Predictors of Persistent Sleep Apnea After Surgery in Children Younger Than 3 Years

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Sleep-related breathing disorders are a common problem in children and involve a spectrum of abnormalities ranging from habitual snoring to obstructive sleep apnea (OSA). Habitual snoring, defined as occurring at least 3 nights per week and not associated with obstructive apneas, sleep fragmentation, or gas exchange abnormalities, has a prevalence of approximately 12%.1,2 Obstructive sleep apnea, the most severe entity in the spectrum of sleep-related breathing disorders, is defined by recurrent partial or complete obstruction of the upper airway during sleep accompanied by snoring, repeated arousals, oxygen desaturations, and hypercapnia. The prevalence of OSA in children ranges between 2% and 3%.3,4 Obstructive sleep apnea can lead to a variety of undesirable sequelae, which range from the physiologically severe, such as cardiovascular complications and failure to thrive,5 to the less severe but more psychologically important symptoms, such as excessive daytime sleepiness, behavioral disturbances, hyperactivity, attention problems, and poor school performance.6

The most common symptom of pediatric sleep-related breathing disorders is loud snoring, present regardless of sleep position and characterized by frequent obstructions with gasping and interruption in breathing. Obstructive sleep apnea can also cause increased nighttime urine production, which may lead to enuresis.7 The first step toward diagnosis of sleep apnea is parental observation of snoring and apneic or choking episodes. The current best study to diagnose OSA is an overnight sleep study, or polysomnogram (PSG), which includes

Importance
Obstructive sleep apnea (OSA) is a common disorder in children and can lead to important sequelae. Predictors of persistent OSA after adenotonsillectomy (T&A) in younger children are not well studied.

Objective
To evaluate residual OSA in a subgroup of children younger than 3 years after T&A and identify predictors of postoperative residual disease.

Design, Setting, and Participants
Retrospective review of medical records at a tertiary academic children’s hospital involving children younger than 3 years who had OSA documented by polysomnogram (PSG) and underwent T&A during the period October 1, 2002, through June 30, 2010. Some of these children had both preoperative and postoperative PSGs.

Main Outcomes and Measures
Effect of T&A on sleep study parameters and predictors of persistent disease after surgery.

Results
A total of 283 patients (mean [SD] age, 22 [7] months) underwent a preoperative PSG, with 70 of these patients having both a preoperative and postoperative PSG. In the group who had preoperative and postoperative PSGs, there were statistically significant improvements in mean (SD) apnea hypopnea index (AHI) (34.8 [40.7] to 5.7 [13.8]; P < .001), baseline oxygen saturation (96.6% [2.1%] to 97.2% [1.4%]; P = .05), minimum oxygen saturation (77.2% [11.4%] to 89.9% [6.8%]; P < .001), and sleep efficiency (84.7% [9.1%] to 88.7% [9.1%]; P = .02) after T&A. When AHI greater than 5 was used as the definition of OSA, 21% of the patients (15 of 70) had residual OSA. The most consistent predictor of residual OSA after T&A was the severity of preoperative OSA (P = .02).

Conclusions and Relevance
In a subgroup of children younger than 3 years with OSA, we found a high rate of residual OSA after T&A. Predictors of residual disease include severity of preoperative OSA as determined by PSG result. Postoperative PSGs might be indicated in these patients.

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monitoring of several variables, such as apneas, hypopneas, oxygen saturations, heart rate, respiratory rate, sleep stages and patterns (by electroencephalogram), and leg movements. The apnea hypopnea index (AHI) is defined as the number of apnea and hypopnea events per hour of sleep. Most sleep experts now consider an AHI from 1 to 5 to indicate mild OSA, AHI greater than 5 to 10 to indicate moderate OSA, and AHI greater than 10 to indicate severe OSA.

The treatment of choice for most children with OSA is adenotonsillectomy (T&A). Children younger than 3 years are considered to be at high risk for the development of respiratory complications after T&A. One reason for this increased risk is thought to be related to postoperative edema in the narrow pharynx of a child. Studies have also shown that recurrent hypoxemia can lead to increased sensitivity to opiates administered during anesthesia. In addition to the risk of postoperative complications in this age group, there is a concern that complete resolution of OSA does not occur in all children after surgery. There are few studies in the literature that measure the success of T&A in resolving OSA in this young population. Mitchell and Kelly in 2005 reported persistent OSA after surgery in this age group, in that they found that only 7 of 20 children (35%) had resolution of OSA as measured by postoperative AHI less than 5. They were among the first to recommend that a postoperative PSG be performed in children younger than 3 years to ensure complete resolution of OSA. In another study evaluating results of PSGs performed before and after T&A in children younger than 5 years, there was a significant improvement in the respiratory disturbance index (RDI) after surgery compared with the preoperative RDI. However, if one considers an RDI less than 5 to be “normal,” only 65% of the children were in that category after surgery. To our knowledge, no studies have examined predictors of persistent disease after T&A in this age group.

To further explore the effect of young age on the efficacy of T&A in children with OSA and try to determine predictors for residual disease, we retrospectively reviewed the medical records of children younger than 3 years with documented OSA who underwent T&A within our practice. We hypothesized that children younger than 3 years with OSA would improve after T&A and that the presence of persistent disease postoperatively would be predicted by the severity of preoperative OSA.

Methods

Study Design
We performed a retrospective review of the medical records of all children younger than 3 years who underwent T&A for PSG-documented OSA between October 1, 2002, and June 30, 2010, at the University of Chicago Comer Children's Hospital by 2 pediatric otolaryngologists. Children with cardiovascular abnormalities, neurological abnormalities, chromosomal abnormalities, concurrent tracheostomies, and craniofacial abnormalities were excluded. This retrospective review was approved by the institutional review board of the University of Chicago.

Adenotonsillectomy
All patients underwent T&A and were admitted to the hospital postoperatively for 24 hours. The tonsils were removed in standard fashion using electrocautery at a setting between 8 and 15 W, and the adenoids were removed with an adenoid curette. Residual bleeding was controlled using a suction electrocautery set between 12 and 15 W (tonsillar bed) and 30 and 35 W (adenoid bed).

Polysomnogram
A standard, overnight, multichannel, polysomnographic evaluation was performed in the sleep laboratory at the University of Chicago Medical Center. The following parameters were measured: chest and abdominal wall movements assessed by means of respiratory impedance or inductance plethysmography, heart rate assessed by means of electrocardiography, and air flow monitored by means of side stream end-tidal capnography, which also provided breath-by-breath assessments of end-tidal carbon dioxide levels and a thermistor. Arterial oxygen saturation was assessed by means of pulse oximetry, with simultaneous recording of the pulse waveform. Bilateral electro-oculograms, 8 channels of the electroencephalogram, chin and anterior tibial electromyograms, and analog output recording body position were also monitored. All measures were digitized with a commercially available polysomnographic system (Nihon Kohden America). Tracheal sounds were monitored with a microphone sensor, and a digital, time-synchronized video recording was obtained.

The proportion of time spent in each sleep stage was expressed as a percentage of the total sleep time (TST). The apnea index was defined as the number of episodes of apnea per hour of TST. Central, obstructive, and mixed apneic events were counted. Obstructive apnea was defined as the absence of airflow with continued chest wall and abdominal movements for the duration of at least 2 breaths. Hypopnea was defined as a decrease in nasal flow of at least 50% with a corresponding decrease in oxygen saturation of at least 4% and/or arousal. The AHI was defined as the number of episodes of apnea and hypopnea per hour of TST. Children with AHI values of at least 1 episode per hour of TST were considered to have OSA. The mean and nadir of oxygen saturation were recorded. Arousals were defined as recommended by the American Sleep Disorders Association Sleep Disorders Atlas Task Force report using the 3-second rule and/or the presence of movement arousal. Polysomnographic studies were scored by a registered polysomnographic technologist and reviewed by a board-certified sleep specialist.

Demographic Parameters
For each patient, we recorded the following demographic parameters: age at surgery, sex, race, height, weight, body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared), percentile BMI, and asthma diagnosis. Asthma was diagnosed on the basis of a review of the medical record. Children with a history of asthma and the requirement of asthma medications (continuous or as needed) mentioned in the record were defined as having asthma for the purposes of the study. No objective criteria were used. We used
persistent sleep apnea after surgery in children

Table 1. Demographic and Preoperative Polysomnogram Characteristics of the Whole Group

<table>
<thead>
<tr>
<th>Demographic Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at surgery, mean (SD), mo (N = 283)</td>
<td>22 (7)</td>
</tr>
<tr>
<td>Sex, No. (%) (N = 283)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>175 (61.8)</td>
</tr>
<tr>
<td>Female</td>
<td>108 (38.2)</td>
</tr>
<tr>
<td>Race, No. (%) (n = 282)</td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>239 (84.8)</td>
</tr>
<tr>
<td>White</td>
<td>43 (15.2)</td>
</tr>
<tr>
<td>Height, mean (SD), cm (n = 263)</td>
<td>81 (10)</td>
</tr>
<tr>
<td>Weight, mean (SD), kg (n = 280)</td>
<td>12 (3)</td>
</tr>
<tr>
<td>Asthma, No. (%) (N = 283)</td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>61 (21.6)</td>
</tr>
<tr>
<td>Absent</td>
<td>222 (78.4)</td>
</tr>
<tr>
<td>Percentile BMI,* No. (%) (n = 263)</td>
<td></td>
</tr>
<tr>
<td>Underweight</td>
<td>11 (4.2)</td>
</tr>
<tr>
<td>Healthy weight</td>
<td>87 (33.1)</td>
</tr>
<tr>
<td>Overweight</td>
<td>42 (16.0)</td>
</tr>
<tr>
<td>Obese</td>
<td>123 (46.7)</td>
</tr>
<tr>
<td>Preoperative PSG parameters (N = 283)</td>
<td></td>
</tr>
<tr>
<td>Total sleep time, mean (SD), minutes</td>
<td>362 (58)</td>
</tr>
<tr>
<td>Sleep efficiency, mean (SD), %</td>
<td>86 (10)</td>
</tr>
<tr>
<td>AHI, mean (SD)</td>
<td>25 (30)</td>
</tr>
<tr>
<td>SaO2, mean (SD), %</td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>96 (8)</td>
</tr>
<tr>
<td>Minimum</td>
<td>81 (12)</td>
</tr>
<tr>
<td>Severity of OSAa</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>41 (14.5)</td>
</tr>
<tr>
<td>Moderate</td>
<td>56 (19.8)</td>
</tr>
<tr>
<td>Severe</td>
<td>186 (65.7)</td>
</tr>
</tbody>
</table>

Abbreviations: AHI, apnea hypopnea index (defined as the number of apnea and hypopnea events per hour of sleep); BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); OSA, obstructive sleep apnea; PSG, polysomnogram; SaO2, arterial oxygen saturation.

* BMI percentiles were categorized as underweight, <5th percentile; healthy weight, 5th to <85th percentile; overweight, 85th to <95th percentile; obese, ≥95th percentile.

a Severity of OSA was categorized as mild (AHI, 1.0-5.0), moderate (AHI, 5.1-10.0), or severe (AHI, >10.0).

the World Health Organization anthropometric calculator to determine BMI percentiles for all the children in the study. We also used the Centers for Disease Control and Prevention definition for obesity: underweight, less than 5th percentile; healthy weight, 5th to less than 85th percentile; overweight, 85th to less than 95th percentile; and obese, ≥95th percentile or more.15 We also recorded the following PSG parameters: TST, sleep efficiency, AHI, baseline oxygen saturation, minimum oxygen saturation, and time between surgery and postoperative PSG. The AHI included obstructive apneas, central apneas, and hypopneas. Almost all individual sleep studies had 0 central apneas. When central apneas were present in some studies, they were few (<5) and were much fewer than the obstructive apneas or hypopneas, suggesting that obstruction was the predominant driver of the AHI. Resolution of sleep apnea was evaluated in a proportion of children who also under-
tions, and minimal oxygen saturations after T&A (Figure and Table 2). All but 3 of the children had a decrease in their postoperative AHI compared with the preoperative value. In these 3 children, the AHI did not worsen by more than 1 to 2 units. When the severity of postoperative AHI was distributed into categories, there was a significantly higher proportion of children with no or mild OSA in the postoperative PSG results and a significantly higher proportion of moderate and severe OSA in the preoperative PSG results of the same group of patients, supporting the marked improvement in OSA after T&A (Table 2).

We then divided these children into 2 groups based on postoperative PSG results. Fifteen of the 70 (21%) had residual OSA as defined by an AHI greater than 5, and 55 (79%) had complete resolution of OSA as defined by an AHI of 5 or less (Table 3). Despite the persistence of OSA in 21% of the children, their mean (SD) AHI improved from 55 (50) to 21 (25) ($P = .03$), and their mean (SD) minimum oxygen saturations increased from 74% (14%) to 82% (10%) ($P = .09$). If residual OSA was defined as any AHI greater than 1, then 41 of the children (59%) had residual OSA and 29 (41%) were considered cured (Table 4). There were no significant differences between the frequency of obesity in patients whose OSA resolved vs those who had residual OSA after T&A (Table 3 and Table 4).

To determine whether any of the preoperative parameters would predict persistence of OSA after T&A, we compared the age, height, weight, BMI, prevalence of asthma, preoperative AHI, and minimal oxygen saturations between the group who had complete resolution of OSA after surgery and the group who did not, as defined at the AHI = 5 level (Table 3). Importantly, preoperative AHI was significantly higher ($P = .02$) in the group with residual OSA compared with the group with complete resolution, suggesting that the severity of OSA before T&A was a predictor of persisting OSA afterward. There were no significant differences between the groups with regard to age, height, weight, BMI, asthma, or preoperative minimum oxygen saturation. The same applied if residual OSA was defined by a postoperative AHI greater than 1, with the exception that in addition to significant differences in preoperative AHI between the 2 groups, the group with residual OSA also had a significantly lower preoperative weight ($P = .04$) (Table 4).

### Discussion

Obstructive sleep apnea in children younger than 3 years is routinely treated with T&A. However, the efficacy of T&A has not been well studied in this patient population. Two small studies have shown that a high percentage of these children may have evidence of residual sleep apnea after T&A. Our data support the finding that, although T&A leads to a dramatic improvement in this age group, a high proportion of this population will have residual OSA. Although this proportion gives some insight into residual disease after T&A in this young population, the result is flawed because of the retrospective de-

![Figure. Effect of Adenotonsillectomy on the Apnea Hypopnea Index (AHI)](image_url)

Individual data are shown for 70 children who had preoperative and postoperative polysomnograms. There was a significant decrease in mean AHI (defined as the number of apnea and hypopnea events per hour of sleep) after surgery ($P < .001$). Horizontal bars represent mean values.

### Table 2. Comparison of Polysomnographic Criteria of the 70 Children for Whom Both Preoperative and Postoperative Sleep Studies Were Performed

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preoperative</th>
<th>Postoperative</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time, mean (SD), minutes</td>
<td>364.7 (57.5)</td>
<td>373.6 (53.6)</td>
<td>.20</td>
</tr>
<tr>
<td>Sleep efficiency, mean (SD), %</td>
<td>84.7 (14.9)</td>
<td>88.7 (9.1)</td>
<td>.02</td>
</tr>
<tr>
<td>AHI, mean (SD)</td>
<td>34.8 (40.7)</td>
<td>5.7 (13.8)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>$\text{Sa}_O_2$, mean (SD), %</td>
<td>96.6 (2.1)</td>
<td>97.2 (1.4)</td>
<td>.05</td>
</tr>
<tr>
<td>Baseline</td>
<td>77.2 (11.4)</td>
<td>89.9 (6.8)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Severity of OSA,* No. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>0</td>
<td>21 (30)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mild</td>
<td>3 (4)</td>
<td>34 (49)</td>
<td>.004</td>
</tr>
<tr>
<td>Moderate</td>
<td>15 (21)</td>
<td>4 (6)</td>
<td>.007</td>
</tr>
<tr>
<td>Severe</td>
<td>52 (74)</td>
<td>11 (16)</td>
<td>.002</td>
</tr>
</tbody>
</table>

Abbreviations: AHI, apnea hypopnea index (defined as the number of apnea and hypopnea events per hour of sleep); OSA, obstructive sleep apnea; $\text{Sa}_O_2$, arterial oxygen saturation.

*Severity of OSA was categorized as mild (AHI, 1.0-5.0), moderate (AHI, 5.1-10.0), or severe (AHI, >10.0).
sign of the study and the fact that only 25% of the children treated received postoperative PSGs. In some cases in which the PSG was repeated after T&A, there were residual clinical symptoms of sleep apnea (snoring, occasional apneic events), whereas in other cases, the physician treating the patient chose to perform a PSG after T&A because of the severity of preoperative disease. Thus, there were inherent flaws to selection of the patients who received a postoperative PSG. Indeed, the children who had a postoperative PSG were younger and shorter, weighed less, and had more severe OSA than the children who did not. This suggests that, had the whole group had preoperative and postoperative PSGs, the success rate of T&A would have been shown to be higher. Almost all children in our study showed a significant improvement in the severity of sleep apnea as demonstrated by an improvement in PSG parameters and the severity of OSA.

The rate of residual OSA depends on how that entity is defined. The magnitude of the AHI that actually indicates OSA is subject to controversy. In a study evaluating PSG results in children without sleep-related breathing disorders, the mean (SD) AHI in healthy children was 0.2 (0.6), yielding a statistically significant lower limit for AHI (at mean plus twice the SD) of 1.5, thus prompting some to consider any AHI greater than 1.5 to be indicative of OSA. Other authors suggest that AHI less than 5 is to be considered primary snoring, not OSA, although adverse neurobehavioral outcomes have been reported in children with an AHI less than 5. Most researchers now define children with AHI between 1 and 5 as having mild OSA, whereas children with AHI greater than 5 are defined as having definite OSA, which usually warrants medical or surgical treatment. If one considers the less conservative definition of AHI greater than 5 to represent OSA, then only 21%...
of the children who had a postoperative sleep study had residual disease after T&A. Conversely, if one uses the more conservative criterion of residual OSA as defined by AHI greater than 1, then 59% of the children (41 of 70) had residual disease. Of note is that among those 41 children with residual OSA, 26 (63%) had mild disease as defined by AHI between 1 and 5.

Several studies have examined the efficacy of T&A in the treatment of OSA in children. Persistence of abnormal PSG findings after T&A in children has been reported for between 20% and 40% of the cases. In these studies, various tools were used to evaluate persistence of OSA, the populations were heterogeneous (including obese children), and the mean age of the children was 6 to 9 years. Tauman and colleagues performed PSGs before and after T&A in 110 children with a mean age of 6.4 years. Similar to our study, there was a significant reduction in the AHI and improvement in oxygen saturation nadir postoperatively but 46% of the children still had an AHI between 1 and 5 and 29% of the children had AHI greater than 5, suggesting residual OSA. In their assessment of predictors for persistent OSA (AHI > 1), older children with higher BMI were less likely to have a cure after surgery. In a more recent study, Bhattacharjee and colleagues performed a prospective, multicenter study evaluating the effect of T&A on OSA as documented by preoperative and postoperative PSG results. They studied 578 children with a mean age of 6.9 years and showed a significant reduction in AHI and improvement in nadir oxygen saturations in the group as a whole. When AHI cutoffs were used to determine residual OSA, 21.6% of the children had residual OSA as defined by AHI at least 5 and 72.8% had residual OSA as defined by AHI greater than 1. Significant predictors of persistent OSA after T&A in these children were older age (>7 years), higher BMI, presence of asthma, and severity of OSA as gauged by preoperative AHI.

Only 2 studies that we are aware of have evaluated the persistence of OSA in younger children. Walker and colleagues evaluated 34 children younger than 5 years (mean age, 3 years) (including some with comorbidities such as Down syndrome) with PSGs before and after T&A. They showed that 35% of the patients had residual OSA as defined by an RDI of at least 5. No predictors of residual disease were examined. Mitchell and Kelly evaluated 20 children younger than 3 years (mean age, 2.2 years) with preoperative and postoperative PSGs. They also had patients with substantial comorbidities such as Down syndrome, cerebral palsy, and craniofacial abnormalities. There was a significant overall improvement in RDI after surgery (from 34.1 to 12.2; P < .001), but 65% of the subjects had residual OSA as defined by RDI at least 5. Again, predictors of residual OSA were not examined. The rate of residual OSA defined by AHI greater than 5 is smallest in our series (21%) compared with the above series, which is probably related to the fact that we excluded patients with comorbidities whereas the other 2 reports did not.

The appropriate AHI cutoff used to determine residual OSA is controversial. Some authors consider any AHI greater than 1 to be abnormal, whereas others suggest that AHI greater than 5 denotes an abnormal sleep study result. Studies suggest that 1 of the sequelae of OSA is chronic systemic inflammation as revealed by elevated serum levels of C-reactive protein (CRP). In 1 study, levels of serum CRP were found to be significantly higher in children with AHI at least 5 compared with children with AHI between 1 and 5 and those with no OSA as defined by AHI less than 1. In another study evaluating serum levels of high-sensitivity CRP and correlating that with neurocognitive dysfunction and severity of OSA, high-sensitivity CRP levels were higher in children with OSA, particularly in those who developed neurocognitive deficits. Moreover, Kaemingk and colleagues showed that children with AHI greater than 5 had more difficulties with learning functions. These and other studies suggest that the magnitude of the inflammatory responses elicited by OSA is a major determinant of increased risk for neurocognitive dysfunction and that these sequelae of OSA might be highest at an AHI greater than 5. More studies are however necessary to help clinicians determine useful AHI parameters in PSGs, especially in the context of residual OSA after T&A in younger children.

It remains a challenge for the practicing otolaryngologist to predict which children will have evidence of residual disease postoperatively, which is important in informing parents what to expect after T&A in younger children. Aiming to find preoperative parameters that would identify children at higher risk for residual disease, we analyzed both groups based on age, BMI, and preoperative severity of disease (including AHI and minimal oxygen saturations). We have found that the severity of OSA according to preoperative PSG result, as well as the patient’s weight (lower weight), provide good predictors of residual disease in this patient population. This, along with the reported proportions of children with residual OSA after T&A, should allow clinicians to give parents some ideas of what to expect from surgery in resolving OSA in their younger children.

Although treatment options are limited, there are some possible therapies for children with persistent OSA after T&A. Intranasal steroid therapy has shown promise in helping to decrease OSA. In a limited, open-label trial, use of a combination of budesonide and montelukast sodium was shown to reduce residual OSA after T&A in a small number of children. There are also emerging data on other possible surgical procedures such as palatoplasty, supraglottoplasty, and lingual tonsillectomy in these children. Some of our patients with residual OSA had no additional follow-up, some were given continuous positive airway pressure treatment and either an intranasal steroid or a combination of intranasal steroid and montelukast, and a few were given supplemental oxygen therapy. All these interventions were performed in consultation with our colleagues from pediatric pulmonary or sleep medicine.

In conclusion, our data suggest that in the treatment of very young patients with OSA, T&A provides substantial improvement of disease severity but a certain subset of these children may have residual disease that may necessitate further identification and treatment. The threshold for repeating the PSG should be low when these young children are evaluated postoperatively, especially the ones who are smaller and have more severe OSA. Future research should focus on prospective evaluation of a large cohort of younger children with OSA for whom the extent of the disease is documented preoperative and postoperatively. This would eliminate the selection bias that we faced in our study and provide better information on predictors of persistent disease after T&A.
Research Original Investigation

ARTICLE INFORMATION
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Author Contributions: Dr Baroody had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.
Study concept and design: Nath, Emani, Baroody.
Acquisition of data: All authors.
Analysis and interpretation of data: Nath, Emani, Baroody.
Drafting of the manuscript: Nath, Emani, Suskind.
Critical revision of the manuscript for important intellectual content: Baroody.
Statistical analysis: Nath, Emani, Baroody.
Administrative, technical, or material support: Suskind.
Study supervision: Baroody.

Conflict of Interest Disclosures: None reported.

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REFERENCES