Pharyngeal critical pressure in children with mild sleep-disordered breathing


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Fregosi, R. F., S. F. Quan, W. L. Morgan, J. L. Goodwin, R. Cabrera, I. Shareif, K. W. Fridel, and R. R. Bootzin. Pharyngeal critical pressure in children with mild sleep-disordered breathing. J Appl Physiol 101: 734–739, 2006. First published May 18, 2006; doi:10.1152/japplphysiol.01444.2005.—There is evidence that narrowing or collapse of the pharynx can contribute to obstructive sleep-disordered breathing (SDB) in adults and children. However, studies in children have focused on those with relatively severe SDB who generally were recruited from sleep clinics. It is unclear whether children with mild SDB who primarily have hypopneas, and not frank apnea, also have more collapsible airways. We estimated airway collapsibility in 10 control subjects (9.4 ± 0.5 yr old; 1.9 ± 0.2 hypopneas/h) and 7 children with mild SDB (10.6 ± 0.5 yr old; 11.5 ± 0.1 hypopneas/h) during stable, non-rapid eye movement sleep. None of the subjects had clinically significant enlargement of the tonsils or adenoids, nor had any undergone previous tonsillectomy or adenoidectomy. Airway collapsibility was measured by brief (2-breath duration) and sudden reductions in pharyngeal pressure by connecting the breathing mask to a negative pressure source. Negative pressure applications ranging from −1 to −20 cmH₂O were randomly applied in each subject while respiratory airflow and mask pressure were measured. Flow-pressure curves were constructed for each subject, and the x-intercept gave the pressure at zero flow, the so-called critical pressure of the upper airway (Pcrit). Pcrit was significantly higher in children with SDB than in controls (−10.8 ± 2.8 vs. −15.7 ± 1.2 cmH₂O; P < 0.05). There were no significant differences in the slopes of the pressure-flow relations or in baseline airflow resistance. These data support the concept that intrinsic pharyngeal collapsibility contributes to mild SDB in children.

METHODS

Subjects. All methods used to recruit subjects and to collect the present data set were approved both by the University of Arizona Human Subjects Committee and the Tucson Unified School District Research Committee. In all cases, we obtained written informed consent from the parents and assent from the children. The details of the subject recruitment have been described in detail previously (7). Our pool of 17 subjects (see Table 1) was drawn from a cohort that previously completed unattended overnight polysomnography (10). From these studies, we computed the apnea-hypopnea index (AHI), which includes central apneas, obstructive apneas, and hypopneas (10). We used this data bank focusing on those subjects who previously had undergone physiological studies as part of the Tucson Children’s Assessment of Sleep Apnea (TuCASA) study to recruit a convenience sample of 10 subjects (3 male subjects, 7 female subjects) with an AHI <4.0 (low-AHI group), and 7 (4 male subjects, 3 female subjects) with an AHI >5.0 (high-AHI group), the goal being to compare Pcrit in the low- and high-AHI groups. Importantly, the subjects in this study had essentially no obstructive apneic events (defined as the absence of airflow for ≥6 s or at least 2 breath cycles) during sleep. However, two subjects in the low-AHI group (0.23 and 0.13 obstructive apneas/h) and one subject in the high-AHI group (0.25 obstructive apneas/h) did have very infrequent obstructive apneas. Thus the AHI in these subjects was almost entirely the result of hypopneas and central apneic events (Table 2). Hypopneas were defined as a ≥30% reduction in the amplitude of the respiratory signal from the “baseline” condition (identified during a period of regular breathing with stable oxygen levels), if this reduction lasted for ≥6 s and for ≥2 breath cycles. Central apneas were not an exclusion criteria because they are sometimes observed in children. A central apnea was defined as an absence of displacement on both chest and the abdominal

ADULTS WITH OBSTRUCTIVE SLEEP disordered breathing often have floppy pharyngeal airways (“anatomic hypothesis”) (15, 16, 33) and/or poor neuromuscular control of pharyngeal airway muscles (“neural hypothesis”) (2, 12, 14, 21, 30, 37). Consistent with findings in adults, the pharynx of children with severe obstructive sleep apnea has been shown to be more collapsible than the pharynx of age-matched children with primary snoring but no evidence of obstructive apnea (24, 25). However, the children from these studies were recruited from sleep disorders clinics and may not be representative of those with milder disease. Furthermore, although many children are diagnosed with sleep-disordered breathing, the majority of breathing events recorded during polysomnography are hypopneas (typically defined as a 30–50% reduction in tidal volume, lasting for at least 2 complete breath cycles). Thus an important but unanswered question is whether children with mild sleep-disordered breathing (i.e., those with little or no evidence of frank obstructive apnea) have more collapsible airways than children without sleep-disordered breathing.

The answer to this question is important because demonstration of a more collapsible airway related to either pharyngeal narrowing and/or increased pharyngeal compliance would signify that the pathophysiology of sleep-disordered breathing in children with mild sleep-disordered breathing is similar to those with severe disease. Furthermore, a pathophysiological linkage between mild and severe disease would suggest that similar therapeutic approaches could be employed and perhaps that more aggressive evaluation and treatment might be indicated to prevent progression. Here we test the hypothesis that the pharynx of children with mild sleep-disordered breathing manifesting primarily as hypopnea and little arterial oxygen desaturation is more collapsible than the pharynx of age- and body mass index-matched children without significant sleep disordered breathing.

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inductance channels that lasted for ≥6 s and for ≥2 breath cycles. Nevertheless, the number of central apneas in the two populations was low compared with the number of hypopneas (Table 2).

Of the subjects selected for study, four in the low-AHI group snored, and three in the high-AHI group snored. Excessive daytime sleepiness was reported in two subjects from each group. One subject in the low-AHI group and two in the high-AHI group reported insomnia. The parents of one subject (in the high-AHI group) reported a witnessed apnea, whereas one parent of a subject in the low-AHI group reported episodes of enuresis. All parents/guardians of TuCASA subjects received notification of the results of their child’s polysomnogram, and for those subjects with a high AHI, it was advised that the results be discussed with the child’s primary care physician. Nevertheless, none of the parents/guardians reported that their child had undergone a tonsillectomy or adenoidectomy. The elapsed time between the initial sleep study and the determination of critical pressure of the upper airway (Perit) for the present study ranged from 7 to 14 mo in the low-AHI group and from 10 to 24 mo in the high-AHI group. To ensure that elapsed time did not have a systematic effect on measured Perit, we ran a correlation analysis between elapsed time and measured Perit; the correlation was not significant ($r = 0.364$, $P = 0.20$). We also ran an analysis of covariance with group (low- or high-AHI groups) as the fixed factor and Perit as a covariate. This analysis also showed that elapsed time is not a significant factor in determining Perit.

Instrumentation. Subjects reported to the sleep research laboratory in the Department of Psychology in the early evening for a nap lasting 2–4 h. Parents were asked to allow their children to refrain from napping and to try and keep their children awake beyond their normal bedtime on the night before the study. For this reason, the majority of studies were done either on Saturday evening, or in the summer, so as not to interfere with school. Height, weight, neck circumference, and diastolic blood pressure. *Different from low AHI group, $P < 0.05$.

To measure $P_{crit}$ during sleep, the subjects were fitted with a nasal mask [Hans Rudolph nasal continuous positive airway pressure (CPAP) series 7800, with head cap and virus filter]. We applied dental impression material (Exaflex) around the edges of the mask to ensure a tight seal. The mouth was prevented from opening by lightly sealing the lips with dermatologically approved tape and by wrapping an Ace bandage around the head (from the chin to the crown of the head).

A pneumotachometer (model 4700, Hans Rudolph) was attached to the nasal mask and connected to a differential pressure transducer (model MP-45, Validyne; range ± 2 cmH2O) to measure airflow, as described previously (7, 9). The distal end of the pneumotachometer was attached to a Y valve (model 8250, Hans Rudolph) with one opening to room air and another to a CPAP device (Respirronics) that was modified to generate negative instead of positive pressure. The Y valve was held in place by attaching it to a Velcro strip located on the front of a custom-made vest that the subjects wore. Mask pressure (Pmask) was measured by connecting Tygon tubing from a mask port to a second differential pressure transducer (Validyne MP-45; range ± 56 cmH2O). System dead space from mask opening to the Y-valve bifurcation was 40 ml.

Two solenoid-operated balloon valves (Hans Rudolph) located inside the Y valve allowed us to periodically apply negative pressure to the airway. With the balloon to the negative pressure source inflated and the balloon to room air deflated, the subject breathed room air at normal pressure (Pmask = 0 cmH2O). With the balloon to room air inflated and the balloon to the negative pressure source deflated, various levels of subatmospheric pressure were generated in the upper airway, as indicated by a decrease in Pmask. We also monitored the pressure produced by the modified CPAP machine [pump pressure (Ppump)] with a third differential transducer (model MP-45, Validyne; range ± 56 cmH2O) that was connected to the tubing that connected the Y valve with the pump.

All instrumentation was located outside the sleeping chamber, and the subject was monitored via closed-circuit television. Tubing and wires were passed from the chamber to the outside through a small portal. Chin EMG, EEG, EOG, Pmask, Ppump, airflow, and $S_{ao2}$ were continuously monitored on a computer screen, using a digital 16-channel integrated hardware and software system (Grass-Telefactor, TWinTM Version 3.3, Astro-Med, West Warwick, RI). Flow, Pmask, and Ppump were also monitored in parallel on a chart recorder (Gould). The investigators operated the balloon valves from outside the sleeping chamber using a remote switch, and they manually regulated the level of Ppump by adjusting a control on the modified CPAP machine.

Experimental protocol. Subjects were placed on the bed in the supine position. A “U”-shaped pillow was placed under the head, and

### Table 1. Anthropometric variables in high- and low-AHI groups

<table>
<thead>
<tr>
<th>Group</th>
<th>BMI, kg/m²</th>
<th>Age, yr</th>
<th>Neck Circumference, cm</th>
<th>SBP, mmHg</th>
<th>DBP, mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low AHI (n = 9)</td>
<td>18.2±1.3 (9.5–26.1)</td>
<td>9.4±0.5 (6–12)</td>
<td>28.6±0.8 (25.5–33.6)</td>
<td>104±3.1 (90–116)</td>
<td>58.6±4.3 (35–78)</td>
</tr>
<tr>
<td>High AHI (n = 5)</td>
<td>19.9±1.1 (15.2–24)</td>
<td>10.6±0.5 (8–12)</td>
<td>30.2±0.7 (27.4–32.5)</td>
<td>111±4.7 (95–128)</td>
<td>67.1±5.5* (50–88)</td>
</tr>
</tbody>
</table>

Values are means ± SE with range in parentheses; $n$, no. of subjects. AHI, apnea-hypopnea index; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure. *Different from low AHI group, $P < 0.05$.

### Table 2. Polysomnography data for low- and high-AHI groups

<table>
<thead>
<tr>
<th>Group</th>
<th>AHI, events/h</th>
<th>Hyp. events/h</th>
<th>CAI, events/h</th>
<th>OAI, events/h</th>
<th>$S_{ao2}$, Nadir, %</th>
<th>TST, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low AHI (n = 9)</td>
<td>3.3±0.1 (2.3–3.7)</td>
<td>1.9±0.2 (1.2–2.9)</td>
<td>1.4±0.1 (0.75–2.2)</td>
<td>0.036±0.02 (0–0.23)</td>
<td>93.7±0.63 (90–96)</td>
<td>50.7±6.7 (33.5–95)</td>
</tr>
<tr>
<td>High AHI (n = 5)</td>
<td>15.0±1.5 (10.5–22)</td>
<td>11.5±1.5 (6.2–18.7)</td>
<td>3.5±0.035 (0.9–9.1)</td>
<td>0.035±0.035 (0–0.25)</td>
<td>90.7±1.0* (87–94)</td>
<td>66.7±12.6 (35.5–119.5)</td>
</tr>
</tbody>
</table>

Values are means ± SE with range in parentheses; $n$, no. of subjects. AHI, sum of hypopnic, central, and obstructive events/hour; Hyp, hypopnea index (hypopnic events/hour); CAI, central apnea index (central events/hour); OAI, obstructive apnea index (obstructive events/hour); $S_{ao2}$ nadir, lowest arterial oxygen saturation observed; TST, total sleep time. See METHODS for more detailed definitions. *Different from low AHI group, $P < 0.05$. †Different from low AHI group, $P < 0.001$.
two pillows were placed on either side of the subject’s upper body to prevent rolling. Airway pressure application began when the subject entered a period of stable non-rapid eye movement (NREM) sleep (stage 2, 3, or 4), as judged by a registered polysomnographic technologist who monitored sleep and wakefulness for the entire study. Pmask was dropped repeatedly, intermittently, and randomly to levels ranging from −1 to −17 cmH2O (corresponding pump pressures from −1 to −20 cmH2O). Intermittent negative pressure was applied and terminated at the end of expiration, and it was maintained for two breaths. Intermittent, rather than sustained, pressure applications were utilized because we wished to evaluate the response in the absence of reflex changes in upper airway motor tone, which can occur if negative pressure applications are maintained beyond two breath cycles (1, 25). The subject was returned to atmospheric pressure after each incremental drop in pressure. The time between each drop ranged from our minimum value of three breath cycles to a minute or more. The actual mask pressure nadir that we measured for each drop ranged from our minimum value of three breath cycles to a minute or more. The actual mask pressure nadir that we measured for all trials without arousal ranged from −3.1 to −15.9 cmH2O. We attempted 20–30 random, subatmospheric pressure trials in each subject, but we did not use any trial associated with an arousal. We ended up with between 20 and 27 valid trials in the 10 subjects in the low-AHI group and between 17 and 27 trials in the high-AHI group subjects. Successive negative pressure applications were applied only when the breathing pattern returned to baseline levels (Fig. 1). Once data collection was finished, subjects were woken, disconnected from all equipment, debriefed, and released.

Data analysis. We measured the peak Pmask (expressed in cmH2O) and corresponding peak flow (expressed in ml/s) values that occurred during each of the pressure applications. These measurements were made manually from the chart recordings of Pmask and flow (see Fig. 1). Average midinspiratory flow was measured as the average of the two breaths recorded at each pressure level (Fig. 1), and the corresponding Pmask values were measured in the same way. Pressure transducers were calibrated before each experiment with a water manometer, and flow was calibrated with a precision rotameter (Matheson). To obtain an estimate of Pcrit, linear regression of all valid pressure-flow trials for each subject was then performed, as shown in Fig. 2. Perit was estimated as the x-intercept, which corresponds to the estimated pressure at a flow of zero (23, 25, 32). As reported by others (23, 25), it is not possible to obtain a valid Pcrit estimate in some children because their airways are very stiff. As a result, reasonable extrapolation to the x-intercept is impossible because flow does not decline significantly even at very negative applied pressures (23, 25). Five of our subjects had such flat pressure-flow curves. To avoid using extreme extrapolation to obtain a Pcrit value in these subjects, we instead arbitrarily applied a floor pressure equivalent to the most negative pump pressure that we applied (−20 cmH2O). This approach has been used by others in the study of the pediatric airway (23, 25), and it allows one to analyze the data statistically while avoiding biasing the data toward subjects with extremely negative Pcrit values.

We measured baseline airway resistance during quiet, unperturbed breathing, by taking the ratio of Pmask at a standard flow of 200 ml/s. Upstream resistance measured during the application of negative pressure was estimated from the inverse of the flow-pressure slope for each subject (see Fig. 2). Both baseline and upstream resistance values are expressed in terms of cmH2O pressure per liter per second of flow.

Statistical analysis. We divided all measured variables into three categories: pressure-flow data (baseline resistance, upstream resistance, and Pcrit); anthropometric data (body mass index, age, neck circumference, systolic blood pressure and diastolic blood pressure); and sleep data (AHI, central apnea index, hypopnea index, nadir SaO2, and total sleep time). All variables in each group were analyzed by one-way ANOVA. If the ANOVA was significant, we used the Student-Newman-Keuls multiple comparison test to examine differences between low- and high-AHI groups for each variable. Results of ANOVA revealed that the pressure-flow slope data were not normally distributed, owing to the very flat slopes in some subjects (see above). Accordingly, the pressure-flow slopes in the low- and high-AHI groups were compared with the nonparametric Mann-Whitney U-test. Finally, visual estimates of airway size were compared with Fisher’s exact test. All statistical analyses were performed with InStat software (GraphPad Prism, San Diego, CA). In all cases, a value of P < 0.05 served as the threshold for statistical significance.
RESULTS

There were no significant differences in age, body mass index, neck circumference, or systolic blood pressure, although diastolic blood pressure was higher in the high-AHI group (P < 0.05, Table 1). As shown in Table 2, the AHI and the hypopnea index in the two groups were significantly different, as designed, but the central apnea index was the same in both groups. The estimated airway size was also the same in both groups (P = 0.606, Fisher’s exact test). As might be expected, the nadir SaO2 was lower in the high-AHI group (Table 2). The amount of stable, NREM sleep required to complete the measurements of Pcrit averaged 50.7 ± 6.7 and 66.7 ± 12.6 min in the low- and high-AHI groups, respectively (P = not significant; Table 2).

Figure 1 is a chart recording from one subject, showing two trials where Pmask was rapidly reduced by switching the inspired line to the negative pressure source (valve switching occurred at arrows). Note the flow pattern during pressure application, demonstrating an increase in flow followed by a characteristic plateau (5, 26, 35, 36), indicative of flow limitation.

Figure 2 shows pressure-flow data from experiments in two subjects, one from the low- (A) and one from the high- (B) AHI groups. In each curve, the solid line is the result of linear regression analysis, and the dashed line is the extrapolation of the regression line to the x-intercept, defining Pcrit. As shown in Fig. 3A, average Pcrit was significantly more negative in the low- (−15.7 ± 1.2 cmH2O) compared with the high- (−10.8 ± 2.8 cmH2O) AHI group (Bonferroni-corrected t value = 2.388, P < 0.05). Baseline airway resistance measured during quiet, unperturbed breathing ranged from 0.96 to 10.5 cmH2O·l−1·s in the low-AHI group and from 1.7 to 7.4 cmH2O·l−1·s in the high-AHI group, with no between-group differences (see average data in Fig. 3B). Upstream resistance measured from the negative pressure-flow slopes ranged from 9.8 to 36.7 cmH2O·l−1·s in the low-AHI group and from −1 to +65 cmH2O·l−1·s in the high-AHI group, with no significant between group differences (Mann-Whitney U = 19, P = 0.264; see Fig. 3C).

DISCUSSION

Our major finding is that children in the high-AHI group have more collapsible airways than age-matched control subjects, at least in NREM sleep. As described in METHODS, subjects in the high-AHI group had mild sleep-disordered breathing manifesting primarily as hypopneas (also see below). These observations indicate that, similar to children with severe sleep-disordered breathing, even those with mild sleep disease have relatively collapsible airways. Thus similar pathophysiological mechanisms may be operative in children over a broad range of sleep-disordered breathing severity.

Critique of methods. The focus of our experiments was on children with mild sleep-disordered breathing manifested primarily as hypopneas, with no evidence of complete airway obstruction and only mild arterial oxygen desaturation. Although we defined hypopnea as a ≥30% reduction in tidal volume, which is a broader definition of hypopnea than often used (4, 22, 25), others also have used a comparable reduction in airflow (13, 31). Furthermore, our laboratory has previously demonstrated that its use as a component of an overall AHI in children correlates significantly with various physiological and behavioral outcomes (6, 10, 11, 18, 27). Although the hypopneas that we measured could be either central or obstructive events, as one cannot differentiate between the two without the simultaneous measurement of esophageal pressure, we could not justify measuring esophageal pressure in our young subjects. More importantly, our goal was to evaluate the relation between mild sleep-disordered breathing and pharyngeal collapsibility.

Prior studies that measured the pressure-flow relation in children used a positive airway pressure of +2 cmH2O as the baseline condition, or “holding pressure” (23, 25). Our subjects, none of whom were being treated, had no experience with continuous positive airway pressure, nor did they have evidence of flow limitation when breathing at atmospheric pressure. When we attempted to apply a positive holding pressure of +2 cmH2O in an effort to be consistent with earlier work, the subjects complained of discomfort. As a result, we chose to use atmospheric pressure as the baseline condition in our experiments. In adults, the difference in airway tone between atmospheric pressure and +2-cmH2O holding pressure is often demonstrable, but we are unaware of any evidence that this is so in children either with or without sleep-disordered breathing. In other words, to our knowledge there is no evidence that either upper airway muscle EMG activity or pressure flow slopes differ significantly in the same children,
when studied at +2- and at 0-cmH2O holding pressures. Nevertheless, comparison of our results with earlier data should be interpreted with this experimental difference in mind.

We used the intermittent Pcrit method to estimate pharyngeal collapsibility. It is important to re-emphasize that our goal was to estimate the mechanical properties of the pharynx in the absence of changes in neuromotor tone (19, 20, 23, 28). The intermittent technique yields more negative Pcrit values than the sustained method, wherein negative airway pressure is maintained for several breaths (25). This is consistent with a time-dependent increase in tonic upper airway muscle activity with protracted applications of negative pharyngeal pressure (23). Thus our use of the intermittent Pcrit method allows the measurement of the pressure-flow relationship in the absence of changes in neuromotor tone (23, 25).

The pharynx in children with sleep hypopnea. In adults with obstructive sleep-disordered breathing, a narrow pharynx (due largely to fat deposits in the pharyngeal walls, tongue, and uvula) and thick neck are often associated with flow limitation at much higher intrapharyngeal pressures compared with control subjects. Previous studies in children with pharyngeal narrowing due to enlarged tonsils and adenoids also show greater pharyngeal collapsibility, with modest improvement after tonsillectomy and adenoïdectomy (25). Our laboratory’s recent work in children with mild sleep-disordered breathing showed that their retropalatal airway was relatively narrow, due to a combination of modest enlargements in the size of their soft palate, adenoids, and pharyngeal tonsils (8). This was true even though none of the children were judged to be candidates for tonsillectomy and/or adenoïdectomy. Moreover, in this study we did not find an association between neck circumference and Pcrit (r = 0.13, P = 0.65). Thus, as opposed to adult subjects, the pharynx is more collapsible in children with mild sleep-disordered breathing, even though they do not have clinically significant pharyngeal narrowing, or abnormally large necks. These observations suggest that the intrinsic properties of the pharyngeal airway are abnormal in these children.

The average values and variability of the Pcrit measurements in our subjects compare very well with those recently reported by Marcus et al. (23, 25), who found average Pcrit values of −25 cmH2O in controls and −5 cmH2O in children with obstructive sleep apnea (see Fig. 3). Nevertheless, our values in both control subjects and children with mild sleep-disordered breathing are considerably more negative than those reported in paralyzed children (16). This is likely due to the failure of NREM sleep to completely abolish the tone of pharyngeal muscles (19, 20, 28), unlike paralysis (see above). Nevertheless, this and all previous studies have consistently shown differences in Pcrit between healthy children and those with sleep-disordered breathing, despite differences in techniques and disease severity.

Pcrit is influenced by the stiffness of the pharyngeal walls and muscles, and the upstream resistance to airflow. In the present study, Pcrit was more positive in the children with sleep hypopnea, although upstream resistance was the same in both high- and low-AHI groups. Marcus et al. (23, 25) also found a more positive Pcrit in children with severe obstructive sleep apnea, with no significant difference in upstream resistance (they report upstream conductance, the reciprocal of resistance, in their Table 3) with the intermittent Pcrit technique, as used herein. Interestingly, when they used sustained negative pressure applications, resistance was lower in the children with obstructive apnea than in the control children (25). Observations such as these underscore the difficulty in interpreting estimates of airway collapsing pressure in human subjects. Nevertheless, a change in Pcrit without a corresponding change in resistance suggests that intrinsic collapsibility of the pharyngeal wall may be an important factor in determining pharyngeal caliber, along with soft tissue-induced airway narrowing in children with sleep-disordered breathing (8). This is consistent with the observation that tonsillectomy and adenoïdectomy does not always improve sleep-disordered breathing (25) and why some children have a recurrence of disease some years after surgery (22).

In summary, our data show that children with mild sleep hypopnea have more collapsible upper airways than age- and body mass index-matched control subjects during NREM sleep. Although the collapsing pressures observed in these children are large compared with values reported in adults with obstructive sleep-disordered breathing (34), our observations suggest that children with mild sleep-disordered breathing and relatively positive Pcrit values should be thoroughly evaluated, as they may be predisposed to more severe pharyngeal collapse later in life.

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