



EDITORIAL

Predicting and managing the development of subglottic stenosis following intubation in children^{☆,☆☆}



Previsão e manejo do desenvolvimento da estenose subglótica após a intubação em crianças

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Post-intubation stridor is a common problem in the pediatric intensive care setting (over 44% in the current article by Schweiger et al.¹ Yet the incidence of airway complications associated with intubation is relatively low. A recent prospective study reported an incidence of post-intubation subglottic stenosis in children was 11.38%.² The challenge is identifying which children are at particular risk of developing airway compromise, or alternatively identifying those at low risk of airway compromise.

The risk factors for the development of airway complications related to intubation are well known. The size of the endotracheal tube relative to the size of the airway remains the single most important variable, but the duration of intubation,^{3–5} whether the intubation was traumatic or not, the number of intubations, agitation, nasal vs. oral intubation, the composition of the endotracheal tube, and factors predisposing to inflammation (e.g., gastroesophageal reflux, viral infection) are all factors to consider

as well.⁶ A new prospective study even implied that under-sedation might be a risk factor for the development of subglottic stenosis in intubated children.⁷ The key concept is that the appropriate size of the endotracheal tube is not the age-appropriate tube, but rather the child-appropriate endotracheal tube. While ideally having an endotracheal tube with a leak pressure of less than 20 cm of water will minimize the risk of iatrogenic airway trauma,^{8,9} in some children the ventilatory needs are such that the risk of post-intubation airway compromise has to be tolerated – in such children, the smallest endotracheal tube that will provide adequate ventilation is a better management guideline. An alternative approach is the use of a smaller-diameter cuffed endotracheal tube. This will carry a lower risk of iatrogenic laryngeal trauma, but attention needs to be paid to cuff pressure to prevent the formation of tracheal stenosis. Although some studies demonstrated that the use of cuffed tubes in pediatric population may not increase respiratory complications,^{10–12} robust evidence is still lacking.¹³ The group at highest risk of cuff-related tracheal stenosis are agitated teenagers who have sustained a brain injury.

In the current study the incidence of post-intubation stridor and the development of subglottic stenosis (SGS; 9.62%) are relatively high in comparison to many other studies. This would appear to be a consequence of the indications for intubation rather than the subsequent management. This is a relatively young cohort of patients (median age 2.7 months), 63.1% of whom were intubated for bronchiolitis.

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Moreover, intubation and viral infection are synergistic in the development of subglottic stenosis.

The potential airway complications of intubation include the development of glottic and subglottic granulation, cricoid ulceration, posterior glottic stenosis, cricoarytenoid joint fixation, SGS, and tracheal stenosis. The stenosis may initially be soft and immature, and may be reversible, or may progress to cicatricial scar formation and a fixed stenosis. The signs and symptoms of airway compromise may be overt or covert. The most noticeable is stridor, typically inspiratory or biphasic. Biphasic stridor is typically seen with a fixed stenosis, while inspiratory stridor is more typically seen with a dynamic collapse (laryngomalacia, vocal cord paralysis, glottic granulation). Stridor should not be confused with an expiratory wheeze. While stridor is the sign that draws the most attention, retractions (whether suprasternal, intercostal or subcostal) are a much better indicator of the severity of an obstruction. In a child with stridor but no retractions, the airway is unlikely to be significantly compromised. However, a child with a severe stenosis may have minimal stridor, yet have marked retractions. Other symptoms may include a hoarse voice, apneas, and cyanosis. Symptoms are typically worse when agitated, or during exertion (primarily when feeding in a baby). With the development of stenosis in the airway, symptoms are typically progressive, and may evolve over a period of weeks.

In a child with post extubation stridor, it is not mandatory to investigate, as often the stridor is mild and transient. However, if the stridor is severe, late onset, or progressive, then investigation is warranted. The single most valuable non-invasive investigation, if available, is an awake transnasal flexible laryngoscopy. This is fast, low risk, does not involve sedation, and provides valuable information about the upper airway, from the nasal aperture to the vocal cords. This is best done with the child sitting up or being supported in an upright position. For dynamic laryngeal problems, including laryngomalacia and vocal cord movement impairment, it is invaluable, and it may also provide valuable information about laryngeal granulation and subglottic stenosis. However, it requires the right equipment and personnel, typically an otolaryngologist. If the child is symptomatic, and there is little evidence to be seen on flexible laryngoscopy, this implies a more distal pathology, and a bronchoscopy under general anesthesia is recommended.¹⁴ Other investigations include imaging of the airway (airway films, CT scan, etc.), and in older stable children, pulmonary function tests may be of value.

However, the gold standard for airway evaluation currently is microlaryngoscopy and bronchoscopy with a rigid Hopkins rod endoscope (whether through a ventilating bronchoscope or with the telescope alone). The optics are superb, and for evaluation of laryngeal and tracheal pathology, especially SGS and posterior glottic stenosis, it remains the recommended investigation in a significantly symptomatic child. Flexible bronchoscopy has some advantages evaluating airway dynamics and malacia, and accessing the peripheral bronchial tree. However, flexible bronchoscopy is not a reliable tool for evaluation of the posterior glottis (e.g., evaluating for posterior glottic stenosis or a laryngeal cleft).

Management of a child with post-extubation stridor may be expectant in most cases, especially if the stridor is mild.

The underlying cause of stridor is tissue reaction at the interface of the endotracheal tube and the laryngeal or tracheal mucosa, and removal of the inciting cause is key to tissue recovery. However, following extubation, there may be reactive edema of damaged mucosa, and obstruction (with resultant stridor and retractions) may become transiently worse over the first 24–36 h before improving. Over this time, supportive measures to prevent the need for re-intubation are recommended, including the use of racemic epinephrine,¹⁵ steroids, heliox,¹⁶ positive airway pressure, or nebulized steroids.¹⁷ If re-intubation is required, a smaller tube may be used to maintain a leak, and steroid antibiotic combination drops may be placed down the tube to help with granulation and swelling. Re-intubation through a different route also is of value – swapping an oral tube to a nasal tube, for example.

A child failing extubation, or in whom stridor is still present after 72 h, in whom stridor occurs after 72 h, or in whom obstruction is getting progressively worse, evaluation should be performed in the operating room, for consideration of intervention. Interventions may be endoscopic, open, or serve to bypass the obstruction. A tracheotomy serves to bypass the obstruction, but may not prevent the need for later endoscopic or open intervention to achieve decannulation.

Endoscopic management may be as simple as removing granulation tissue. However, if stenosis is present, whether from soft tissue edema or from scar tissue, then other endoscopic interventions may be considered. The key is patient selection. If the mucosa is very “unhealthy” then it may be better to place a tracheotomy and wait until the larynx is quiescent, and then consider intervention. If there is stenosis present, then considering the cartilaginous “exoskeleton” of the laryngotracheal complex is helpful in planning interventions. If the outer cartilaginous framework of the airway is intact, and there is intraluminal scar, then endoscopic interventions such as steroid injections, scar division, and balloon dilation may be effective. But if the cartilaginous structure is compromised, whether congenitally narrow, such as an elliptical cricoid causing subglottic stenosis, or there is mucosal ulceration exposing damaged cartilage, then endoscopic procedures may be ineffective.

If open surgery is required, whether to prevent the need for a tracheotomy or to allow removal of a tracheotomy, there are three main classes of procedure to assist with stenosis, namely expansion surgery, resection surgery, and the slide tracheoplasty. Expansion surgery, or laryngotracheal reconstruction (LTR), involves placing cartilage grafts to expand a stenotic segment of the airway. This may be done for posterior glottic stenosis, SGS, or tracheal stenosis, and the grafts may be placed in the anterior cricoid, anterior trachea, or posterior cricoid. Costal cartilage is the graft material of choice. Resection surgery (tracheal resection or cricotracheal resection [CTR]) involves removing a scarred segment of the airway, and anastomosing healthy tissue to healthy tissue. CTR is an operation for treating SGS, and for severe stenosis has better outcomes than LTR.¹⁸ However, it is a more challenging operation, with a higher risk of post-operative complications. The slide tracheoplasty is an operation for treating tracheal stenosis, and expands the size of the stenosis by transecting the airway and overlapping the stenotic segments.

These various potential interventions are time dependent – it is more challenging dealing with a fixed, thick cicatricial scar than a soft tissue stenosis, and medical management and endoscopic interventions – if implemented early enough – may prevent the need for more complex invasive surgery. Therefore early identification of patients at risk for the development of SGS is of value. This study examines one clinical indicator, namely post-extubation stridor, and relates this to findings on transnasal flexible laryngoscopy, and in the more symptomatic cases, the findings on bronchoscopy. For children with stridor present 72 h after extubation, the positive predictive value was 40%, and the negative predictive value was 96%. This does suggest that bronchoscopic evaluation in an operating room is not required in children who do not have stridor 72 h following extubation. However, the presence of stridor 72 h after extubation does suggest that consideration should be given to formal evaluation of the airway in an operating room setting, as subglottic stenosis may be present in 40% of cases, and be potentially amenable to endoscopic intervention. In this series, 18 patients had SGS, eight requiring open reconstruction, but with a further eight only requiring endoscopic intervention (usually balloon dilation). Of the remaining two patients, one died of sepsis, and one did not require intervention.

In conclusion, post-extubation stridor is common, while intubation related SGS is comparatively rare. There are few current guidelines to suggest which patients warrant further evaluation. The study by Schweiger et al.¹ is valuable in suggesting that if stridor is persistent after 72 h, then evaluation for possible SGS, and appropriate early intervention, may prevent the need for complex subsequent airway reconstruction in a significant number of children.

Conflicts of interest

Dr. Rutter is a consultant for Tracoe and Bryan Medical. He has a patent for a balloon dilator that is licensed by Bryan Medical, for which he receives royalties. He developed a suprastomal stent marketed by Boston Medical Products that he has declined royalties on. Dr. Kuo has no disclosures.

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