Arousal pattern following central and obstructive breathing abnormalities in infants and children

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McNamara, Frances, Faiq G. Issa, and Colin E. Sullivan. Arousal pattern following central and obstructive breathing abnormalities in infants and children. J. Appl. Physiol. 81(6): 2651–2657, 1996.—We analyzed the polysomnographic records of 15 children and 20 infants with obstructive sleep apnea (OSA) to examine the interaction between central and obstructive breathing abnormalities and arousal from sleep. Each patient was matched for age with an infant or child who had no OSA. We found that the majority of respiratory events in infants and children was not terminated with arousal. In children, arousals terminated 39.3 ± 7.2% of respiratory events during quiet sleep and 37.8 ± 7.2% of events during active (rapid-eye-movement) sleep. In infants, arousals terminated 7.9 ± 1.0% of events during quiet sleep and 7.9 ± 1.2% of events during active sleep. In both infants and children, however, respiratory-related arousals occurred more frequently after obstructive apneas and hypopneas than after central events. Spontaneous arousals occurred in all patients with OSA during quiet and active sleep. The frequency of spontaneous arousals was not different between children with OSA and their matched controls. During active sleep, however, infants with OSA had significantly fewer spontaneous arousals than did control infants. We conclude that arousal is not an important mechanism in the termination of respiratory events in infants and children and that electroencephalographic criteria are not essential to determine the clinical severity of OSA in the pediatric population.

OBSTRUCTIVE SLEEP APNEA (OSA) is associated with repetitive arousals from sleep in the adult patient (23). Similarly, respiratory events in sleeping children are terminated with electroencephalographic (EEG) arousals or behaviorally determined movement arousal (14, 15). In infants, however, respiratory events are not usually associated with a behavioral arousal (22, 24), although EEG arousal has not been thoroughly examined in normal infants or infants with OSA.

Arousal is thought to be important for reestablishing a patent upper airway in adults (20) and is considered a protective reflex against life-threatening hypoxemia (15). It is induced by a variety of mechanisms, including changes in blood gases and stimulation of upper airway mechanoreceptors and respiratory muscle afferents (3, 4, 8, 11). Unlike in adult patients with OSA, full polysomnography with EEG has not been used widely in examining the role of arousal in terminating apnea in infants and children. In addition, the frequency of spontaneous arousals in normal infants and children has not been previously investigated. To investigate the interaction between sleep-disordered breathing and arousal in the pediatric population, we examined the association between obstructive and central breathing abnormalities occurring during each stage of sleep with arousal. In addition, we investigated the frequency of spontaneous arousals in this population.

METHODS

Patients

The polysomnographic records of 15 children (10 boys, 5 girls, age: 4.7 ± 1.1 yr, range: 1–14 yr) and 20 infants (10 boys, 10 girls, average age: 9.5 ± 1.3 wk, range: term corrected-21 wk) were selected for analysis. These patients represented infants and children who had undergone all-night polysomnographic studies for investigation of OSA (OSA group). The criteria for selection included 1) a minimum of 5 obstructive events/h of sleep in infants, defined according to the criteria used for adult OSA (≥5 s in duration); 2) a minimum of 2 obstructive events/h of sleep in infants, defined as cessation of breathing of ≥3 s. Nineteen of the infants of the OSA group were full-term. One infant, who was born premature, was matched for corrected age. All patients were neurologically normal and had no cardiovascular abnormalities on clinical examination at the time of study. We also selected a control group representing age-matched children (9 boys, 6 girls, mean age: 4.6 ± 1.1 yr) and infants (10 boys, 10 girls, mean age: 9.6 ± 1.3 wk), who were studied polysomnographically for OSA but were found to have normal polysomnographic studies and no obstructive apneas and hypopneas.

All-Night Polysomnography

All-night-sleep studies were performed in the National Sudden Infant Death Syndrome Council David Read Paediatric Sleep Disorders Unit in the presence of one parent. Recording of standard sleep and cardiopulmonary parameters was performed in each patient (1). Thus each subject was monitored with two EEGs (C3/A2, O2/A1, 10–20 international placement system), two electrocortiograms, and submental electromyogram (EMG). The electrocardiogram was measured continuously. Arterial oxyhemoglobin saturation (SaO2) was monitored continuously with a pulse oximeter by using a foot probe (Ohmeda Biox 3700E, Denver, CO). Airflow was measured using small infant nasal prongs placed near the nostrils. The tubing from the prongs was attached to a pressure transducer (Validyne DP103). Chest and abdominal wall movements were measured by using inductance plethysmography (Respitrace Ambulatory Monitoring, Ardsley, NY). For differentiating obstructive from central events, we recorded the diaphragm and abdominal EMG, using bipolar surface electrodes placed on the right subcostal region and the uppermost region of the left lower abdominal quadrant, respectively. All signals were amplified, filtered and recorded on a Grass multichannel polygraph recorder (model 8, Grass Instruments). Audio-video recording of the infant/child was performed continuously during the study using a video camera (Panasonic, Tokyo, Japan). The time-coded sound and images were recorded on a VHS-FM tape for later analysis.
The study commenced at the time the subjects normally went to sleep at night, usually between 7:00 and 9:00 P.M., and was terminated at ∼6:00 A.M. The infants were fed and changed by the attending parent when they woke spontaneously during the night, similar to their routine at home. All subjects were observed throughout the study by the sleep technician who marked all movements, changes in body position, crying, and nursing interventions on the polygraph paper. Arousal caused by external stimuli, e.g., noise, were also noted on the polygraph paper.

Analysis

The polysomnographic record was initially analyzed for sleep stages and wakefulness. Sixty-second epochs were analyzed and assigned as either awake, quiet (non-rapid-eye-movement) sleep, or active (rapid-eye-movement) sleep. Sleep was staged according to the standard criteria for neonates and infants up to ∼4 mo of age (2) and according to the criteria for older infants and children (19). The time spent in quiet and active sleep and the total sleep time were calculated for each subject.

Apneas represented cessation of breathing lasting 3 s or longer in infants, similar to the criteria used by other investigators (10). Apneas in children represented cessation of breathing for at least 5 s, similar to criteria used by Praud et al. (17). Apneas in adolescents were scored according to the adult criteria, i.e., cessation of breathing for 10 s associated with a decrease in SaO2 of at least 4%. Hypopneas were defined as a decrease in amplitude of the airflow signal by ≥50%, accompanied by desaturation (≥4%). Furthermore, hypopneas were considered obstructive when flow-limitation pattern was observed or when the reduction in flow was associated with paradoxical rib cage and abdominal movements or with increased respiratory efforts represented by increased thoracoabdominal motion and EMG signal, but they were considered central in type when the diminished airflow was associated with reduced EMG activity and chest and abdominal wall movement. The total number of apneas and hypopneas was calculated for each sleep state, and they were separated according to event type, i.e., into central and obstructive events. Central events included central apneas and hypopneas, whereas obstructive events included mixed apneas, obstructive apneas, and obstructive hypopneas. A respiratory disturbance index (RDI), expressed as events per hour, was calculated for each event type during each sleep state for each group. The length of each respiratory event was also recorded and averaged for each sleep state and event type. The SaO2 level at the end of apneas and hypopneas was recorded and averaged for each sleep state and apnea type.

Because of lack of standard definitions of arousal in infants, children, and adolescents, we used the criteria defined by other investigators and those used for adult polysomnography (21). Arousal was defined as an abrupt shift in EEG frequency from sleep to wakefulness pattern. Arousal from active sleep was set to include, in addition to a shift in EEG pattern, a sudden augmentation of submental EMG amplitude. The change in EEG frequency at arousal included the appearance of theta or alpha rhythm or frequencies >16 Hz and followed at least 10 s of continuous sleep. The minimum duration of an arousal was regarded as 1 s, similar to the criteria previously used in children (14). Arousals were classified as either one of two types: respiratory-related or spontaneous arousals. Respiratory arousals were those occurring during or immediately after an apnea or hypopnea associated with desaturation. A spontaneous arousal was not associated with a respiratory event. Arousals that occurred in response to noise or other external stimuli were not included in the present analysis. The routine use of audio-visual recordings allowed identification of such externally induced arousals. An arousal index, expressed as the number of arousals per hour, was calculated for both respiratory and spontaneous arousals for quiet and active sleep and according to apnea type. The percentage of respiratory events inducing arousal in each sleep state was also calculated for each event type.

Statistical Analysis

Data are expressed as means ± SE. Differences between respiratory events that were and were not associated with arousal and between OSA and control groups were analyzed by using the unpaired t-test. Differences between the frequency of arousals associated with central and obstructive events were analyzed by using the $\chi^2$ test. The relationship between factors determining arousal and the arousal index was examined by using a linear-regression analysis. A P value of <0.05 was considered significant.

RESULTS

Sleep and Respiratory Events

Central and obstructive events were recorded in all infants and children of the OSA group, but the severity of sleep disorders varied from one patient to another. On the other hand, central events and occasional or no obstructive events were recorded in control children and infants. The time spent in each sleep stage and the RDI in OSA infants and control subjects are summarized in Table 1, whereas the data for OSA and control children are shown in Table 2. All subjects slept well in the laboratory with long epochs in both quiet and active sleep. As evident in Tables 1 and 2, obstructive apneas were more frequent during active than during quiet sleep in both infants and children. Notably, the duration of active sleep was significantly less in both OSA groups compared with the respective control group.

Respiratory-Related Arousals

OSA group. CHILDREN. Figure 1 shows a typical example of arousal associated with obstructive apnea.

Table 1. Respiratory and sleep data for infants with OSA and control infants

<table>
<thead>
<tr>
<th></th>
<th>OSA</th>
<th>Control Subjects</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quiet sleep time, h</td>
<td>6.3 ± 0.2</td>
<td>5.2 ± 0.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Active sleep time, h</td>
<td>1.3 ± 0.1</td>
<td>2.2 ± 0.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Awake time, h</td>
<td>2.1 ± 0.2</td>
<td>2.3 ± 0.3</td>
<td>NS</td>
</tr>
<tr>
<td>Active sleep, %</td>
<td>16.9 ± 11</td>
<td>29.8 ± 0.8</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Central RDI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quiet sleep</td>
<td>30.6 ± 5.2</td>
<td>10.6 ± 1.3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Active sleep</td>
<td>44.3 ± 6.3</td>
<td>18.1 ± 2.4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Total</td>
<td>32.2 ± 5.3</td>
<td>12.8 ± 1.4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Obstructive RDI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quiet sleep</td>
<td>5.9 ± 1.1</td>
<td>0.2 ± 0.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Active sleep</td>
<td>21.2 ± 5.7</td>
<td>0.7 ± 0.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Total</td>
<td>8.7 ± 1.4</td>
<td>0.3 ± 0.1</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Values are means ± SE. OSA, obstructive sleep apnea; RDI, respiratory disturbance index; NS, not significant. *Significantly different compared with OSA children.
The mean respiratory arousal index in OSA children was 7.0 ± 2.6 arousals/h during quiet sleep and 16.0 ± 3.2 arousals/h during active sleep. The proportion of respiratory events in quiet sleep terminating in arousal (mean: 39.3 ± 5.9%, range: 10.0–77.4%) was not different from those recorded during active sleep (mean: 37.8 ± 7.2%, range: 4.2–100%).

We also examined the possible factors contributing to respiratory-related arousals, including age, type and duration of the respiratory event, sleep stage, and the level of arterial oxyhemoglobin desaturation. The number of respiratory-related arousals in quiet, but not in active, sleep correlated with age (Fig. 2, A and C). It was noteworthy that the child with the highest proportion of respiratory events terminating by arousal was the oldest child in the group (14 yr old). Conversely, the

The mean respiratory arousal index during quiet and active sleep was 3.9 ± 0.4 arousals/h during quiet sleep and 3.1 ± 0.5 arousals/h during active sleep. The proportion of respiratory events in quiet sleep terminating in arousal (mean: 23.6 ± 1.5%, range: 10.0–77.4%) was not different from those recorded during active sleep (mean: 20.5 ± 1.1%, range: 14.7–25.0%). There was no difference in the number of respiratory events associated with arousal during quiet and active sleep.

Table 2. Respiratory and sleep data for children with OSA and control subjects

<table>
<thead>
<tr>
<th></th>
<th>OSA</th>
<th>Control</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quiet sleep time, h</td>
<td>5.5 ± 0.4</td>
<td>5.3 ± 0.4</td>
<td>NS</td>
</tr>
<tr>
<td>Active sleep time, h</td>
<td>1.4 ± 0.1</td>
<td>2.2 ± 0.3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Awake time, h</td>
<td>1.2 ± 0.3</td>
<td>1.1 ± 0.2</td>
<td>NS</td>
</tr>
<tr>
<td>Active sleep, %</td>
<td>19.4 ± 1.2</td>
<td>23.6 ± 1.0</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Central RDI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quiet sleep</td>
<td>3.2 ± 0.5</td>
<td>2.5 ± 0.4</td>
<td>NS</td>
</tr>
<tr>
<td>Active sleep</td>
<td>7.4 ± 1.1</td>
<td>7.0 ± 1.5</td>
<td>NS</td>
</tr>
<tr>
<td>Total</td>
<td>3.9 ± 0.4</td>
<td>3.1 ± 0.5</td>
<td>NS</td>
</tr>
<tr>
<td>Obstructive RDI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quiet sleep</td>
<td>10.8 ± 3.0</td>
<td>36.0 ± 4.7</td>
<td></td>
</tr>
<tr>
<td>Active sleep</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>15.5 ± 2.8</td>
<td></td>
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</table>

Values are means ± SE. *Significantly different compared with OSA children.

The mean lowest SaO2 in infants was 7.9 ± 1.0% during quiet sleep (range: 2.1–20.5%). Similarly, during active sleep, 7.9 ± 1.2% of respiratory events were terminated with an arousal (range: 2.4–25.0%). There was no difference between the number of events inducing arousal during quiet and active sleep (P > 0.05, t-test).

As in children, the incidence of arousal in infants was significantly greater in response to obstructive than to central events during both quiet and active sleep. Although arousal occurred at the end of only 178 of 3,900 (4.6%) central events, it was recorded in 133 of 742 (17.9%, P < 0.05) obstructive events. Similarly, fewer central events (66 of 1,165, 5.7%) were associated with arousal during active sleep compared with 56 of 480 obstructive events (11.7%) terminating with arousal (P < 0.05). The mean lowest SaO2 and mean duration of the respiratory events in infants with OSA were similar for central and obstructive events terminated with or without arousal (P > 0.05, Table 3). Unlike the relationship found in children, we could not detect a significant relationship between age and the respiratory arousal index in infants (Fig. 2, B and D).

Control subjects. The mean arousal index associated with central events in control children was 0.2 ± 0.1 and 0.2 ± 0.1 in quiet and active sleep, respectively. Only 10 of 185 (5.4%) and 5 of 169 (3.0%) central events in quiet and active sleep, respectively, were terminated with arousal in control children. The mean arousal index in quiet sleep in control infants was 0.3 ± 0.2, 0.1 ± 0.1, and 0.4 ± 0.1 for central apnea, mixed apnea,
and total average, respectively. The mean arousal index in active sleep in control infants was $1.0 \pm 0.2$, $0.2 \pm 0.1$, and $1.2 \pm 0.3$ for central apnea, mixed apnea, and total average, respectively. The number of arousals associated with obstructive apneas in quiet sleep ($8$ of $14$, $57.1\%$) was significantly higher than that with central apneas ($33$ of $1,061$, $3.1\%$, $P < 0.05$) recorded during the same sleep stage in control infants. During active sleep, the proportion of obstructive apneas terminating with arousal ($8$ of $25$, $32.0\%$) was also significantly higher than that in central apneas ($42$ of $795$, $5.3\%$, $P < 0.05$).

**Spontaneous Arousals**

Spontaneous arousals occurred in all subjects during quiet and active sleep. The number of spontaneous arousals in quiet and active sleep in children with OSA was not different from that of control subjects. During quiet sleep, children with OSA had $7.1 \pm 0.8$ arousals/h, whereas the control subjects had $6.8 \pm 1.5$ arousals/h ($P > 0.05$, t-test). During active sleep, children with OSA had $9.5 \pm 1.3$ arousals/h, whereas $7.1 \pm 0.9$ arousals/h were recorded in control subjects ($P > 0.05$, t-test).

The mean number of spontaneous arousals during quiet sleep in infants with OSA ($9.1 \pm 0.9$ arousals/h) was similar to that in control subjects ($8.9 \pm 0.7$ arousals/h, $P > 0.05$, t-test). In contrast, normal infants had significantly more spontaneous arousals during active sleep ($19.1 \pm 1.4$ arousals/h) compared with infants with OSA ($11.6 \pm 0.9$ arousals/h, $P < 0.05$, t-test).

**DISCUSSION**

The major findings of the present study are that 1) arousal responses are not the dominant mechanisms terminating apnea in infants and children with sleep-disordered breathing, 2) obstructive apneas are more

**Table 3. Central and obstructive event lengths and $\text{SaO}_2$ during quiet and active sleep for children and infants**

<table>
<thead>
<tr>
<th></th>
<th>Children</th>
<th>Infants</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Arousal</td>
<td>No arousal</td>
</tr>
<tr>
<td><strong>Quiet sleep</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\text{SaO}_2$, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central</td>
<td>$89.8 \pm 0.7$</td>
<td>$89.8 \pm 0.7$</td>
</tr>
<tr>
<td>Obstructive</td>
<td>$85.2 \pm 1.3$</td>
<td>$86.9 \pm 1.3$</td>
</tr>
<tr>
<td>Length, s</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central</td>
<td>$12.0 \pm 0.8$</td>
<td>$10.2 \pm 0.6$</td>
</tr>
<tr>
<td>Obstructive</td>
<td>$13.7 \pm 1.2$</td>
<td>$11.9 \pm 0.9$</td>
</tr>
<tr>
<td><strong>Active sleep</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\text{SaO}_2$, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central</td>
<td>$87.6 \pm 1.1$</td>
<td>$86.9 \pm 1.5$</td>
</tr>
<tr>
<td>Obstructive</td>
<td>$82.6 \pm 2.1$</td>
<td>$85.0 \pm 1.6$</td>
</tr>
<tr>
<td>Length, s</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central</td>
<td>$9.4 \pm 0.8$</td>
<td>$8.5 \pm 0.6$</td>
</tr>
<tr>
<td>Obstructive</td>
<td>$16.1 \pm 1.4$</td>
<td>$13.4 \pm 1.2$</td>
</tr>
</tbody>
</table>

Values are means $\pm$ SE. $\text{SaO}_2$, arterial oxyhemoglobin saturation.

Fig. 2. Relationship between respiratory arousal index and age in children (A and C) and infants (B and D). A and B: quiet (non-rapid-eye-movement) sleep. C and D: active (rapid-eye-movement) sleep.
likely to interrupt sleep than central apneas, and 3) in a sleep-laboratory setting, the arousal index in infants and children with sleep apnea is, in general, not worse than that in control children.

Our results demonstrated that in pediatric patients respiratory events were often terminated spontaneously, without an EEG arousal. Although arousal at the termination of an event was relatively uncommon, it occurred more frequently in association with obstructive events than with central events. Obstructive sleep apnea in adults is associated with arousal, which is thought to reestablish upper airway patency with the return of upper airway muscle dilator tone (15, 20). Although our results indicate that arousal-linked changes are not the dominant mechanism terminating apnea, the duration of apneas associated with arousal was longer compared with self-terminating apneas. This finding suggests that mecanochemical reflexes, rather than stimuli originating from the peripheral chemoreceptors, contributed to arousal, e.g., CO2, upper airway, or thoracic muscle mechanoreceptors (8, 11, 22).

The lack of correlation between SaO2 and arousal strongly implied that carotid chemoreceptor inputs do not play a dominant role in terminating apnea in infants and children. Although hypoxia, by stimulating carotid body afferents, has been shown to be a powerful arousal-promoting mechanism in sleeping dogs (16), it is known that the arousal response to eucapnic hypoxia is less clearly defined in adult humans (3, 4). In adult humans, it appears that the gain of the carotid body plays a critical role, because hypercapnia and hypoxia readily and rapidly induce arousal in normal humans (9). The results of the present study are similar to those of Davidson Ward et al. (7), who performed hypoxic challenges in normal infants and found that, although all infants displayed a ventilatory response to the hypoxic stimulation, arousal was uncommon despite decreases in SaO2 to levels <75%.

What are the mechanisms that allow a spontaneous resolution of obstructive apneas, without arousal, in children but not in adults? The likely mechanism that terminates obstructive apneas in infants and children is that neuromuscular drive increases sufficiently to overcome upper airway closure, as suggested previously by Stark and Thach (22). Alternatively, the apneic events are the result of central neuronal respiratory control instability, such that the duration of each event is predetermined and not dependent on any subsequent reflex response at the end of apnea. Certainly, the latter is likely to be the case for the common pattern of recurrent central apnea, which occurs in infantile periodic breathing. Although the present study did not provide a direct evidence for such mechanisms, our results demonstrated that age had a direct effect on arousal, i.e., apneas in children were more likely to induce arousal, at least those occurring during quiet sleep, in older than in younger children. Although the neural mechanisms controlling sleep and wakefulness are poorly understood, it is generally understood that the neuronal network involved in such process extends throughout several areas of the central nervous system. Maturational changes may alter the mechanism of and response to apnea from one dominated by central neural control instability to another, dominated by peripheral reflex responses. Alternatively, local neuromechanical factors acting on upper airway structures may allow the infant and child to generate sufficient muscle tension to reopen the collapsed upper airway well before arousal occurs.

It is possible that the method used in the present study for scoring arousals in infants and children may underestimate the number of arousals. Previous investigators (14, 22) demonstrated using EEG criteria and video monitoring that most obstructive events in children were terminated with an arousal. Other investigators found that during quiet sleep, 12% of apneic events in prepubertal children were terminated with an EEG arousal, whereas the remaining events were terminated with “movement” arousal, and during active sleep all apneic events were terminated with movement arousal without a change in the EEG pattern (17). Although movement arousal without EEG arousal pattern still needs further definition, as recommended by the recent American Thoracic Society Assembly (1), in the present study, we only examined respiratory events terminated by an EEG arousal.

An alternative explanation for the lack of EEG arousal in infants is the relative insensitivity of surface EEG to detect arousal in this age group. A growing body of evidence related to seizures indicates that epileptic discharges generated in the subcortical areas (deep limbic, diencephalic, and brain stem) can occur in the absence of surface EEG discharge. Hence, it is possible for “respiratory” component of arousal (sudden increase in neuromuscular drive) to occur in the absence of EEG or behavioral changes. This phenomenon may represent a protective mechanism to minimize the ill effects of frequent arousals in the developing brain.

Spontaneous arousals occurred in all sleep stages in infants and children. It is possible that some of the spontaneous arousals were due to undetected respiratory events, since partial upper airway obstruction is also known to cause arousal from sleep. This is un-
likely, however, since spontaneous arousals were not different between children with OSA and age-matched controls. Contrary to the results in children, the number of spontaneous arousals during active sleep in infants with OSA was less than that recorded in age-matched controls. This interesting difference may be consistent with previous findings in infants with obstructive breathing abnormalities; such infants had fewer movements per hour of sleep and, therefore, likely, fewer arousals per hour of sleep (12). Repeated arousals during sleep can result in sleep fragmentation, which, in turn, can cause depression of arousal responses (5). Although our data suggest a reduction in the number of spontaneous arousals during active sleep in infants, we cannot determine whether this phenomenon is caused by a depression of arousal. However, it could be evidence for a selective depression of arousability in active sleep. This is consistent with our previous work which showed that obstructive events in infants can reduce overall active sleep, a phenomenon that is reversed by nasal continuous positive airway pressure (13).

Spontaneous arousals in children with OSA and in age-matched controls occurred on average every 6–10 min and in infants with OSA and controls every 3–6 min during quiet and active sleep. Although the arousals were usually transient, this level of sleep disturbance may be expected to result in sleep fragmentation in the children and infants studied. The sleeping pattern of infants is known to be fragmented, consisting of several episodes of sleep during the 24-h period (6). It is possible that the sleep study montage and the extent of instrumentation used may have influenced the number of spontaneous arousals. Previous investigators, however, described a lack of night-to-night variability of sleep and breathing variables during full polysomnographic studies in infants (18). In addition, it has also been shown that arousals in infants with and without instrumentation in the sleep laboratory were similar (24). Because the number of spontaneous arousals in quiet and active sleep in children and during quiet sleep in infants in the present study was similar to their respective control groups, we believe that the effects of the polygraphic study on sleep were minimum.

The results of the present study indicate that EEG arousal is not an important phenomenon in OSA of infants and children. At present, polysomnography is considered the gold standard for the diagnosis of OSA in children and infants (1, 10). Such studies allow recording of sleep signals, including EEG and electrooculogram, and measurement of breathing, including airflow, EMG, respiratory movements, and blood gas variables. The polysomnographic study provides the means to calculate the frequency of arousals in response to apneic events and, therefore, gives an indication of sleep fragmentation. Our results suggest, however, that reliance on EEG as an indicator may be misleading by giving an underestimate of the clinical severity of sleep-disordered breathing in infants and children.

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