Obstructive Sleep Apnea in Infants and Its Management With Nasal Continuous Positive Airway Pressure*

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Study objectives: Nasal continuous positive airway pressure (nCPAP) is the most common treatment for obstructive sleep apnea (OSA) in adults, and it has been effective in the treatment of OSA in children. We wanted to determine the effectiveness of long-term nCPAP therapy for OSA in infants.

Patients: Twenty-four infants who had OSA were treated with nCPAP via nose mask. These infants had clinical histories that included a family history of sudden infant death syndrome, an apparent life-threatening event, or facial and upper airway anatomic abnormalities.

Interventions: Overnight polysomnographic studies were performed to assess the severity of OSA in each infant and to determine the appropriate level of continuous positive airway pressure (CPAP). Studies were repeated to determine the progress of OSA and the continuing need for CPAP in each infant.

Results: nCPAP pressures between 4 and 6 cm H2O prevented obstruction and reversed sleep disturbances that were associated with OSA. Eighteen of the infants continued treatment at home from 1 month to > 4 years. CPAP therapy was discontinued in 13 infants after their OSA resolved. Five infants who have upper airway anatomic abnormalities remain on CPAP, and the pressure level required to prevent obstructive events during sleep has needed to be increased to as high as 10 cm H2O.

Conclusions: nCPAP is an effective therapy for the management of OSA in infants, and it can be used effectively in the home environment. Regular follow-up is necessary, because the requirements for CPAP and pressure levels change with the infant’s growth and development.

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Key words: infantile apnea; nasal continuous positive airway pressure; obstructive sleep apnea; sleep fragmentation

Abbreviations: ALTE = apparent life-threatening event; CPAP = continuous positive airway pressure; EMG = electromyogram; EOG = electro-oculogram; nCPAP = nasal continuous positive airway pressure; NREM = nonrapid eye movement; OSA = obstructive sleep apnea; PD = pressure determination; REM = rapid eye movement; SaO2 = arterial oxygen saturation; SIDS = sudden infant death syndrome; SWS = slow-wave sleep

Nasal continuous positive airway pressure (nCPAP) via nose mask is the treatment of choice for the obstructive sleep apnea (OSA) syndrome in adults, and nCPAP has been demonstrated to effectively treat OSA in children. nCPAP via nose mask has been shown to prevent OSA and to improve daytime function in these patients, and it has been a successful long-term treatment in the home setting. Sleep apnea is recorded commonly in infants, it is described as central in nature, and it is believed to be associated with immaturity of the CNS. OSA, on the other hand, is thought to be rare in infants. OSA has, however, been recorded in infants who are considered to have an increased risk for sudden infant death syndrome (SIDS) in preterm infants, and in infants who have syndromes that involve craniofacial and upper airway abnormalities, such as Pierre Robin, Apert’s,
and Crouzon’s syndromes.10,11 The treatment of upper airway anatomic abnormalities in infants has previously included surgery (for example, tracheostomy) or the placement of a nasopharyngeal airway.11 Other infants have undergone home monitoring of cardiorespiratory functions using an alarm system to detect prolonged apnea and bradycardia, and their OSA has remained untreated.

nCPAP has been successful as a treatment for OSA in adults and children in the home; however, its use as a long-term therapy for infants is limited. nCPAP via mask has been used to treat OSA6,7 and the upper airway resistance syndrome8 in infants considered to have an increased risk for SIDS and in infants with congenital abnormalities. These investigators showed that treatment with nCPAP via mask prevented obstruction, reduced the work of breathing, decreased the amount of central apnea in nonrapid eye movement (NREM) sleep, and reversed the sleep disturbances associated with infantile OSA. Long-term follow-up of OSA in infants and its treatment with nCPAP via mask has not been studied.

We have now used nCPAP via mask to treat OSA in 24 infants. These patients presented to the sleep disorders clinic with a variety of clinical histories, and their conditions were subsequently diagnosed as OSA in an overnight sleep study. We used nCPAP via mask to determine whether we could effectively treat OSA in infant patients who possibly have a variety of mechanisms of upper airway obstruction. We wanted to determine whether nCPAP could be used as a long-term therapy in the infant’s home and whether it could be considered as a possible alternative to more common methods of treatment, such as a nasopharyngeal airway. We also wanted to determine the effects of increasing age and development on both OSA severity and continuous positive airway pressure (CPAP) requirements in infants.

### Materials and Methods

Studies were performed on 24 infants (15 boys and 9 girls from 1 to 51 weeks old), who had been referred to the sleep disorders clinic for investigation of OSA (Table 1). The infants were selected for CPAP treatment because they had mixed and obstructive apneas of > 5 apneas/h of sleep that were recorded during an overnight polysomnographic study (in 21 infants) or an overnight ambulatory study (in 3 infants). The mean (± SEM) gestation age was 37.6 ± 0.7 weeks old (range, 30 to 42 weeks old); 16 infants had been born at full term.

#### Polysomnographic Sleep Studies

The polysomnographic data were recorded on a 12-channel EEG polygraph recorder (model 8; Grass Instrument; Quincy, MA). For each patient, sleep was monitored with 2 channels of EEG (C3/A2 and O2/A1; 10–20 international placement system); 2 channels of electro-oculogram ([EOG]; ie, left and right eye movements); and submental electromyogram (EMG). Diaphragm and abdominal EMG were also measured. The ECG was measured continuously. Pulse oximetry (Biox model 3700; Datex-Ohmeda; Helsinki, Finland) using a foot probe was measured continuously as an indication of arterial oxygen saturation (SaO2). Transcutaneous CO2 was measured continuously with a transcutaneous probe (model TCM3; Radiometer; Copenhagen, Denmark) as an indication of PCO2. Airflow was measured using small infant nasal prongs that were placed in the infant’s nostrils and then attached to a pressure transducer (model DP103; Validyne Engineering; Northridge, CA). Chest wall and abdominal movements were measured using inductance plethysmography (Respirac; Ambulatory Monitoring; Ardsley, NY). The ambulatory diagnostic study in three of the infants was performed overnight using a 4-channel monitor (Edentrace II; Nellcor Puritan Bennett; Pleasanton, CA), and it included measurements of nasal and oral airflow, chest impedance, heart rate, and SaO2.

Each patient had at least two sleep studies: one to assess the severity of sleep-disordered breathing and another to determine the adequate pressure level of CPAP. All infants underwent further polysomnographic studies and clinical assessments at 2-month to 4-month intervals until they reached 1 year of age; thereafter, assessments at 6-month follow-up intervals were made to determine their continued needs for nCPAP. Each study was started at the time the infant normally went to sleep for the night, usually between 7:00 PM and 9:00 PM. Each study was stopped at 6:00 AM the following morning. All patients were observed

#### Table 1—Patient History and CPAP Treatment Details

<table>
<thead>
<tr>
<th>History</th>
<th>No. of Infants</th>
<th>Age CPAP Started, wk</th>
<th>Original Pressure, cm H2O</th>
<th>Current Pressure, cm H2O</th>
<th>Time on CPAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family history of SIDS</td>
<td>5</td>
<td>9–12</td>
<td>4.0–6.0</td>
<td>—</td>
<td>1–9 mo</td>
</tr>
<tr>
<td>ALTE</td>
<td>8</td>
<td>4–28</td>
<td>4.0–6.0</td>
<td>—</td>
<td>6 wk–5 mo</td>
</tr>
<tr>
<td>Anatomic abnormalities, total</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pierre Robin syndrome</td>
<td>1</td>
<td>1</td>
<td>4.5</td>
<td>8.5</td>
<td>3 yr</td>
</tr>
<tr>
<td>Micrognathia</td>
<td>3</td>
<td>21–51</td>
<td>3.7–5.0</td>
<td>6.5 and 10</td>
<td>1 wk–4 yr</td>
</tr>
<tr>
<td>Micrognathia, macroglossia</td>
<td>1</td>
<td>38</td>
<td>4.5</td>
<td>7.0</td>
<td>4.3 yr</td>
</tr>
<tr>
<td>Micrognathia, facial clefts</td>
<td>1</td>
<td>33</td>
<td>5.0</td>
<td>7.0</td>
<td>3.5 yr</td>
</tr>
<tr>
<td>Choanal atresia</td>
<td>1</td>
<td>37</td>
<td>4.5</td>
<td>—</td>
<td>2 yr</td>
</tr>
<tr>
<td>Laryngomalacia</td>
<td>1</td>
<td>10</td>
<td>3.7</td>
<td>—</td>
<td>1 wk</td>
</tr>
<tr>
<td>Syndromes, total</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beckwith-Wiedemann</td>
<td>1</td>
<td>15</td>
<td>3.7</td>
<td>—</td>
<td>1 wk</td>
</tr>
<tr>
<td>Smith-Lemli-Opitz</td>
<td>1</td>
<td>51</td>
<td>4.5</td>
<td>—</td>
<td>7 mo</td>
</tr>
<tr>
<td>Moebius</td>
<td>1</td>
<td>25</td>
<td>5.0</td>
<td>—</td>
<td>1 wk</td>
</tr>
</tbody>
</table>
throughout each study by the nursing staff. Any movements, changes in body position, crying, mouth breathing, and nursing interventions were recorded on the polygraph paper.

**nCPAP Studies**

The appropriate level of CPAP for each infant was determined during an all-night CPAP pressure determination (PD) study. A polysomnographic study was performed with the same setup as the diagnostic study, except that nCPAP was applied during sleep. The nCPAP equipment included a commercially available CPAP machine, to which was attached a small infant CPAP mask (Sullivan APDII; ResMed; Sydney, Australia). The mask was fitted over the infant's nose and secured with a head strap (RemCap; ResMed).

The nCPAP was started at the lowest pressure (3.7 cm H2O) and was gradually increased by small (0.3 cm H2O) increments until the obstructive events were prevented. As the pressure was increased, the breathing patterns and CO2 measurements were monitored carefully. The optimal pressure was the level that minimized obstruction and did not increase the CO2 level or the length of central apneas.

The infants who continued to use nCPAP at home were treated with the pressure level that was determined during the CPAP PD study, and the parents were asked to administer nCPAP to their infants during all sleeping periods, including the daytime naps.

**Data Analysis**

Sleep was staged according to established criteria for neonates and infants. Sixty-second epochs were assigned as the following: awake, NREM, or rapid eye movement (REM) sleep. NREM sleep was further subdivided as follows: slow-wave sleep (SWS), corresponding to stage III/IV NREM sleep, and lighter sleep, corresponding to stage I/II NREM sleep. Total sleep time, stage I/II NREM, SWS, and REM sleep times, and the percentage of total sleep time spent in each sleep state were calculated. The number and length of episodes of each sleep state were calculated and averaged. An apnea was defined as a cessation of respiration for a duration of at least two respiratory cycles (approximately 3 to 5 s in length). This criterion is similar to that used by other investigators. Apneas were classified as either central, mixed, or obstructive, according to the absence or presence of respiratory efforts during the apnea. Central apneas were defined as flat tracings on airflow and plethysmography and absent diaphragm EMG activity. Obstructive apneas were defined as flat tracings on airflow, persisting movements on thoracic and abdominal plethysmography, and continuing diaphragm EMG activity. Mixed apneas were apneas that had a central component and an obstructive component. Obstructive and mixed apneas were combined as an obstructive index in the analysis. For each study, the number of apneas were totaled and expressed as an apnea index (apneas/h) for each sleep state and apnea type. Decreases in SaO2 by at least 4% that were associated with a respiratory event were also recorded. A desaturation index was calculated (desaturations/h) for each sleep state.

The respiratory and sleep variables from all infants were combined and averaged according to sleep state and event type, and they were compared according to sleep study type (diagnostic or CPAP study). The differences between data from diagnostic studies and CPAP studies were analyzed using the rank-sum test and paired t test. All data are expressed as mean (± SEM). A p value < 0.05 was considered significant.

**RESULTS**

**Effects of nCPAP on Sleep Apnea and Sleep**

All infants had obstructive and central events recorded on their diagnostic study that were associated with decreases in SaO2 (Fig 1). Sixteen of the 24 infants had a CPAP PD study the night after their diagnostic study. The remaining infants were treated with low-level nCPAP (3.7 cm H2O) during sleep as inpatients of the hospital, until their CPAP PD study could be performed. In total, 21 of the infants underwent a CPAP PD study within 2 weeks of their diagnostic study. Of these 21 infants, 18 had undergone a polysomnographic diagnostic study previously, and the other 3 infants had an ambulatory study performed before the CPAP PD.

nCPAP between 4 and 6 cm H2O was required to treat OSA in these infants (Fig 2). The total amount of apneas recorded during NREM and REM sleep was significantly less during the CPAP PD study than during the previous diagnostic study (rank-sum test, p < 0.05; Table 2). The reduction in the total amount of apnea during NREM and REM sleep was mainly because of the significant reduction in the number of obstructive events (Table 2). The number of central events was reduced during NREM sleep with CPAP therapy (Table 2). During REM sleep, the amount of central events was reduced in 11 infants and increased in 7 infants during the CPAP
CPAP therapy was also associated with changes in the sleeping pattern of each infant. During the diagnostic study, sleep was fragmented with short episodes of NREM and REM sleep. The total amount of sleep was similar for the diagnostic and CPAP studies; however, the total amount of each sleep stage was significantly different during the CPAP studies; there was no evidence of obstruction. See Figure 1 legend for expansion of abbreviations.

![EEG](https://example.com/eegelectrode.png)

**Figure 2.** Polygraphic example of the respiratory pattern in REM sleep during the application of nCPAP in the same infant as in Figure 1. Note that the respiratory pattern is irregular, but the sleeping pattern of each infant. During the CPAP study, sleep was fragmented with short episodes of REM sleep. The total amount recorded previously. During the CPAP study, sleep was fragmented with short episodes of NREM and REM sleep. The proportion of sleep spent in REM sleep and the duration of individual REM sleep episodes during the CPAP PD study were approximately twice those recorded during the diagnostic study (Table 2). The increases in amount of REM sleep resulted in a decreased amount of NREM sleep. The proportion of sleep spent in SWS was similar during both studies, but there was a significant decrease in the amount of stage I/II NREM sleep during the CPAP PD study. Despite the overall decrease in NREM sleep, nCPAP treatment was associated with an increased length of NREM sleep episodes (Table 2).

**Table 2—Average Respiratory and Sleep Variables for Diagnostic and CPAP Studies**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Diagnostic Study</th>
<th>CPAP Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time, min</td>
<td>488.6 ± 14.6</td>
<td>445.1 ± 14.7</td>
</tr>
<tr>
<td>Mean length NREM episodes, min</td>
<td>15.9 ± 1.0</td>
<td>21.6 ± 1.4</td>
</tr>
<tr>
<td>Mean length REM episodes, min</td>
<td>6.3 ± 0.6</td>
<td>13.2 ± 0.8</td>
</tr>
<tr>
<td>% REM sleep</td>
<td>16.0 ± 1.2</td>
<td>28.8 ± 0.9</td>
</tr>
<tr>
<td>% SWS</td>
<td>34.2 ± 1.9</td>
<td>37.5 ± 2.5</td>
</tr>
<tr>
<td>% stage I/II NREM</td>
<td>49.8 ± 1.6</td>
<td>33.7 ± 1.9</td>
</tr>
<tr>
<td>NREM</td>
<td>44.4 ± 9.3</td>
<td>9.5 ± 1.2</td>
</tr>
<tr>
<td>Central apnea index, apneas/h</td>
<td>29.8 ± 7.6</td>
<td>9.4 ± 1.2</td>
</tr>
<tr>
<td>Obstructive apnea index, apneas/h</td>
<td>14.6 ± 3.9</td>
<td>0.1 ± 0.1</td>
</tr>
<tr>
<td>Desaturation index, desaturations/h</td>
<td>37.8 ± 8.9</td>
<td>4.1 ± 0.9</td>
</tr>
<tr>
<td>REM</td>
<td>68.6 ± 8.9</td>
<td>22.7 ± 2.3</td>
</tr>
<tr>
<td>Central apnea index, apneas/h</td>
<td>25.0 ± 4.3</td>
<td>22.3 ± 2.2</td>
</tr>
<tr>
<td>Obstructive apnea index, apneas/h</td>
<td>43.6 ± 8.3</td>
<td>0.4 ± 0.1</td>
</tr>
<tr>
<td>Desaturation index, desaturations/h</td>
<td>63.4 ± 9.5</td>
<td>9.8 ± 1.4</td>
</tr>
</tbody>
</table>

*Data represent mean ± SEM for the diagnostic and CPAP polysomnographic studies of 18 infants.

†Significant difference from diagnostic study is denoted by t test (p < 0.05).

‡Significant difference from diagnostic study is denoted by rank-sum test (p < 0.05).

Eighteen infants have been treated successfully with nCPAP in their home for a duration of from 1 month to > 4 years (Table 1). In general, infants tolerated the CPAP and the nasal mask well. Problems were not reported by parents concerning the CPAP pressure or the mask itself. The infant nasal mask did not cause any facial skin breakdown or skeletal changes in any of the infants during treatment. Currently, 5 of these 18 infants continue nCPAP therapy because repeat diagnostic polysomnographic studies have shown persisting obstructive apnea during sleep. Each of these 5 infants have conditions with a primary diagnosis that involve upper airway anatomic abnormalities (Table 1). Repeat diagnostic and CPAP PD studies have been performed in each of these infants to review their OSA and the appropriate level of CPAP. We found that the amount of OSA recorded during the follow-up diagnostic studies remained similar to the amount recorded previously. During the CPAP studies performed using the previously determined pressures, obstructive events were being recorded and the CPAP was increased. Significantly higher CPAP has been required to prevent obstructive events during sleep as each infant grew older (Table 1). When nCPAP was first applied, an average pressure of 4.6 ± 0.2 cm H₂O prevented obstructive apnea; currently the average pressure has increased to 7.7 ± 0.7 cm H₂O in these 5 infants after 2 to 4.3 years of treatment (t test, p < 0.05).

nCPAP therapy was discontinued in 13 infants when follow-up polysomnographic studies showed that the previously recorded obstruction had resolved with the infant’s development. This occurred in 11 infants who were referred because of an apparent life-threatening event (ALTE) or a family history of SIDS, and in 1 infant who had choanal atresia. In another infant, who had Smith-Lemli-
Opitz syndrome, the obstructive apnea was believed to result from a vascular ring around the trachea. The surgical correction of the vascular ring was performed at 19 months of age, after 7 months of nCPAP treatment. A study performed 1 month after surgery demonstrated a complete resolution of her obstructive apnea, and nCPAP was discontinued.

Improvement of sleep-disordered breathing in these 13 infants was associated with a parental report of a decreased compliance to CPAP; each infant had begun to tolerate CPAP less. At the time CPAP was removed in the 13 infants, the average amount of central apnea was 8.2 ± 1.0 and 16.5 ± 1.7 apneas/h during NREM and REM sleep, respectively. In nine infants, obstructive events had been completely abolished, whereas occasional obstructive events were recorded in the other infants. The average amount of obstructive events was 0.2 ± 0.1 and 0.7 ± 0.3 apneas/h during NREM and REM sleep, respectively. The sleeping patterns of these infants had also improved since their first diagnostic study. The proportion of sleep spent in REM sleep when CPAP therapy was discontinued was 25.1 ± 0.4%.

Six infants were not treated with long-term nCPAP therapy. Because of parental noncompliance, three of these infants discontinued CPAP therapy while they were inpatients before undergoing a CPAP PD study. Although nCPAP was managing the sleep-disordered breathing in these three infants effectively, their parents refused to allow therapy within 1 week of initiating treatment. Subsequently, one of these infants, who had laryngomalacia, underwent surgery; whereas the other two infants, who were considered to have mild OSA, were treated with prone positioning rather than surgery. In the three other infants, a CPAP PD study was performed and CPAP effectively treated their sleep-disordered breathing, but we decided that the families of these infants would not be able to manage the treatment at home. These infants were also considered to have mild OSA and were monitored with a cardiorespiratory alarm system, and repeat polysomnographic studies were performed at 1-month to 2-month intervals to ensure that their sleep-disordered breathing did not worsen.

After treatment with nCPAP, the parents commented that there was an improvement in their infant’s daytime behavior. They described their infants as being more alert during wakefulness, and feeding was completed more easily without rests. The parents also reported that treatment with nCPAP required checking the infant during the night to ensure that the mask had not moved and that CPAP was being delivered correctly. However, the parents stated that the improvement in their infant’s sleep and daytime behavior was worth this necessary effort. In addition, in three infants who had been treated previously with nasopharyngeal intubation, their parents mentioned that nCPAP therapy was easier to manage and that they preferred nCPAP treatment to the nasopharyngeal tube. Additionally, nCPAP therapy was followed by an accelerated growth in three infants who had failure to thrive at the time OSA was diagnosed. Within 3 months of CPAP therapy, there were rapid increases in both their length and weight; however, owing to the small number of infants, no statistical analysis was performed.

**Discussion**

We successfully used nCPAP via mask to treat OSA in > 85% of infants, and it was possible to use CPAP as a long-term treatment in the infants’ homes. The previous use of nCPAP to treat OSA in infants has been limited. CPAP delivered via nasal prongs or an endotracheal tube has been used extensively to treat respiratory distress and apnea of prematurity in preterm infants. This application of CPAP was applied to support and inflate the lungs, rather than to support the upper airway. There have been a few reports describing CPAP therapy for OSA and upper airway resistance syndrome in infants; however, the long-term follow-up of OSA and CPAP requirements in infants has not been documented previously. Our results have demonstrated that nCPAP is an effective treatment for infantile OSA and that OSA and CPAP requirements change with growth and development.

Sleep-disordered breathing that involved both central and obstructive events during sleep was recorded in all of our infants during NREM and REM sleep. nCPAP treatment was associated with a reduction in the total amount of apnea during NREM and REM sleep in each infant, and this was mostly due to the prevention of nearly all of the obstructive events in each infant, regardless of their clinical history and possible mechanism of obstruction. It has been shown previously that nCPAP prevents complete and partial obstruction in infants who are considered to have an increased risk for SIDS. nCPAP treatment has also been reported to be effective in treating OSA in children with facial and upper airway anatomic abnormalities. nCPAP likely prevented obstructive events in our infants by providing a pneumonic splint to the upper airway that prevented its collapse.

The amount of central apnea during NREM sleep was reduced by nCPAP treatment in all of the infants of the present study. This is similar to previous findings in infants and adults. The reduction of
Central apnea by CPAP could indicate that upper airway reflexes are involved. It is possible that some of the central apneas in the infants of the present study were obstructive; if no effort is made against an obstruction, an apnea will appear to be central. It has been suggested that one mechanism of central apnea is the result of a reflex inhibition of respiration in response to upper airway stimulation, and that CPAP may prevent central apneas by stimulating pharyngeal mechanoreceptors.16

The sleep-disordered breathing in the infants of the present study was associated with and was likely responsible for sleep fragmentation and REM sleep deprivation. This was evident by the several episodes of NREM and REM sleep of short duration throughout the diagnostic sleep recordings. Polysomnographic studies performed throughout the 24-h period have shown that the amount of REM sleep recorded from normal infants is approximately one third of sleep at birth, and it decreases to one fourth of sleep by 6 months of age.17 Sleep fragmentation and sleep deprivation are known to have detrimental effects on arousability and sleep-disordered breathing.18,19 CPAP therapy was associated with an increase in the proportion of REM sleep to amounts recorded previously in normal infants.17 It is likely that preventing upper airway obstruction by CPAP reversed the associated sleep disturbances. These findings are consistent with our previous findings20 that any cause of sleep-disordered breathing may lead to sleep disturbances.

The sleep-disordered breathing in many of the infants of the present study improved with age. The majority of these infants had a history of an ALTE or a family history of SIDS. Central apnea decreased, and the obstructive events were completely resolved in most of the infants during the first year of life. Central sleep apnea is common in infants,21–23 it is believed to be related to the immaturity of respiratory control systems, and it is known to decrease in frequency with age.24,25 We have previously demonstrated20 that the amount of central and obstructive apnea in infants who are considered to have a high risk for SIDS progressively diminishes with time. CPAP may have influenced the resolution of obstructive events; however, it is likely that maturation of respiratory control systems, including the control of upper airway muscles, may have been involved in the decrease in central apnea and the elimination of obstructive apnea. CPAP likely protected these infants from apnea, obstruction, and its associated effects during this developmental period. CPAP was likely a more suitable form of treatment for OSA than alternative therapies such as surgery. In these infants, CPAP therapy was an effective noninvasive and temporary treatment while they had sleep-disordered breathing.

Some infants, however, have remained on CPAP without an improvement in their OSA. These infants all have upper airway anatomic abnormalities. In addition, increases in the level of pressure have been necessary to prevent OSA in all cases. We are not certain why an increase in age is associated with increases in CPAP pressure requirements; it may reflect increased growth and development. Long-term CPAP therapy has been effective in the management of OSA in these infants, and there have not been any detrimental effects to date. It is not known whether these children will remain on CPAP throughout their lives. It is likely that infants with anatomic abnormalities resulting in upper airway obstruction are likely to need CPAP as a long-term therapy. One infant with an identified anatomic obstruction had an improvement of OSA after surgery to correct the site of obstruction. An improvement of OSA in children has been demonstrated after adenotonsillectomy surgery.21 Treatment with CPAP possibly stabilized OSA in our patient until surgery could be performed to relieve the obstruction. CPAP can therefore be used as a bridging therapy for some infants until other interventions, such as surgery, can be performed.

CPAP therapy caused increased daytime alertness in each infant. Excessive daytime hypersomnolence is the most common daytime symptom associated with OSA in adults,27 and it is believed to be secondary to sleep fragmentation resulting from repetitive arousals during sleep. Daytime hypersomnolence and behavior problems have been reported in children with OSA.28 Although the level of daytime sleepiness in the infants could not be measured, it is possible that the sleep-disordered breathing and its associated sleep disturbances resulted in increased daytime sleepiness. The prevention of the sleep disturbances by CPAP certainly improved the quality of each infant’s sleep, and there was evidence of an altered daytime level of alertness with the loss of naps occurring during feeding.

The finding that CPAP therapy was associated with increased growth velocities in three infants who had failure to thrive was interesting. Failure to thrive has been associated previously with OSA in children.21,29 The surgical relief of upper airway obstruction in children has been followed by catch-up growth and the resumption of normal growth velocities.30 The same observation has been described in response to CPAP treatment in children.25

There were some difficulties encountered with the application of CPAP in infants. To provide successful CPAP therapy in infants, it is necessary to have motivated caregivers and a cooperative staff. Non-
compliance by the infant’s caregivers is likely to result in the discontinuation of CPAP therapy. The parents of infants who had used other forms of treatment volunteered that nCPAP was easier to manage than a nasopharyngeal airway. nCPAP therapy is not easy to perform in infants; however, the parents of our infants considered that the improvement in symptoms was worth the effort required to initiate the treatment. In cases, however, in which an infant does not tolerate CPAP and has severe OSA, an alternative form of therapy such as surgery may need to be considered.

nCPAP therapy in the present study has been shown to be effective in the management of OSA in infants, regardless of the believed site and mechanism of obstruction. nCPAP is effective as a short-term treatment for infants who have OSA for a transient period of time, and it is effective as a long-term treatment for infants with facial and upper airway anatomic abnormalities. nCPAP was a safe alternative to the placement of a nasopharyngeal airway or surgery, and it served as a temporary treatment for infants who would eventually require surgery. Our findings have stressed the need for regular follow-up, because the amount of OSA and the level of nCPAP needed to prevent obstructive apnea often change with age and development.

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