Observation following Tonsillectomy May Be Inadequate Due to Silent Death
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Observation following Tonsillectomy May Be Inadequate Due to Silent Death

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Abstract
The focus on the first 24 hours of care for respiratory events following tonsillectomy may be misplaced and a broader focus is warranted. Nocturnal hypoxemia, an elevated apnea-hypopnea index, or obstructive sleep apnea contributes to an increased sensitivity to narcotics and postoperative complications. Narcotic pain management depresses respiration through an increase in the frequency of central sleep apnea, decreased minute ventilation, increased hypercarbia pressure, and a decrease in the hypoxic ventilator response. Residual pain gives some margin of safety as it stimulates respiration. Children dying following tonsillectomy do so silently during sleep, often without arousing the attention of caregivers or nursing personnel in close proximity. Perioperative education of caregivers, use of the least morbid surgical technique, and the control of pain rather than its elimination are prudent steps in the management of tonsillectomy patients.

Keywords
central apnea, death, hypoxemia, narcotics, postoperative pain, pulse oximetry, respiratory drive, sleep apnea, sleep disordered breathing, tonsillectomy

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Introduction
Traditionally, the focus on reducing postoperative morbidity or mortality has been whether the patient should be admitted to the hospital and monitored overnight. Most studies that have identified the onset of respiratory compromise following tonsillectomy have found it to occur less than 15 hours after surgery. For most of the past 3 decades, posttonsillectomy patients have been sent home after less than 24 hours with most of the monitoring relegated to parental observation. Recent reports suggest that our focus may be misplaced and that respiratory complications may occur up to 3 days postoperatively. This article seeks to review available information about postoperative care of pediatric tonsillectomy patients. This study was exempt from institutional review board review.

Host Factors

Intrinsic Pathophysiology
Children undergoing tonsillectomy often have underlying ventilation disturbances. About one-third of the unselected tonsillectomy population preoperatively demonstrate either baseline hypoxemia or recurrent hypoxemia during sleep. These differences in preoperative oxygen regulation suggest intrinsic alterations in ventilation. With this background of ventilation disturbances, hypoxia and episodes of airway obstruction occur following surgery. Available evidence suggests that no ventilatory adaptations occur in the timeframe of the immediate recovery from surgery and that ventilation is unchanged by the surgery itself, so whatever ventilatory issues present at the time of surgery persist.

Respiratory Drive
Obstructive sleep apnea (OSA) is a clear risk factor for postoperative respiratory complications and an apnea-hypopnea index (AHI) greater than 10 is 1 threshold for complications, although some have found mild OSA as much a risk factor for postoperative desaturations as more severe OSA. As the severity of nocturnal hypoxemia increases, the incidence of respiratory complications increases. This is probably due in part to an increased susceptibility to opioid analgesia in patients with nocturnal hypoxemia. Chronic intermittent hypoxia and sleep apnea upregulate opioid receptors favoring inhibitory µ receptors rather than excitatory neurokinin 1 receptors, predisposing children with sleep apnea to exhibit a heightened respiratory and analgesic sensitivity to narcotics. Increases in interleukin (IL)-1β and PGE₂ impair respiration and adversely alter autoresuscitation of respiratory drive.

Children with OSA have normal ventilator responses while awake, although some subtle changes have been reported by others. Obstructive symptoms from adenotonsillar hypertrophy are associated with diminished responsiveness to CO₂ in children. It is important that the episodes of adverse

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respiratory events following tonsillectomy occur during sleep, not while awake. Central sleep apnea intrinsically occurs at a higher rate in children, particularly obese children with OSA, suggesting some abnormalities in ventilator control. A blunted respiratory drive to CO$_2$ during sleep despite normal findings when awake is not uncommon in obese children. When both OSA and central sleep apnea are present, patients appear to have a narrower reserve of PaCO$_2$ between eupnoea and the apneic threshold than patients with just OSA, indicating greater respiratory control instability.

Silent Death Pathophysiology

We posit that tonsillectomy patients die silently due to insufficient oxygenation of the brain to derive an arousal, once the point of no return desaturation has been surpassed. Repetitive reductions in airflow and SPO$_2$ during sleep from repeated collapses of the upper airway lead to brief arousal. Each arousal leads to a brief hyperventilation, which drives PaCO$_2$ below normal. The drop in PaCO$_2$ causes a decrease in central drive and a further drop in upper airway tone. Because the upper airway is unstable, it collapses again, causing the cycle to repeat. For OSA patients experiencing repeated episodes of collapse and hypercarbia, these effects may take oxygen desaturations beyond the point of no return, resulting in silent death.

Experimental data suggest that arterial oxygen can fall precipitously without hypercarbia development when starting from a non-oxygen-enriched environment. Each airway cycle takes the oxygen levels lower, providing the foundation for the final oxygen desaturation. Narcotics and cyclic hypoxemia, common elements in postoperative tonsillectomy patients, are known causes of arousal failure and likely contribute to the progression beyond the point of no return saturation. Compression of the range from arousal to the point of no return may occur from the repetitive exposures to rapid declines in SaO$_2$ saturation over time. As a result, increasing levels of arousal are needed to trigger a response. Patients with initial high hypercapnic ventilator responses would appear to be at greatest increased risk for sudden death following tonsillectomy independent of narcotic administration.

Treatment Issues

Narcotics

Narcotic exposure following tonsillectomy profoundly depresses respiratory drive through depression of both central and peripheral chemoreception and increased central sleep apnea. Studies of the effects of narcotics on breathing during sleep are limited, even among healthy patients with no respiratory complaints. Findings have demonstrated that narcotics increase the frequency of central sleep apneas, decrease minute ventilation, increase hypercarbia pressures, and decrease the hypoxic ventilator response without changes in hypercapnic responsiveness or pharyngeal resistance with mild oral narcotic doses in normal patients. When opioids interact with the altered and atypical neural environment following tonsillectomy, the suppression of respiratory drive may lead to silent death. Narcotic exposure appears to be the clearest link between desaturations in the first 3 postoperative days following tonsillectomy, the days of highest narcotic use, and silent deaths following tonsillectomy.

Monitoring Observation

Obstructive events will occur beginning the day of surgery in virtually every child following tonsillectomy. Independent of any factors that may predispose children to respiratory embarrassment, we rely on monitoring systems to identify episodes of respiratory compromise before adverse events occur. The 2 mainstays of our current monitoring—periodic assessments of breathing and conventional pulse oximetry—are probably inadequate to identify clinical instability early in its evolution.

Foundationally, the assumption that children who experience respiratory trouble following tonsillectomy show some observable distress appears to be faulty. Published anecdotal reports simply do not support such an assertion. Specifically, the respiratory rate is what is most commonly monitored. An altered, typically lower, ventilation rate is considered to be a reasonably accurate indicator of respiratory depression caused by narcotics. Admittedly, parenteral narcotics usually produce a slowing of ventilation rate. However, there are pitfalls in the reliance on bradypnea as a sign of respiratory depression. In normal volunteers, a depression in tidal volume with little or no change in respiratory rate was elicited when carefully examined. When postoperative epidural anesthesia patients were examined, no change or increase in ventilation rate was evident despite a reduction in minute ventilation. Even after narcotic administration following elective thoracotomy, one-third experienced episodes of apnea not preceded by slow respirations. Bradypnea, the specific outcome sought, may be infrequently associated with critical hypoxemia and may not be a sign of marked respiratory depression, just as a normal respiratory rate does not exclude respiratory depression or oxygen desaturation. Simple observation, even in an intensive care setting, has proven to be inadequate to reliably identify respiratory compromise prior to respiratory failure.

Oximetry

For pulse oximetry, the standard monitoring tool on hospital general care wards, the key assumption inherent in its use is the genu of the oxyhemoglobin dissociation curve identified by the 90% value. This assumption is far from reliable in post-tonsillectomy patients. Respiratory alkalosis, commonly seen with hyperventilation, can perpetuate SpO$_2$ values well above 90% relatively independent of lowered PaO$_2$ such that SpO$_2$ values fall below 90% only in terminal phases. Conventional threshold pulse oximeters may not be capable of identifying cyclical desaturation clusters or arousal failure. Their signal sampling smoothing and averaging algorithms may render arousal failure difficult to specifically identify.
The suggestion has been made for home oximetry monitoring following tonsillectomy; however, capnography more readily detects hypoventilation compared with pulse oximetry or visual observation and thereby provides a better opportunity for early recognition of depressed respiratory activity and may be a better choice.

**Recommendations**

Some perspective is warranted. Deaths from all causes following tonsillectomy have been reported to occur in the range of 1 in 30,000, an order of magnitude short of the risk of being struck by lightning. By any measure, the vast majority of 500,000 children undergoing tonsillectomy do well, yet 20 to 30 will not survive the surgery. When posttonsillectomy respiratory disaster strikes, it most often follows uneventful surgery carried out by a competent surgical team on an otherwise unremarkable patient. The effect on the family, surgeon, and medical community is often quite profound. Economic arguments for change are unlikely to be justified due to the infrequent nature of the event. Most surgeons, regardless of their compliance with published treatment recommendations, will never lose a child. We would be wise to remember that lightning does strike and we do take precautions to prevent being struck. For tonsillectomy, our personal experience is not sufficient to justify our behavior.

Setting expectations for caregivers of children about expected postoperative pain and identifying the frequency and dose of pain medications are warranted. Intraoperatively, the least morbid surgical technique should be used to reduce postoperative pain. Imparting a clear understanding of the adverse outcomes that can occur and what caregivers should do to obtain care to mitigate them is imperative.

The role of narcotics in postoperative analgesia following tonsillectomy remains unclear. The sensitivity of a child’s respiratory drive or propensity for central sleep apnea in response to narcotics is unknown. The presence or absence of early desaturations or any other clinical marker identified to date may not reflect narcotic sensitivity or cumulative or progressive pathophysiology that may take days to manifest, so practitioners must remain vigilant.

The goal of narcotic pain management should be only to attempt to marginally control pain rather than alleviate it. Fortuitously, the typical narcotic pain regimens following tonsillectomy inadequately relieve pain. The uncontrolled acute pain stimulates respiration and antagonizes the respiratory depressant effects of opioids, providing a greater margin of safety. Prudence, recent guidelines and US Food and Drug Administration recommendations suggest that codeine should be avoided together. The rationale for around-the-clock administration of narcotics is dubious given the risks, despite recent reports of their safety.

**Future Implications**

Most of the tonsillectomies performed in the United States are done on an ambulatory basis. Whereas 30 years ago, tonsillectomy was a 3-day admission, the average time from operation to discharge for tonsillectomy is now often less than 1 hour. It is unlikely that current postoperative monitoring of patients will permit the identification of those at risk of respiratory complications. Although our discussion has focused on tonsillectomy, any surgery requiring significant narcotic pain use postoperatively is likely to be similarly affected. The greater number of tonsillectomies performed in the United States may simply account for our current recognition of this problem.

**Author Contributions**

Reginald F. Baugh, data analysis, drafting, final approval, responsibility for content of manuscript.

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