statistically significant improvement.[9] Another study examined arousals and sleep dynamics —distributions of contiguous sleep and sleep stage durations—in 68 children (48 with sleep disordered breathing and 20 control subjects) aged 5-12 years. Several variables distinguished the sleep disordered breathing and control groups at baseline.[26] In contrast to the prior study, the arousal index was 14.6 ± 7.3 for children with SDB versus 8.7±2.5 for control children ($p = <0.0001$). Children with SDB also had more sleep stage changes, higher percentage of stage 1 sleep, higher percentage stage 2 sleep, shorter contiguous stage 2 durations, and shorter contiguous REM sleep durations. However, multiple regression models showed that only stage 2 bout durations distinguished children with SDB from controls after accounting for the other variables. One year later, children treated with adenotonsillectomy now experienced longer stage 2 sleep durations, and sleep stage durations could no longer differentiate original SDB patients from controls. The changes in stage 2 durations, rather than changes in sleep stage proportions, significantly distinguished SDB subjects from controls. Sleep dynamic analyses, and especially attention to mean durations of individual sleep stages, may offer useful clinical information in the evaluation of sleep fragmentation caused by childhood sleep disorders. [26]

Autonomic Measures

Considerable evidence now suggests that computer analysis of autonomic measures may identify arousals that are not reflected in visually scored EEG. Earlier explanations postulated that arousal-stimulating sensory information ascends through the central nervous system, does not always reach the cerebral cortex, and could still have impact on sleep and subsequent sleepiness. However, virtually all comparisons of autonomic measures to EEG have used computer algorithms to analyze the former and the human eye to score the latter. More recent data, as reviewed above, show that subtle, computer-detected changes in cortical EEG, with relevance to sleep fragmentation and daytime sleepiness, can be detected with appropriate algorithms. Therefore, the most cogent conclusion until proven otherwise is that autonomic measures of several types may have practical utility in assessment of clinically relevant arousals, but that concomitant cortical arousals too subtle to detect by eye may still explain consequent daytime sleepiness.

Heart rate variability

Several measures of autonomic function have garnered interest as markers for arousals that may be more sensitive than EEG changes that are visible to the human scorer. The heart rate, and its variability from beat to beat, is believed to provide an eloquent window into sympathetic and parasympathetic balance. One study found that overall heart rate variability changes, analyzed by the time domain method, were only modest in children with obstructive sleep apnea.[27] However, another study that evaluated the beat to beat variability of R-R intervals, also by time domain method, did find significant differences between children with and without SDB.[28] The seven children with OSAS had reduced R-R intervals (faster heart rate) beginning a few seconds after initiation of obstruction, followed by slower heart rate, and then variable heart rates when breathing resumed. For each recording, dispersion of next-intervals was determined for short, long, and intermediate R-R intervals and plotted onto scattergrams. These Poincare plots showed much greater next-interval dispersion following slower heart rates in

OSAS children than in control children. These results suggest that calculation of the ratio of next-interval dispersion following long versus intermediate R-R intervals may reliably separate the two groups of children. Specifically, the largest ratio in the normal children group was 0.9 and ratios in the children with SDB ranged from 1.6 to 2.4.[28] However, this study again found no difference in heart rate or heart rate variability between the two groups of children. [28]

Heart rate variability has also been investigated in other sleep disorders. For example, in 11 sleep walking adults, the total energy in the spectral analysis of heart rate variability, analyzed by the frequency domain method, increased in stage 4 sleep during the 5 minutes immediately prior to the start of pathological arousal, suggesting autonomic activation preceding cortical arousal.[29]

Another study evaluated the changes in pulse rate (by time domain frequency method) and peripheral vascular resistance, markers of autonomic function, in 25 children aged 1-18 years with moderate to severe obstructive sleep apnea.[30] Following adenotonsillectomy, the mean pulse rate decreased in 21/25 children from 99.7±11.2 to 90.1 ±10.7 beats per minute. Furthermore, pulse rate variability decreased in 23/25 children, with a decreased frequency of pulse rate rises greater than 6, 7, or 8 beats per minute.[30]

Pulse transit time

Pulse transit time (PTT) is measured as the time delay between the EKG R wave and the arrival of the pulse wave at the finger, detected through the transmittance signal of the oximeter probe. [31] Pulse transit time is non-invasive, varies inversely with blood pressure, and may also reflect stiffness of arterial walls at rest, as modulated by the autonomic nervous system. The use of PTT improved detection of respiratory events and microarousals especially during REM sleep and slow wave sleep.[32] In children undergoing esophageal pressure monitoring, more apneas, hypopneas, and respiratory effort related arousals terminated in PTT-defined arousals than in visible EEG arousals.[33]

Peripheral arterial tonometry

Peripheral Arterial Tonometry (PAT) allows noninvasive beat-to-beat assessment of sympathetic tone by using finger plethysmography to detect changes in peripheral vascular cutaneous perfusion. In 40 children with SDB, 35% of respiratory events were associated with visible EEG arousals, whereas 92% were associated with PAT-attenuation.[34] When PAT artifacts, such those caused by movement, were taken into account, sensitivity increased to 95% and specificity was 35%. Of note, the significant correlations between PAT and arousals were found only for spontaneous arousals, not respiratory arousals. This somewhat unexpected finding may be attributed to the fact that the majority of respiratory events do not elicit EEG arousals in children. Another possibility is that the study misclassified RERAs as spontaneous arousals as the children wore thermocouplers instead of nasal pressure cannulas.[34]

Further evidence for the potential clinical usefulness of PAT and PTT was provided by a study in which 10 healthy children underwent overnight polysomnography with PAT and PTT.[35] The sensitivity and specificity of PTT for recognizing arousals were 0.74 and 0.25, respectively, whereas the sensitivity and specificity of PAT for recognizing arousals were 0.57 and 0.13, respectively. When movement artifact events were included to aid in analysis, sensitivity of PTT for recognizing arousals rose to 0.96, sensitivity of PAT rose to 0.92, specificity of PTT rose to 0.30, and specificity of PAT rose to 0.19.[35]

Pulse Wave Amplitude

A new approach of using pulse wave amplitude, ie the difference between the peak and nadir values of corresponding photoplethysmogram pulse waveform for each cardiac cycle, may be

relevant in SDB. A criterion ≥ 30% drop in the waveform improved detection of hypopneas and cortical arousals. Concordance between 2 trained scorers in identification of respiratory events was also achieved. This technique has yet to be applied to children.[36]

Movements

Movements are frequently associated with arousal. Arousal are often accompanied by movements, however, not all movements should be considered arousals. The AASM Scoring Manual provides guidance to the scoring of movements and when to score the corresponding epoch as wake. [2]

Infants

Infants three months of age or younger move more during sleep than older infants, yet these movements often cause no arousal. In infants and children up to 18 months of age, all types of body movements decrease with age, until a basal level 9 to 13 months post-term.[37] The AASM Pediatric Task Force cautions that use of movement or position change (particularly in infants < 3 months of age) as a proxy for cortical arousal will lead to overscoring of arousals.[3]

Given available data on infant movements and the observation that infants have relatively few EEG arousals from sleep, the International Pediatric Work Group on Arousals published consensus-based definitions of arousals in infants aged 1 month to 6 months.[38] In NREM sleep they recommended scoring subcortical activation, in the absence of visible EEG shift, when 2 or more of the following criteria are observed: gross body movement on video or limb sensor, >10% increase in heart rate over baseline, or changes in frequency or amplitude of the breathing pattern (including even a single augmented breath). In REM sleep similar criteria were proposed to score subcortical activation, except that a change in breathing pattern was replaced by an increase in chin EMG tone.[38] The AASM Pediatric Task Force chose not to endorse scoring of subcortical activation until more evidence becomes available.[3]

Children

Movements as a surrogate for arousals have also been investigated in 15 children aged 2-11 years.[39] When only the 7-channel cardiorespiratory montage (one EKG lead; pulse rate; thoracic, abdominal and sum channel respiratory inductance plethysmography belts; oxygen saturation; and end tidal carbon dioxide) was used, compared to the typical 16-channel polysomnogram montage including EEG, 82.9±7.6% of arousals were identified. This suggests movements can be used to estimate (albeit likely underestimate) arousal indices in children who do not tolerate EEG leads. The addition of videotaping further improved differentiation of arousals into respiratory-related, technician-induced or spontaneous arousals.[39] As the current definition for hypopneas in children includes desaturation or arousal, perhaps with improved cardiorespiratory sensors that can better detect movement as a surrogate for cortical arousal, portable monitoring may become more useful in children in the near future. Such devices are currently under evaluation in adults, which include a small sensor pad located under the thorax, able to detect breathing movements and general body movements.[40]

Conclusion

Increasing evidence suggests that not only the amount, but also the quality of nocturnal sleep plays a critical role in a child's development, alertness, behavior, cognition, and health in many other respects. Furthermore, the similar behavioral outcome—inattentive and hyperactive behavior—across several unrelated types of sleep disorders provides further evidence that sleep fragmentation is likely to play an important mechanistic role between sleep disorders and their outcomes. Quantification of amounts of sleep obtained on a polysomnogram, and enumeration

of arousals with standard definitions have lent some uniformity to pediatric sleep medicine and facilitated communication between clinicians, investigators, and sleep laboratories. However, amounts of sleep and numbers of arousals are not likely to explain adequately the neurobehavioral morbidity commonly ascribed to the sleep disorders under study. Emerging technology may make a critical difference in the future. Initial data suggest that computer-assisted identification of nonvisible arousals, cyclic alternating patterns, or respiratory cycle-related EEG changes may complement what can be accomplished by human scorers. Focus on contiguous bouts of sleep or specific sleep stages may prove similarly useful. Incorporation of autonomic arousals measures—heart rate variability, pulse transit time, or peripheral arterial tone—into standard reports may capture subtle sleep fragmentation that focus on arousals missed by visible cortical EEG. In the future, research efforts to link such measures as well as simpler yet understudied variables, such as enumerated movements during sleep, to key patient outcomes may well improve the clinical utility and predictive value of polysomnography in children.

Acknowledgments

This work was supported through the following grant: HL080941

References

1. Lopes MC, Marcus CL. The significance of ASDA arousal in children. Sleep Medicine 2007;(9):3–8. [PubMed: 17638593]
2. Iber, C.; Ancoli-Israel, S.; Chesson, AL., et al. The AASM Manual for the Scoring of Sleep and Associated Events. American Academy of Sleep Medicine; West Chester, IL: 2007.
3. Grigg-Damberger M, Gozal D, Marcus CL, et al. The visual scoring of Sleep and arousal in infants and children; development of polygraphic features, reliability, validity, and alternative methods. J Clin Sleep Med 2007;3(2):201–240. [PubMed: 17557427]
4. American Sleep Disorders Association. EEG arousals: scoring rules and examples. SLEEP 1992;15:173–184. [PubMed: 11032543]
5. Wong TK, Galster P, Lau TS, Lutz JM, Marcus CL. Reliability of scoring arousals in normal children and children with obstructive Sleep apnea syndrome. SLEEP 2004;27:1139–1145. [PubMed: 15532208]
6. Goh DYT, Galster P, Marcus CL. Sleep architecture and respiratory disturbances in children with obstructive sleep apnea. Am J Respir Crit Care Med 2000;162:682–686. [PubMed: 10934106]
7. Tauman R, O'Brien LM, Holbrook CR, Gozal D. Sleep Pressure Score: a new index of sleep disruption in snoring children. SLEEP 2004;27(2):274–278. [PubMed: 15124722]
8. Scholle S, Zwacka G. Arousals and obstructive sleep apnea syndrome in children. Clin Neurophysiol 2001;112(6):984–991. [PubMed: 11377255]
9. Tal A, Bar A, Leiberman A, Tarastuk A. Sleep characteristics following adenotonssillectomy in children wih obstructive sleep apnea syndrome. Chest 2003;124:948–953. [PubMed: 12970022]
10. Kalyeyias J, Grant M, Darbari F, Ajagbe O, et al. Detection of cortical arousals in children using frontal EEG leads in addition to convention central leads. J Clin Sleep Med 2006;2:305–308. [PubMed: 17561542]
11. Roth M, Shaw J, Green J. The form, voltage distribution and physiological significance of the K-complex. Electroenceph Clin Neurophysiol 1956;8:385–402. [PubMed: 13330651]
12. Linden RD, Campbell KB, Hamel G, Picton TW. Ear Hear May-Jun;1985 6(3):167–174. [PubMed: 4007303]
13. Rechtschaffen, A.; Kales, A. A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects. National Institute of Neurological Disease and Blindness; Bethesda, MD: 1968.

14. Penzel T, Hirshkowitz M, Harsh J, Chervin RD, Butkov N, Kryger M, Malow B, Vitiello MV, Silber MH, Kushida CA, Chesson AL. Digital analysis and technical specification. J Clin Sleep Med 2007;3 (2):109–120. [PubMed: 17557421]
15. Bonnett MH, Doghramji K, Roehrs T, Stepanski EJ, Sheldon SH, Walters AS, Wise M, Chesson AL. The Scoring of arousal in sleep: reliability, validity and alternatives. J Cin Sleep Med 2007;3(2):133–145.
16. O'Brien LM, Tauman R, Gozal D. Sleep pressure correlates of cognitive and behavioral morbidity in snoring children. SLEEP 2004;27(2):279–282. [PubMed: 15124723]
17. Parrino L, Smerieri A, Rossi M, Terzano MG. Relationship of slow and rapid EEG components of CAP to ASDA arousal in normal sleep. SLEEP 2001;24:881–885. [PubMed: 11766157]
18. Terzano MG, Parrino L, Sherieri A, Chervin R, Chokroverty S, Guilleminault C, Hirshkowitz M, Mahowald M, Moldofsky H, Rosa A, Thomas R, Walters A. Atlas, rules, and recording techniques for the scoring of cyclic alternating pattern (CAP) in human sleep. Sleep Med 2001;6:537–553. [PubMed: 14592270]
19. McNamara F, Issa FG, Sullivan CE. Arousal pattern following central and obstructive breathing abnormalities in infants and children. J Appl Physiol 1996;81:2651–2657. [PubMed: 9018518]
20. Bandla HPR, Gozal D. Dynamic changes in EEG spectra during obstructive apnea in children. Pediatr Pulmonol 2000;29:359–365. [PubMed: 10790247]
21. Chervin RD, Burns JW, Subotic NS, Roussi C, Thelen B, Ruzicka DL. Method for detection of respiratory cycle-related EEG changes in sleep-disordered breathing. SLEEP 2004;27(1):110–115. [PubMed: 14998246]
22. Chervin RD, Burns JW, Subotic NS, Roussi C, Thelen B, Ruzicka DL. Correlates of respiratory cycle-related EEG changes in children with sleep-disordered breathing. SLEEP 2004;27(1):116–121. [PubMed: 14998247]
23. Chervin RD, Burns JW, Ruzicka DL. Electroencephalographic changes during respiratory cycles predict sleepiness in sleep apnea. Am J Resp Crit Care Med 2005;171:652–658. [PubMed: 15591467]
24. Chervin RD, Weatherly RA, Ruzicka DL, et al. Subjective sleepiness and polysomnographic correlates in children scheduled for adenotonsillectomy vs other surgical care. SLEEP 2006;29:495–503. [PubMed: 16676783]
25. Chervin RD, Malhotra RM, Burns JW. Respiratory cycle-related EEG changes during sleep reflect esophageal pressures. SLEEP 2008;31(12):1713–1720. [PubMed: 19090327]
26. Chervin RD, Fetterolf JL, Ruzicka DL, Thelen BJ, Burns JW. Sleep stage dynamics differ between children with and without obstructive sleep apnea. SLEEP. (in press).
27. D'Andrea LA, Rosen CL, Haddad GG. Severe hypoxemia in children wih upper airway obstruction during sleep does not lead to significant changes in heart rate. Pediatr Pulmonol 1993;16:362–369. [PubMed: 8134159]
28. Aljadeff G, Gozal D, Schechtman VL, Burrell B, Harper RM, Ward SL Davidson. Heart rate variability in children with obstructive sleep apnea. SLEEP 1997;20(2):151–157. [PubMed: 9143075]
29. Busek P, Vankova J, Opavsky J, et al. Spectral analysis of the variations in heart rate and cardiac activation on waking up in sleep walking. Rev Neurol 2005;41(6):338–343. [PubMed: 16163654]
30. Constantin E, McGregor CD, Cote V, Brouillette RT. Pulse Rate and Pulse Rate Variability Decrease After Adenotonsillectomy for Obstructive Sleep Apnea. Pediatric Pulmonology 2008;43:498–504. [PubMed: 18383115]
31. Stradling JR, Barbour C, Glennon J, Langford BA, Crosby JH. Prevalence of sleepiness and its relation to autonomic evidence of arousals and increased inspiratory effort in a community based population of men and women. J Sleep Res 2000;9:381–388. [PubMed: 11386205]
32. Pepin JL, Delavie N, Pin I, et al. Pulse transit time improves detection of sleep respiratory events and microarousals in children. Chest 2005;127:722–730. [PubMed: 15764750]
33. Katz ES, Lutz J, Black C, Marcus CL. Pulse transit time as a measure of arousal and respiratory effort in children with sleep disordered breathing. Pediatr Res 2003;53:580–588. [PubMed: 12612196]
34. Tauman R, O'Brien LM, Mast BT, Holbrook CR, Gozal D. Peripheral arterial tonometry events and electroencephalographic arousals in children. SLEEP 2004;27(3):502–506. [PubMed: 15164906]

35. O'Brien LM, Gozal D. Potential usefulness of noninvasive autonomic monitoring in recognition of arousals in normal healthy children. J Clin Sleep Med 2007;3(1):41–47. [PubMed: 17557452]

36. Zacharia A, Haba-Rubio J, Simon R, John G, Jordan P, Fernandes A, Gaspoz JM, Frey JG, Tschopp JM. Sleep apnea syndrome: improved detection of respiratory events and cortical arousals using oximetry pulse wave amplitude during polysomnography. Sleep Breath 2008;12:33–38. [PubMed: 17687577]

37. Fukumoto M, Mochizuki N, Takeishi M, et al. Studies of body movements during night sleep in infancy. Brain Dev 1981;3:37–43. [PubMed: 7258548]

38. IPWG. The scoring of arousals in healhy term infants (between the ages of 1 and 6 months). J Sleep Res 2005;14:37–41. [PubMed: 15743332]

39. Mograss MA, Ducharme FM, Broullette RT. Movement/Arousals. Description, classification, and relationship to sleep apnea in children. Am J Crit Care Med 1994;150:1690–1696.

40. Rauhala E, Virkkala J, Himanen SL. Periodic limb movement screening as an additional feature of Emfit sensor in sleep-disordered breathing studies. J Neurosci Methods 2009;178(1):157–161. [PubMed: 19100767]