Clinical Brief

# Ventilatory Management of Severe Tracheal Stenosis

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**ABSTRACT.** We present here a 4 year old child with severe tracheal stenosis and respiratory failure. The patient was not responding to conventional ventilation settings and had significant hypercarbia. The difficulty in mechanical ventilation was handled successfully with specific ventilatory strategy: use of low respiratory rate, long inspiratory time and normal inspiratory time : expiratory time ratio. Thereafter the child was managed surgically and the stenosis was corrected. The child was discharged after a Montgomery T-tube placement. **[Indian J Pediatr 2006; 73 (5) : 441-444]**
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Laryngotracheal stenosis is a congenital or acquired narrowing of the airway that may affect the glottis, subglottis or trachea. It causes severe symptoms and should be suspected in children less than 1 year of age with either multiple episodes of croup or croup which fails to respond to medical management or requires endotracheal intubation.[1] The term subglottic stenosis was previously used as the subglottic region is the most common site of airway stenosis, mostly secondary to prolonged endotracheal intubation. The onset may be acute or it may develop over a period of time and a thorough assessment, radiologic and endoscopic evaluation may be necessary to guide further therapy and management. We present a case of acquired tracheal stenosis that required prolonged ventilatory support with adaptations for the obstructive pathology and discuss the implications for ventilation of a child with upper airway obstruction.

## CASE REPORT

A 4-year-old boy presented with history of intermittent high grade fever and cough without expectoration of 3 months' duration. After 5 days of onset of fever, he developed noisy breathing and progressively increasing respiratory distress. There was no history of cyanosis, wheezing episodes or any history of foreign body aspiration. There was no history of contact with tuberculosis and no past history of any significant illness. The respiratory distress and the intensity of noisy breathing increased gradually over 3 months and were now also persisting during sleep. There was no history suggestive of diphtheria and the child was immunized for age.

On presentation, the child had marked respiratory distress, was sitting up bending forward and had marked suprasternal recessions and severe biphasic stridor. He had a respiratory rate of 48/minute, and had good volume pulses with no cyanosis and $SpO_2$ of 99% on facemask oxygen. Chest examination showed hyperinflation of the chest wall with symmetrical movements of both sides of the chest wall, centrally placed trachea on palpation and equally resonant percussion note on both sides. Chest auscultation revealed equal air entry on both sides and conducted breath sounds. Examination of cardiovascular and other body systems was unremarkable. Initial arterial blood gas analysis showed a $pCO_2$ of 48.6 mm Hg and $PaO_2$ of 198.4 mm Hg with pH of 7.391.

At presentation the possibility of severe upper airway obstruction was kept and radiographs of soft tissue of neck (lateral view) did not show any evidence of epiglottitis or narrowing of the cervical portion of the trachea. Chest X ray showed bilateral hyperinflated lung fields with no lung parenchymal abnormalities and normal cardiothoracic ratio (48%). However, over the next few hours child showed progressive deterioration with rising $pCO_2$ values up to 80 mm Hg and had to be intubated for respiratory support and was therefore transferred to the Pediatric Intensive Care Unit (PICU) for further management.

On arrival to the PICU he was ventilated with Siemens Servo 300 ventilator on SIMV (volume controlled) mode with initial conventional settings but did not show adequate chest rise and had progressive $CO_2$ accumulation (values up to 150 mm Hg) on blood gas analysis. The initial settings were: rate 30/min, tidal volume 80 ml, Ti 0.7 seconds, I:E ratio of 1:2 and positive

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end expiratory pressure of 4 cm $H_2O$. On increasing the tidal volume alone, there was no further improvement in $PaCO_2$ levels assessed after 1 hour. The peak pressure requirement was 38 cm $H_2O$. On bagging with self-inflating bag, high resistance was felt and adequate chest rise could be achieved only if long inspiratory time was used. Subsequently the settings were modified in keeping with the requirements so that the ventilator now had low rates (10/min) and a prolonged inspiratory time (Ti) of 1.8 seconds, an I:E ratio of 1:2 and PEEP of 5 cm $H_2O$. With these settings, he maintained oxygenation and showed improvement in $PaCO_2$ levels. The peak pressure requirement also fell to 23 cm $H_2O$. The rationale for these settings is discussed below.

Subsequently, a fibreoptic bronchoscopy was done through the endotracheal tube in the PICU after initial stabilization and it showed severe narrowing at the distal end of the trachea just above the carina. A diagnosis of supracarinal tracheal stenosis was established and the child underwent endoscopic balloon dilatation of trachea under general anaesthesia the next morning. Intraoperatively, the stenotic area showed some granulation tissue with inflamed mucosa and loss of tracheal rings, and from this a biopsy specimen was obtained and dilatation was done with Fogarty's Catheter. An endotracheal tube of 4.5 Fr size was inserted under bronchoscopic guidance with tip extending beyond the stenotic segment and lying above the carina. He was subsequently continued on ventilatory support for the next 10 days with gradual weaning. A diagnosis of tubercular tracheal stenosis was considered and antitubercular therapy was started but subsequent work up did not reveal any evidence of tubercular infection. However, there was satisfactory clinical response as the fever subsided and tracheal granulations on repeat examination were absent. The biopsy form the lesion showed mild non-specific inflammatory changes with no granulomas.

A repeat fibreoptic bronchoscopy done 8 days after the dilatation procedure in the PICU showed no significant stenosis but persistence of inflamed mucosa. He was then weaned off ventilatory support but endotracheal tube was

**Table 1. Case-series of Tracheal Stenosis**

| Series | No. | Etiology | Techniques used | Outcome |
|---|---|---|---|---|
| (1) Kumar P *et al*[7] | n = 17 | External vascular compression=9,<br>Post transplant strictures=4<br>Malignant masses=2<br>Postintubation =2 | Airway Stenting with self-expanding metal stents in 10 cases, silicone stents in 7. | 47% alive on follow up, 6/8 ventilator dependent extubated |
| (2) Anton-Pacheco JL *et al*[8] | n = 13 | All cases Congenital;<br>Mild – 4<br>Moderate – 6,<br>Severe – 3 | Costal cartilage tracheoplasty (CCT)=5,<br>Tracheal resection = 3,<br>Slide tracheoplasty=2,<br>Endoscopic dilation=3<br>Laser Resection=1 | Overall mortality 23%, 3 early deaths-all after CCT |
| (3) Dunham ME *et al*[9] | N= 23 | Congenital complete tracheal rings producing long segment stenosis of trachea | Pericardial Patch Tracheoplasty | 83% survival at mean follow up of 4.5 years |
| (4) Har-El G *et al*[10] | N= 19 | Post-intubation or tracheotomy in 80% | Circumferential tracheal resection with end-to-end anastomosis | Anastomosis success rate of 94.7% |
| (5) Loeff DS *et al*[11] | N= 22 | Vascular rings/slings in 50% | Localized-dilatation, tracheostomy, and resection with end-to-end anastomosis, Funnel shaped defects-tracheal reconstruction with grafts | Overall mortality rate 77% |
| (6) Weber TR *et al*[12] | N= 62 | Acquired Tracheal Stenosis (4 weeks to 14 years age)<br>Endotracheal intubation-44<br>Caustic aspiration-6<br>Recurrent infection-5<br>Bronchoscopic perf-4<br>Gastric aspiration-3<br>Site of airway stenosis<br>Subglottic/upper – 47<br>Midportion-8<br>Distal/Carinal-7 | Individualized treatment:<br>Balloon dilatation-20,<br>Bronchoscopic electrocoagulation resection- 44, Steroid injection-48, T tube stent-8,<br>Resection anastomosis-12,<br>Cricoid split-3, and<br>Rib cartilage graft-12. | 7(11%) died of unrelated causes. 44 of 55 patients (80%) are without tracheostomy. |

left in situ as it was bypassing the stenosed segment. He had recurrence of respiratory distress, which required resumption of ventilatory support and he then underwent repeat dilatation after 20 days of admission, which showed collapse and re-stenosis of trachea, which could be easily dilated. Tracheostomy and Montgomery T tube insertion was done for maintaining long-term patency of the airway. He was discharged from the hospital 2 months after admission and is on regular follow up

## DISCUSSION

The importance of appropriate ventilatory management of severe proximal airway obstruction is highlighted by this case and it is important to understand the rationale for the ventilatory settings described above. The trachea and the upper airway although considered to be relatively rigid conducting airways, do show some changes in caliber during the normal respiratory cycle. There is expansion of the intrathoracic airways along with the expanding lungs while the extrathoracic airway diminishes in caliber due to their intraluminal pressure being lower than atmospheric pressure. The reverse of this process occurs during expiration. If intrathoracic trachea is soft (tracheomalacia), the narrowing will accentuate during expiration due to positive intrathoracic pressure. The flow in these large airways is usually turbulent due to the high flow rates and this turbulence increases in the presence of an obstruction in the airway. This turbulence further increases the airway resistance.

The mechanics of critical tracheal stenosis is such that it would severely compromise delivery of gases beyond the obstruction and it would not allow adequate emptying out of the lungs as well. Other obstructive upper airway anomalies like subglottic stenosis could be overcome by use of tracheostomy, which would bypass the site of stenosis, but in more distal lesions of the trachea (as in this case, just above the carina) ventilatory management is more challenging.

With conventional ventilator settings in the patient with severe tracheal stenosis, there would be inadequate delivery of gases beyond the site of obstruction and there would be a build up of pressure proximal to it. In this scenario, there would be progressive $CO_2$ retention and inadequate lung expansion. Also because of inadequate emptying of the lungs during expiration due to limited airflow across the site of stenosis, there would be a progressive air-trapping and subsequent decrease in cardiac output. Increasing the airway pressures or frequency alone will not help overcome these effects of the obstruction. Increase in the airway pressure may increase the risk of barotraumas. In order to ensure adequate delivery of gases beyond the obstruction, it is necessary to prolong the inspiratory time (Ti) so that adequate lung expansion can be achieved. In view of the markedly increased resistance the time constant will also be increased justifying the need for high Ti. For this, the frequency of breaths would have to be kept at low values in order to allow adequate time for expiration also and at the same time avoid progressive gas trapping (auto-PEEP). It is unlikely that use of CPAP alone would have lead to adequate delivery of volumes across such severe tracheal stenosis.

There have been reports of use of percutaneous transtracheal jet ventilation for cases with tracheal stenosis and other forms of airway obstruction in the more proximal airways.[2] For patients undergoing surgical repair of the tracheal lesions, extracorporeal membrane oxygenation has also been used.[3] In animal models of tracheal stenosis, a comparison of conventional ventilation versus transtracheal jet ventilation showed that although there were no significant differences in $PaCO_2$ but mean peak airway pressure values, both at the distal portion of stenosis and at the proximal portion, decreased more significantly during jet ventilation than during conventional mechanical ventilation.[4] Overall the mean arterial pressure, mean pulmonary arterial pressure, central venous pressure, and cardiac output did not change significantly between conventional mechanical ventilation and jet ventilation with the stenosis. Pulmonary Artery Occlusion Pressure (PAOP) increased significantly more during conventional mechanical ventilation than during jet ventilation in animal models with stenosis.

During ventilation, the use of flow-volume loops would demonstrate plateaus in both inspiratory and expiratory limbs due to the limitation in both phases caused by the obstruction. Miller and Hyatt[5] showed that in patients with fixed airway obstruction, the FEV1 diminishes progressively as the resistance increases but the FVC may remain unchanged, suggesting that the flow rates will be reduced and the ratio of maximal expiratory and inspiratory flow at 50% of vital capacity ($MEF_{50}$/$MIF_{50}$) may approach unity (0.9 to 1).

Another contentious issue was the underlying cause of tracheal stenosis in this child since the duration of symptoms was 3 months and he had no respiratory symptoms prior to this illness. Initial impression of granulation tissue being visible on bronchoscopy directed towards a diagnosis of tuberculosis but the work up was negative and the biopsy from the lesion was inconclusive. However, the tracheal inflammation could be due to a tubercular lymph node in the mediastinum. Despite no objective evidence of tuberculosis, response to antitubercular therapy supports the diagnosis of tuberculosis. A possibility of acquired tracheal stenosis secondary to an initial episode of bacterial tracheitis was considered.

The most common cause of acquired laryngotracheal stenosis is prolonged endotracheal intubation, accounting for 90% cases. Other acquired causes include postinfectious scarring, autoimmune disorders (Wegener's granulomatosis, sarcoidosis), inhalation

injuries, blunt trauma to the neck, previous tracheostomy or cricothyrotomy, and gastroesophageal reflux.[6] Prior to the advent of antibiotics, scarring from infections like diphtheria and syphilis were usually a common cause of stenosis. In the trachea, the narrowing may be extrinsic or intrinsic, and diffuse or localized. Among the extrinsic lesions, disorders of the thyroid gland and great vessels are the most common while the intrinsic ones may be due to infectious, granulomatous, neoplastic, traumatic, immunologic and post-inflammatory conditions.

There are several case series of laryngotracheal stenosis in children, with majority of reports being that of surgical repair techniques (Table 1). The ventilatory management of these patients has not been highlighted but it does pose problems in the perioperative and post-operative period. There have been reports where unstable children with critical stenoses requiring high airway pressures for ventilation and still having $CO_2$ retention, have required initiation of extracorporeal support, which was followed by diagnostic endoscopy and then finally definitive surgical repair.[3] But these facilities may not exist at all institutions and initial optimal ventilatory support in such sick children may be crucial before they can be shifted to centers where surgical repair can be done. Therefore, it is only with a understanding of the mechanics of tracheal stenosis and its optimal ventilatory management that we can ensure adequate treatment and outcome for these patients by surgical or non-surgical (e.g. balloon dilatation) means.

In conclusion, this case demonstrates the importance of appropriate ventilatory management of airway obstruction caused by tracheal stenosis and highlights the fact that critical stenosis of trachea may be ventilated with low rates and high inspiratory times. Management of tracheal stenosis in children is complex and requires the teamwork of specialists involved in emergency management, intensive care and otorhinolaryngology.

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