The Elephant in the Room: Lethal Apnea at Home after Adenotonsillectomy

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Imagine the heartbreak of a parent who gives permission for his/her child to have a tonsillectomy only to find the child dead in bed shortly after surgery. In this issue's "Death or Neurologic Injury after Tonsillectomy in Children with a Focus on Obstructive Sleep Apnea: Houston, We Have a Problem!" Cote et al.[1] report 86 children who died or suffered permanent neurologic injury after adenotonsillectomy (T&A).[1] A similar survey of otolaryngologists documents 38 cases of death/anoxic brain injury in children after T&A.[2] As response rates to both surveys were low (30% and 22%, respectively), the number of cases was undoubtedly underestimated.

Although post-T&A deaths have most often been linked to hemorrhage,[3] hemorrhage accounted for only 28%[1] and 15%[2] of the cases in the 2 reports. Excluding a few surgical or anesthetic misadventures, most cases were suggestive of an apneic event/respiratory insufficiency, sometimes associated with narcotic use. Cote et al.[1] report that a greater proportion of children at risk for obstructive sleep apnea (OSA) had death/permanent neurologic injury attributed to postoperative apnea, whereas "all others" had a greater proportion of events attributed to hemorrhage. Most of the pediatric cases reported by Goldman et al.[2] are likewise suggestive of postoperative apnea as the child was found unresponsive in an unobserved and/or unmonitored setting. The majority of deaths/permanent neurologic injuries occurred following discharge from hospital.[1,2] Both authors have acknowledged the elephant in the room, a concern long known but little discussed: following T&A, children are suffering lethal apneic events at home.

As most of the deaths occurred in an unmonitored setting, the cause of death was often specified as "unknown" or "unexplained." It seems likely that the final event in most cases was either a central or obstructive apnea. Previous studies have demonstrated important respiratory insufficiency on the initial nights following T&A.[4–8] Nixon et al.[9] monitored 10 children who had T&A for OSA using polysomnography the night after surgery. All children were awake and drinking the afternoon of surgery, but during sleep all children continued to have apneic events and dips in saturation to <90%. Sleep quality was poor in all children. Five patients with severe OSA, characterized by repetitive episodes of desaturation to <85% preoperatively, had on the first night following T&A, a high frequency of apneas and hypopneas: 21 events per hour (interquartile range 15–112) with nadir saturations to 83% (interquartile range 73%–89%). Despite removal of enlarged lymphoid tissue, saturation measurements, in several cases, were worse the night after surgery than preoperatively.

Following T&A, opioids were administered to 50%[1] and to 87%[2] of patients who suffered death/neurologic injury. Opioids result in a dose-related decrease in the ventilatory response to hypercarbia and hypoxemia. Children with severe OSA have a blunted response to hypercarbia.[10] During sleep, the combination of opioids and severe OSA acts to attenuate the main determinants of the respiratory drive. Furthermore, termination of an obstructive apnea in children is dependent on an arousal from sleep and/or reflex recruitment of the upper airway dilating muscles, both of which are blunted by opioids.[11–13] Goldman et al.[2] specifically implicated opioids in 8 pediatric deaths related to ultrarapid metabolism of codeine, overdose, or nonprescribed opioids. We emphasize, however, that all narcotics depress the arousal response to asphyxia, induce central apnea, and depress the upper airway musculature required to maintain upper airway patency.[11,12] Preoperative nocturnal hypoxemia, which occurs with severe OSA, lowers the required narcotic dose to alleviate pain.[14] In children with OSA, desaturation <85% on a preoperative sleep study reduces the post-T&A analgesic opioid requirement by half.[15,16]

To quote Cote et al.,[1] "death due to apnea is preventable." The authors of both the articles recommend a national dialogue to discuss aspects of patient safety and resource allocation. What risk factors are known to be associated with post-T&A life-threatening events? Is there a readily available method to determine the risk for these events? What data are needed to inform public policy? How can practice be modified to avoid such catastrophes?

WHAT RISK FACTORS ARE KNOWN TO BE ASSOCIATED WITH POST-T&A LIFE-THREATENING RESPIRATORY EVENTS?

Previous work has identified several risk factors that predict serious post-T&A respiratory events: age <3 years;

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significant cardiorespiratory, neurologic, craniofacial and genetic disorders; and severe OSA. Indeed both Cote et al.[1] and Goldman et al.[2] identify a high proportion of children suffering death/neurologic injury following T&A as having associated neurologic, genetic, cardiorespiratory disorders, or obesity. Severe OSA is the risk factor that is hardest to define as the history and physical examination are notoriously imprecise.[17,18] Severe OSA is often quantified as an apnea–hypopnea index in excess of 10 events per hour and a saturation nadir <80%. It is generally accepted that these high-risk patients should be monitored as inpatients the night after surgery.[3–5,19,20] Polysomnography is considered the most reliable and comprehensive method of diagnosing and quantitating the severity of OSA.[3,20] Such testing requires in-laboratory overnight multichannel and video recording of sleep and breathing with a highly trained technician in attendance. However, there are more than 500,000 pediatric tonsillectomies performed annually in the United States alone, a number that dwarfs the capabilities for pediatric polysomnography. This capacity is further challenged by the increasing number of children undergoing T&A who are being managed as outpatients.[21]

IS THERE AN ALTERNATIVE, READILY AVAILABLE METHOD TO DETERMINE BOTH THE SEVERITY OF OSA AND THE RISK FOR LIFE-THREATENING RESPIRATORY EVENTS?

In Montreal, Quebec, Canada, we reserve polysomnography for complex cases and have developed an expedited diagnostic system for OSA that is based on history, physical examination, and oximetry. Our goal has been to identify the severity of OSA in those children with adenotonsillar hypertrophy who do not have comorbidities. These are the children who present the greatest challenge to ambulatory T&A programs as the signs and symptoms of severe OSA are imprecise.[17] The rationale for basing a severity scoring system on oximetry is that the frequency of desaturation events has a close correlation (r = 0.88) with the apnea–hypopnea index, the most frequently used metric for adult and pediatric OSA.[22] Using polysomnography for diagnosis of OSA requires the full resources of a sleep laboratory whereas use of oximetry requires that parents perform the oximetry at home, a much less expensive approach. The McGill Oximetry Score has a 97% positive predictive value versus polysomnography, although an inconclusive study does not rule out milder OSA.[19,22] Night-to-night consistency showed agreement on 143 of 148 tests (r = 0.90).[23]

Beyond confirming a positive diagnosis of OSA, the McGill Oximetry Score provides an estimate of OSA severity with categories 2, 3, and 4 specifying repetitive desaturation to <90%, <85%, and <80%, respectively. Post-T&A respiratory complications were more common with increasing oximetry scores agreeing with other studies showing increased complication rates with lower nadir saturations.[4–6,9] Furthermore, the metrics of nocturnal hypoxemia have proven useful to inform the anesthetic and analgesic management of individual patients.[24] However, until third-party payers agree to reimburse oximetry as a diagnostic test for pediatric OSA, implementation will likely continue to lag.

WHAT DATA ARE NEEDED TO INFORM PUBLIC POLICY?

Estimates for mortality rates following T&A (1 in 16,000 to 1 in 35,000) are based on data from the 1970s[3] and predate the shift to outpatient T&A and the shift to perform T&A for obstructive breathing rather than infection.[21,25] Because OSA is a risk factor for respiratory morbidity following T&A,[3] mortality estimates relevant to current practice are needed. There is an urgent need for national, population-based outcome monitoring. Such a process should link mortality in the month following operation to a registry of operations performed. Goldman et al.[2] report that the American College of surgeons is working on such registries, but this effort will require collaboration between otolaryngologists, anesthesiologists, and pediatricians. A National Institute of Health consensus development conference could facilitate the necessary dialogue among stakeholders.

HOW CAN PRACTICE BE MODIFIED TO AVOID SUCH CATASTROPHES?

Cote et al.[1] comment on the performance pressures to efficiently manage patients. For many ambulatory T&A programs, performance is assessed with metrics of time. Modern anesthetic practice often allows a child to meet discharge criteria within an hour of surgery. Yet, a child with residual risk for OSA following T&A may breath adequately when awake but become severely obstructed when asleep.[9] Indeed, Cote et al.[1] suggest that 16 children could have been rescued if proper monitoring had continued—without defining the proper monitor. Most bedside monitors detect respiration with thoracic impedance plethysmography, a technology that is poorly suited to monitor children with OSA as it detects only central apnea. Thoracic impedance plethysmography does not distinguish obstructed breaths from normal unobstructed breathing.[26] It is our routine following T&A to monitor all children with oximetry in the initial recovery period and to continue monitoring saturation during sleep in the high-risk patient. Discharge criteria following T&A should recognize the risk factors for sleep-related airway obstruction.

As "Houston reflects on the problem," anesthesiologists should be cognizant of the limitations of a clinical diagnosis to both quantitate OSA severity and predict respiratory morbidity and mortality following T&A. Risk factors including young age (<3 years), medical comorbidity, and African American ethnicity signal a higher risk for perioperative respiratory adverse events following T&A.[3,27] The efficacy of non-opioid analgesia requires study. When opioids are used, dosage should be adjusted for the severity of OSA with provisions that children receive the minimum effective dose. New recommendations from the American Academy of Pediatrics recommend that, even in the absence of a preoperative sleep study, children who exhibit desaturation <80% following T&A should be admitted to hospital and monitored for respiratory depression.[20]

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