

## Management of stridor in neonates and infants

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**Abstract.** Stridor is the sound caused by abnormal air passage into the lungs and can exist in different degrees and be caused by obstruction located anywhere in the extra-thoracic (nose, pharynx, larynx, trachea) or intra-thoracic airway (tracheobronchial tree). Stridor may be congenital or acquired, acute, intermittent or chronic. Laryngotracheal inflammation (croup) is the most common cause of acute stridor. Laryngotracheomalacia is the most common cause of congenital, chronic stridor.

Stridor is a clinical sign and not a diagnosis. The golden standard in the workup of stridor is an upper and lower airway endoscopy under general anaesthesia. Endoscopic examination under general anaesthesia requires a multidisciplinary approach and close cooperation between anaesthesiologist, paediatrician, ENT surgeon and nursing staff. Following this procedure, a place in the intensive care unit should be available for those cases presenting with stridor in which a definite diagnosis could not yet be established.

Although important, pre-endoscopy assessment including history, physical examination and radiological examination, is only a guide to the type and degree of pathology found during endoscopy. About 1 out of 10 infants are found to have lesions in more than one anatomical site of the upper aerodigestive tract.

### Definition

The basic functions of the upper airway and larynx are: ventilation, protection of the lungs during deglutition, clearance of secretions through coughing, and vocalization. Stridor is the sound caused by abnormal air passage into the lungs, exists in different degrees and has many causes. Accompanying phenomena such as aspiration, coughing, difficulties in feeding and changes in voice quality can be important additional signs in the evaluation of neonates and infants.

### Pathophysiology

The mouth and pharynx are much more compact in the neonate compared to the adult. Until 3 or 4 months of age, an infant's soft palate and epiglottis are in con-

tact. The mandible is relatively small with minimal space between the soft palate and the oesophagus. The infant's tongue touches all borders of the oral cavity. This makes the infant a preferential nasal breather for 2 to 5 months and nasal obstruction can lead to significant respiratory distress. The act of oral breathing is a reflex that often does not develop at all until several weeks after birth.

At birth, the thyroid cartilage lies at the level of the 2<sup>nd</sup> and 3<sup>rd</sup> cervical and the cricoid cartilage is situated at the level of the 3<sup>rd</sup> or 4<sup>th</sup> vertebral body. The infant's hyoid bone and larynx are situated more anteriorly and superiorly than that of the adult. With maturation, the larynx descends, resulting in vertical elongation of the pharynx and enlargement of the oral cavity. Swallowing difficul-

ties often arise around 4 months of age as the pharynx starts to elongate, leaving the larynx less protection from aspiration. In the adult, the larynx lies opposite the body of the 5<sup>th</sup> and the cricoid lies opposite the 7<sup>th</sup> cervical vertebra.

The vocal cords of the newborn infant are 6-8 mm long and the vocal processes of the arytenoids extend one half of that length. The posterior glottis' transverse dimension is approximately 4 mm. The subglottis has a diameter of between 5 and 7 mm. The trachea itself is about 4 cm long and has a diameter of 5 mm, growing to an adult size of 11 to 13 cm in length and 12 to 23 mm in width.

The Venturi principle explains the narrowing of the supraglottic airway during inspiratory effort. Poiseuille's law (flow through a cylinder is proportional to the radius to the fourth power) is

mainly important at the subglottic level, where upper airway dimensions are smallest and where a narrowing by 1 mm can increase airway resistance 16-fold while decreasing the cross-sectional area by 75%.<sup>1</sup>

### Diagnostic management

Stridor can result from abnormalities located in the nose, pharynx, larynx, subglottis or tracheo-bronchial tree. The purpose of a carefully taken history and clinical examination is to provide some orientation about the most likely causes of stridor in a child. In most children, the diagnosis of stridor can only be confirmed by endoscopy and this is the golden standard.<sup>2</sup>

The *history* of a child admitted because of stridor should include the following items:

- \* Prenatal and obstetric history, associated medical problems, evidence of birth trauma and intubation records. Important elements in a history of intubation are date of first intubation, duration, size of endotracheal tube, number of intubations, time interval between intubated periods and whether any of these intubations were traumatic. Was stridor present after extubation?
- \* Onset of stridor (immediately after birth, after a few weeks or months ...)
- \* Diurnal variation of stridor and relationship between airway symptoms and feeding. The body position in which stridor worsens can provide information about the possible aetiology.
- \* Growth charts should be reviewed to determine if the child has failed to thrive.

- \* Suspicion of foreign body aspiration or ingestion.
- \* Changes in quality of voice or cry.
- \* Symptoms suggestive of aspiration due to gastro-oesophageal reflux: postprandial cough, regurgitation, emesis, bronchospasm, laryngospasm, central apnoea, and bradycardia.
- \* Presence of nasopharyngeal reflux during feeding or poor sucking habits.
- \* Associated symptoms such as retraction, cyanosis, dyspnoea, cough, hoarseness, concurrent illness and general medical history.
- \* Pulmonary and neurological status.

During the *clinical examination*, it is important to observe the child's breathing pattern and behaviour. Ideally, the child should remain with the parent or caregiver while the physician determines the degree of distress. Inspiratory stridor is typically caused by extra-thoracic airway obstruction whereas expiratory stridor or a prolonged expiratory phase points towards bronchial obstruction or an obstruction in the distal part of the trachea. In the case of inspiratory and expiratory stridor, the cause might be found anywhere in the tracheobronchial tree. The characteristics of the stridor may be a clue to the site of upper airway obstruction. An inspiratory, harsh, high-pitched and crowing noise often indicates abnormalities in the larynx. Low-pitched stridor with snoring and excessive secretions is suggestive of a pharyngeal or nasopharyngeal obstruction. Stridor that is both inspiratory and expiratory with a prolonged low-pitched expiratory phase suggests obstruction of the trachea or main bronchi from compression or collapse.

Positional stridor most often results from laryngomalacia, micrognathia, macroglossia or vascular compression. In these cases, breathing can be improved by placing the child in the prone position with the neck extended. In the case of epiglottitis, the child assumes a characteristic sniffing position in an attempt to maximize the airway.

In the case of serious nasal obstruction such as with bilateral choanal atresia, mouth closure is associated with vigorous respiratory efforts and chest retraction. If the infant cries and takes a breath through the mouth, airway obstruction is relieved.

One should look for retraction, which may be either subcostal, intercostal or suprasternal. The severity of airway compromise is better correlated to the degree of retraction than to the degree of stridor. Cyanosis is usually a very late sign of a severely compromised airway.

Crying quality in an infant and voice quality in a child should be evaluated for weakness, hoarseness, breathiness, or complete absence. A normal voice does not rule out a laryngeal cause of stridor.

In the case of congenital stridor, one can open the neonate's mouth and pull the mandible and tongue forward. If these manoeuvres results in an improvement of stridor, the obstruction is at the level of the larynx or higher.

Babies with an obvious syndrome such as Pierre Robin sequence, Treacher Collins, Crouzon or Apert syndrome may present with upper airway problems due to oropharyngeal obstruction caused by micrognathia and a retroposed tongue.

After observation, the child's facies should be evaluated and

nasal patency must be assessed. This can be done by means of a small mirror or a Politzer balloon. Passing a nasal catheter to determine the patency of the nasopharyngeal airway is not recommended. The pharynx must be inspected for an evaluation of pharyngeal dimensions and tongue size. Neck and laryngeal structures should be palpated and jaw position has to be noted. A suspicion of epiglottitis is a formal contra-indication for pharyngeal inspection without an experienced anaesthesiologist or neonatologist who can immediately intubate.

The indication for *additional investigations* should be based upon judicious decision-making.

In the case of acute airway obstruction, oxygen saturation should be monitored keeping in mind that severe airway obstruction might be present without desaturation. In addition, CO<sub>2</sub> retention can be present without desaturation, especially in those cases where oxygen is administered.

Tostevin *et al.*<sup>3</sup> found that imaging studies have little role in the screening of a child presenting with stridor and the initial radiological assessment should be limited to a chest radiograph including the neck using soft tissue windows.

Lateral neck and Cincinnati views show the subglottis, oropharynx and nasopharynx and allow identification of gross pathologies such as rare space occupying lesions. More common disorders such as laryngomalacia and subglottic stenosis are rarely identified radiologically. Additional studies might be considered if abnormalities are found at laryngotracheobronchoscopy.

The main indications for CT and MRI are the confirmation of

an extrinsic compression detected during endoscopy. Contrast swallow studies might be indicated when a tracheo-oesophageal fistula is suspected.

Other investigations, which might be indicated in a particular case, are echocardiography, gastroscopy or pH probe studies.

When ordering these additional examinations, the extra-burden for the child caused by these procedures should be kept in mind, especially as the definite diagnosis comes from endoscopy.

#### *Laryngotracheobronchoscopy*

The following situations are indications for diagnostic endoscopic examination in stridor.<sup>4</sup>

1. Severe stridor
2. Progressive stridor
3. Stridor associated with unusual features, such as cyanotic attacks, apnoeic attacks, dysphagia, aspiration, failure to thrive, or a radiological abnormality
4. Stridor causing undue parental anxiety

Performance of a safe and accurate endoscopy in a child requires good cooperation between a skilled anaesthesiologist, ENT surgeon and nursing staff, proper endoscopic equipment and the availability of a bed in the intensive care unit.

Flexible endoscopy, performed at the ward or in the outpatient clinic, might be considered in a very young child or in an older child, who is able to cooperate during the procedure. Flexible endoscopy provides a dynamic image of the naso-oropharynx and larynx but the view obtained from the latter, is not always optimal. Limiting the investigation to

laryngoscopy alone should be regarded as incomplete in the case of congenital stridor. Friedman *et al.*<sup>5</sup> reported 661 endoscopic procedures and found 11.5% of the patients had lesions in more than one anatomical site of the upper aerodigestive tract. In about 5% of the cases, abnormal findings at flexible endoscopy were associated with a second pathology, which can only be detected during rigid laryngotracheobronchoscopy.<sup>2</sup>

Endoscopy under general anaesthesia examines the oropharynx, larynx, trachea, bronchi; often the oesophagus and upon indication the nasal cavities and oropharynx.<sup>4</sup> Limiting the examination to laryngoscopy is incomplete because pathology in the tracheobronchial tree may remain unnoticed.

Laryngotracheobronchoscopy is performed under general anaesthesia, using material suited for paediatric procedures. It is highly recommended to digitalize data or record all data on videotape which provides an ideal opportunity to discuss the findings with colleagues, to compare data with those recorded during a previous procedure and for teaching purposes. Use of television monitors in the operating room enables the anaesthesiologist, nurses and trainees to follow the procedure and anticipate where necessary.

The procedure usually starts with the child breathing spontaneously under general anaesthesia. A flexible endoscopy can be performed to examine the entire upper airway including the nasal passages, rhinopharynx, oropharynx and larynx. In those cases where no nasal or pharyngeal pathology is suspected, a paediatric laryngoscope is introduced and vocal cord mobility is

assessed. During this part of the procedure, one should check vocal cord and arytenoid mobility; look for laryngomalacia or paradoxical vocal cord movements. Pressure from the laryngoscope blade or the direction of its introduction into the larynx should not disturb laryngeal dynamics and vocal fold mobility.

Direct laryngoscopy not only includes an examination of the larynx but also inspection of the oropharynx, base of tongue and valleculae, the piriform fossae, the postcricoid region, the epiglottis, aryepiglottic folds, arytenoids, false cords and ventricles, the vocal cords, the subglottic region and the trachea. The posterior glottis should be palpated to exclude the presence of a laryngeal cleft.

In patients with suspected tracheomalacia, a topical anaesthetic is applied to the vocal cords to prevent laryngeal spasm and a rigid or flexible endoscope is introduced into the trachea while the child continues to breathe spontaneously. The dosage of the local anaesthetic agent should be appropriate for usage in children because preparations suitable for adults might result in over dosage. Lidocaine (up to 5 mg/kg) in a dilute solution (1%) is appropriate for this purpose.

As the final step in the procedure, a muscle relaxant is given intravenously. After complete relaxation, the mobility of the arytenoids is assessed and the subglottis and trachea are inspected up to the level of the carina or main bronchi.

During any step of the procedure, it is essential to proceed very carefully and gently in order to avoid trauma to the mucosa at the larynx or trachea as this might

cause significant postoperative oedema. Also the suctioning of secretions should be done with the greatest care.

### Causes of stridor

The most common reasons for stridor in infants and children are listed in Table 1. This list is not exhaustive and more details can be found elsewhere.<sup>2,4</sup>

Croup is the most common cause of acute stridor. Laryngomalacia accounts for the most common cause of congenital, chronic stridor. A brief discussion of the most common laryngeal causes of stridor is presented.

### Laryngomalacia

In laryngomalacia the laryngeal tissues show an abnormal flaccidity. Stridor worsens with crying, feeding, excitement or activity and when the child is lying on his back with the head and neck flexed. Laryngomalacia is the most common cause of stridor in infants, usually resolving spontaneously by the age of 12 to 24 months. The infant epiglottis is longer, narrower and more tubular as compared to adults. This Omega-shaped epiglottis is more often found in

children with laryngomalacia but it is neither a constant finding nor a diagnostic feature. Laryngomalacia has been attributed to a delayed neuromuscular control of the larynx or to anatomic abnormalities, but its exact cause is unknown.<sup>6</sup>

### Subglottic haemangioma

Airway haemangiomas are most commonly located in the subglottic region and half of them are lateralized to the left side. They are absent at birth and have a peak presentation at the age of 6 weeks and 85% manifest themselves before age 6 months. A subglottic haemangioma initially presents as intermittent stridor, which might be misdiagnosed as recurrent croup. As the haemangioma increases in size, the stridor becomes more constant and worsens with crying or upper respiratory tract infections. Associated features are dyspnoea, feeding difficulties, growth disturbance and hoarseness. At endoscopy, they present as a pink or bluish, smooth sessile mass arising from the lateral wall of the subglottis and this appearance is pathognomonic.<sup>2</sup> After the first year of life, there is a spontaneous involution and

Table 1

Most common causes of stridor in infants and children

	Nasopharyngeal	Laryngeal	Tracheal
Neonates	Neonatal rhinitis Choanal atresia or stenosis Craniofacial abnormalities Micrognathia	Laryngomalacia Intubation trauma  Reflux laryngitis  Laryngotracheal stenosis Vocal cord palsy	Tracheobronchomalacia   Tracheal Stenosis  Vascular Compression
Children	Allergic Rhinitis Adenoiditis Adenotonsillar hypertrophy Foreign bodies	Croup Haemangioma Papillomatosis  Intubation trauma Vocal cord palsy	Foreign bodies Tracheal Stenosis

symptoms most frequently resolve between 2 and 3 years of age.

#### *Subglottic stenosis*

Subglottic stenosis can be either congenital or acquired and may be classified as either a hard stenosis (due to scar tissue or cartilage) or a soft stenosis.<sup>7</sup>

Congenital subglottic stenosis is caused by histopathological abnormalities at the level of the soft tissues or cartilage. By definition, congenital subglottic stenosis exists when the subglottis has a diameter of less than 4 mm in a full-term newborn or less than 3 mm in a premature infant.<sup>4</sup> Symptoms depend on the severity of the stenosis but most infants become symptomatic within 3 months of birth. Minimal laryngeal oedema due to upper respiratory tract infections or manipulations may cause severe respiratory distress because the cricoid cartilage cannot expand and swelling will therefore extend towards the laryngeal lumen.

Acquired subglottic stenosis might be due to external or internal laryngeal trauma. Trauma was found to be the most frequent cause of acquired subglottic stenosis. Stenosis in these cases can be prevented by early exploration of laryngeal fractures.

About 90% of cases of acquired chronic subglottis stenosis in children are secondary to prolonged endotracheal intubation.<sup>8</sup> Benjamin describes the pathophysiology and classification of acquired subglottic stenosis in detail. Other causes of acquired subglottic stenosis are laryngeal surgery, chronic infection, chronic inflammatory diseases and laryngeal neoplasm.

At endoscopy, the diameter of the stenosis should be measured

and recorded for further reference. This can be done by noting the outside diameter of a bronchoscope or endotracheal tube which can be comfortably passed through the subglottic region or by carefully passing Hegar dilators of varying diameter.

#### *Vocal fold paralysis*

Vocal fold paralysis can be congenital or acquired.

Congenital vocal fold paralysis typically manifests itself within the first month of life.<sup>9</sup> Bilateral paralysis results in stridor, cyanosis and apnoea, unilateral paralysis presents as dysphonia. Both types are associated with feeding difficulties and sternal retraction. Causes of unilateral and bilateral vocal cord paralysis in children include neurological disease (18%), meningomyelocele with Arnold-Chiari malformation and hydrocephalus (14%), birth trauma (19%), other trauma (11%), malignant disease (1%), familial disease (1%). In the remaining 36% of congenital vocal cord paralysis, no cause can be found<sup>10</sup> but there is an association with other congenital abnormalities (pulmonary, cardiovascular or oesophageal).

Acquired vocal cord paralysis in children might be due to surgical trauma (e.g. repair of a tracheo-oesophageal fistula or congenital cardiac defects), traumatic intubation with luxation causes ankylosis of the arytenoid joint and a fixed cord which is not paralysed.

Spontaneous recovery of vocal fold paralysis in children is unpredictable but recovery usually starts within 6 months and is rare after 36 months.<sup>11</sup> Spontaneous recovery is more likely in acquired (64%) than in congenital

(29%) paralysis<sup>12</sup> and more frequent in unilateral (70%) than in bilateral (50%) cases.<sup>9</sup> If vocal fold paralysis is suspected at endoscopy, the movement of the vocal folds should be related to the phase of respiration (inspiration versus expiration). Vocal fold paralysis can be diagnosed if one or both cords are fixed in a median or paramedian position during in- and ex-piration or by observing paradoxical movements: inspiratory adduction of the cords with passive movement to the paramedian position during expiration.

#### *Larynx papillomatosis*

Larynx papillomatosis is caused by human papillomaviruses type 6 and 11. Children most likely become infected in utero or intrapartum and clinical manifestations most frequently occur between 2 and 5 years of age, sometimes earlier in life. Symptoms depend upon the extent of the disease with hoarseness or abnormal cry, dyspnoea and inspiratory stridor. During endoscopy in cases of papillomatosis, seeding virus or inhalation of papilloma downwards into the tracheobronchial tree should be prevented.

#### *Foreign body*

Stridor due to foreign body aspiration most often occurs in children between 1-3 years of age. Boys are more frequently affected than girls. Foreign body aspiration was found to be the 4<sup>th</sup> leading cause of accidental death in children of this age group. Foreign bodies located in the oesophagus might also result in airway compromise because of tracheal compression. The natural history of foreign body aspiration involves 3 stages. In the first stage, there is an episode of choking followed by

coughing, gagging or wheezing eventually associated with hoarseness and / or aphonia, and occasionally complete airway obstruction. Secondly, there is an asymptomatic interval as the protective reflexes fatigue and irritation subsides. This period is often responsible for a delayed diagnosis. Finally, signs and symptoms of complications manifest as cough, haemoptysis, pneumonia, lung abscess, fever or malaise. Inspiratory/expiratory chest films or lateral decubitus films may provide valuable information if a foreign body is suspected. Lateral decubitus films are more useful in young children who cannot cooperate with the timing of the respiratory cycle. Inspiratory hypo-inflation and expiratory hyperinflation are the hallmarks of a bronchial foreign body. It is important to realize that most foreign bodies are radiolucent and that a chest x-ray is commonly normal in the first 24 hours following aspiration. Radiolucent foreign bodies in the proximal trachea may induce subglottic oedema. Extraction of a foreign body is always performed with rigid endoscopes.

#### *Tracheomalacia*

*Tracheomalacia* is a relatively common anomaly of the upper respiratory tract in which there is a functional weakness of the trachea resulting in dynamic collapse of the trachea during breathing.<sup>12</sup> Primary tracheomalacia results from a congenital abnormality of the tracheal cartilage. The tracheal cartilages are shorter than normal, providing less support for the trachea, which therefore assumes a flatter shape in cross section. Because most lesions are intrathoracic, airway collapse typically

occurs during expiration. Extrathoracic lesions in the cervical trachea are rare and lead to collapse during inspiration. Tracheomalacia commonly occurs in association with oesophageal atresia and in these cases is thought to result from the same underlying abnormality in embryological development of the primitive foregut.

Tracheomalacia might also be due to extrinsic tracheal compression by cardiovascular structures or space occupying lesions (tumours, lymph nodes...) or result from prolonged positive pressure ventilation or an infectious/inflammatory process that undermines the intrinsic cartilaginous support of the trachea (secondary forms).

Collapse of the trachea, particularly during expiration manifests as wheezing, a barking cough, frequent respiratory infections and in extreme cases, cyanotic episodes or "dying spells"<sup>12</sup>.

A definitive diagnosis is usually made on tracheoscopy during spontaneous breathing where the trachea is seen to collapse during expiration with antero-posterior flattening of the tracheal walls. Most affected infants improve spontaneously by 6 to 12 months of age as airway calibre increases and cartilage develops. However, some adults remain symptomatic or have exercise intolerance.

### **Therapeutic strategies**

#### *Acute Medical management*

Together with the initial diagnostic evaluation, the physician will assess the degree of respiratory distress through evaluation of alertness of the child, presence of retractions and cyanosis, O<sub>2</sub> saturation. Additional administration

of oxygen does not reduce CO<sub>2</sub> retention.

The prophylactic use of systemic steroids in intubated neonates and in children with stridor seems to have a positive effect.<sup>13</sup> Aerosols with racemic epinephrine together with systemic steroid administration have been reported effective in treatment of children with croup.<sup>14</sup>

Paediatric gastro-oesophageal and laryngotracheal reflux are better recognised over the past years. Although there is general agreement that children with reflux frequently present with respiratory disorders, the effect of reflux treatment on the outcome of surgery for stridor in children is still controversial.<sup>15</sup>

#### *Aetiological treatment*

Specific treatments, such as antibiotics for epiglottitis, intralesional steroid injections for subglottic haemangioma, cidofovir for papillomatosis, endoscopic removal of foreign objects, among other treatments, are beyond the scope of this text. They are obvious and of utmost importance whenever applicable.

#### *Intubation*

Intubation is a sensible first measure in the acute situation when diagnosis and prognosis are still unclear and when humidification aerosols, oxygen or CPAP are not efficient or not applicable. A nasal tube is more secure. The choice of a fitting tube depends on a leak test and the consideration that the smaller tubes cause less mucosal damage. Mucosal damage, especially at laryngeal posterior commissure level, occurs in a high percentage of prolonged intubations<sup>9</sup> but lesions are frequently reversible and the neonates' and

small children's larynges seem to tolerate long term intubation better than an adult larynx. There is general agreement that, especially in neonates, intubation for several weeks is acceptable, although some advocate systematic endoscopic control for laryngeal damage in these situations.<sup>9</sup> The simultaneous presence of a nasogastric tube possibly adds to the development of post-intubation trauma.<sup>16</sup>

At the time of intubation a first inspection for laryngeal pathology and aspiration of tracheobronchial secretions can be done. This however in no way replaces the elective endoscopic examination of larynx and tracheobronchial tree.

In some instances intubation can be impossible and the precipitate nature of paediatric airway obstruction can make a transtracheal airway necessary.

Obviously intubation is not a solution for chronic stridor problems.

#### *Surgical treatment for laryngotracheal stenosis*

After the management of the acute phase and after thorough and systematic endoscopic examination, a number of cases will not be eligible for medical or etiologic treatment. These are grouped with the common denominator "laryngotracheal stenosis". They are caused by tracheomalacia, congenital subglottic stenosis but there are also a rising number of acquired subglottic stenosis cases, which are probably explained by the higher success rate of neonatal intensive care. Surgical management of these children is required but challenging and dependent on close cooperation between paediatrician, anaesthesiologist and otolaryngologist. Through the last 30 years a tendency has developed

towards techniques that avoid tracheostomy.

#### *Laryngotracheoplasty*

Evans was the first to propose laryngotracheoplasty.<sup>17</sup> He used castellated incisions of the cricoid and tracheal anterior wall to enlarge the lumen of the subglottic airway. Stenting and tracheostomy were still needed and especially in the acquired subglottic stenosis cases, where chondritis is present, the technique is difficult to perform.<sup>18</sup>

#### *Anterior cricoid splitting*

Cotton introduced an anterior cricoid split procedure in the 1980's. This is indicated as a first surgical measure in premature neonates where multiple attempts at extubation have failed.<sup>19-21</sup> The procedure is followed by a 1- to 2-week period of sedation, during which the patient is dependent on mechanical ventilation. Cotton later stressed that the intervention should be restricted to neonates or young infants whose pathology is limited to the glottis and subglottis or both, and in whom there is adequate pulmonary reserve.<sup>21,22</sup>

#### *Laryngotracheal reconstruction*

Simultaneously with the growing experience with the anterior cricoid split, variations of this technique were described, which all had in common that the lumen of the subglottic region was further expanded with interposed material in the anterior cricoid incision. In this way the laryngotracheal reconstruction became a widely accepted procedure.<sup>23-25</sup> Costal cartilage is most often used for interposition and appears to be well tolerated in the majority of cases.<sup>22</sup> The procedure involves an anterior midline incision of the

lower third of thyroid cartilage, cricothyroid membrane, cricoid and the first two tracheal rings, with interposition of costal cartilage. It can be extended to a complete laryngofissure with posterior division of the cricoid plate and placement of a posterior graft. It can be performed as a single stage procedure, or in combination with the placement of a stent and with tracheostomy. Children typically stay intubated and sedated for 1 week with a cover of antibiotics, steroids and anti-reflux medication. Extubation is done after downsizing the tube for 24 hours and with routine endoscopic control at extubation and after 1 week.<sup>26</sup> Stents are typically left in place for 4-6 weeks.

#### *Partial cricotracheal resection*

Partial cricotracheal resection<sup>27</sup> involves the resection of a stenosed cricoid and/or tracheal segment with end-to-end re-anastomosis. The technique is an adaptation of what Pearson first described in adults in 1975. When longer segments need to be resected, a tracheal and/or laryngeal release is necessary. The operation can be performed as a single stage procedure without tracheostomy (most often: with resection of the existing tracheostoma) and is followed by an intubated period with similar management as in laryngotracheal reconstruction. The technique is more extensive and carries a risk of recurrent nerve damage, airway dehiscence, re-stenosis and arytenoid prolapse.<sup>28</sup>

#### *Staging of laryngotracheal stenosis*

Cotton *et al*<sup>29</sup>. (1994) proposed a grading system based on the percentage of obstruction of the lumen of the subglottis and tra-

Table 2  
Stenosis Grading system

Classification	Description
Grade I	0-50% obstruction
Grade II	51-70% obstruction
Grade III	71-99% obstruction
Grade IV	No detectable lumen

chea (Table 2), where the remaining lumen is compared to the largest fitting endotracheal tube size. This grading system has since been universally accepted in reports on laryngotracheal stenosis. The grade of stenosis is a relevant predictor of decannulation/extubation rates in large series studied.<sup>30</sup> Of course the localization and extent of stenosis must also be taken into account when decisions are made in the treatment of this problem.

#### Application of different surgical techniques

The anterior cricoid split is indicated for neonatally acquired subglottic stenosis, provided a number of additional criteria have been established. They mainly involve the general respiratory reserve of the child.<sup>31</sup>

A grade I laryngotracheal stenosis usually does not need surgical intervention, or may be eligible for endoscopic CO<sub>2</sub> laser treatment.<sup>31</sup> Grade II stenoses can be treated with laryngotracheal reconstruction with anterior graft placement and without stenting. Grade III stenoses can be treated with laryngotracheal reconstruction with anterior and posterior cricoid splitting and anterior cartilage grafting.<sup>26</sup> The more severe grade III cases or cases where the stenosis is close to the glottis also need posterior grafting and prolonged stenting. For these cases and for grade IV stenoses partial cricotracheal resection is probably a better option.<sup>32,33</sup>

#### Treatment of tracheomalacia

Mild forms of primary tracheomalacia are best treated by non-surgical means. Aortopexy with concomitant intra-operative bronchoscopy is the treatment of choice for distal tracheomalacia that is idiopathic, pulsatile or associated with tracheo-oesophageal or vascular anomalies. Aortopexy attaches the aorta to the sternum, pulling the anterior wall of the trachea forward, thereby preventing its collapse. For proximal or diffuse tracheomalacia, tracheostomy might be necessary to stent the airway.

#### Paediatric tracheostomy technique

Tracheostomy is preferably performed in the operating theatre under general anaesthesia and with a secured airway. Additional airway equipment such as a bronchoscope is essential. Check for the availability of a range of tracheostomy tube sizes and styles. Table 3 gives a list of approximate sizes of tracheostomy tubes for infants and children.<sup>34,35</sup>

With the patient in hyperextension a horizontal incision of the skin is performed, 1.5cm long and 1 finger's breadth above the sternal notch. Subcutaneous fat is removed for good visualisation and prevention of secondary wound infection. Progressive division of the strap muscles is then

performed by splitting the midline raphe. After identification the tracheal fascia is opened. Take care not to dissect the trachea too laterally. If necessary divide the thyroid isthmus by electro-coagulation or suture/ligate it to avoid bleeding. Plan a vertical incision of the trachea through 2 to 3 rings usually within the 2<sup>nd</sup> and 5<sup>th</sup> tracheal rings. Place a nylon traction suture on either side of the planned incision. Incise the trachea. Withdraw the endotracheal tube to a level just above the incision and introduce the tracheostomy tube. Secure it to the skin. Leave the traction sutures in place, without actually pulling on them and secure them to the skin with some adhesive tape. It is useful to mark on the tape "don't remove!" It is also useful to mark the left traction suture with a piece of tape indicating "left" and vice versa for the right one and fixing them at their respective sides to the chest. If in a moment of panic like in early decannulation the traction sutures get mixed up problems can ensue.<sup>2,35-37</sup>

For complications of paediatric tracheostomy please consult the guideline on adult tracheostomy.

In children it is mandatory to perform a standard chest X-ray in the early postoperative phase.<sup>2,36</sup>

In 1992 Fitton published an article focusing on the practical aspects of paediatric tracheostomy care.<sup>38</sup> More care than in adults

Table 3  
Approximate sizes of tracheostomy tubes for infants and children.<sup>34</sup>

Patient Age	Inner diameter (mm)	Length (mm)	
Premature		neonatal	paediatric
< 1000g	2.5	30	not indicated
1000-2500g	3.0	30	39
Neonate – 6 months	3.0-3.5	30-32	39-41
6 months – 1 year	3.5-4.0	32-34	40-41
1-2 years	4.0-4.5-5.0	34-36	41-42-44
Beyond 2 years	(Age in years + 16)/4		

should be taken to humidify the ambient air of the tracheotomised child in order to prevent plugging of the tracheostomy tube. Paediatric tubes don't have a removable inner cannula! Every one to two hours during the first day, administration of some drops of saline followed by aspiration is useful to prevent clogging.

Plan a first change of the tracheostomy tube at the 5<sup>th</sup> postoperative day.<sup>35-38</sup> At that moment the traction sutures are not needed anymore, and following successful cannula change, can be removed.

From then on a weekly change of the cannula with the aim of cleaning can be done safely and can be taught to the parents. Sometimes plugging of the cannula requires it to be changed more often and it can be safely done on a daily basis.

Certainly in children who are prone to infection take care to minimize infections by using aseptic technique in suctioning, tube changing and providing wound care.

Psychological support for both the child and the parents is highly recommended.<sup>38</sup>

### Decannulation

Once the underlying disease is resolved decannulation can be planned.

With some exceptions this is most of the time going to be a rather long period (often until 1 year of age). Always inspect the upper airway for its patency before taking out the tracheostomy tube.<sup>2,39</sup> Remove any granulation tissue; judge on eventual suprastomal collapse.

If the airway is patent and the tracheostomy was present for less than one month, simple removal

of the cannula and a cover dressing on the stoma can be performed.<sup>39</sup> It is still recommended in this case to admit and observe the patient for 24 hrs.

If the airway is patent and the tracheostomy was present for a longer time, the patient should be admitted to the hospital. Downsizing of the cannula to its smallest possible size in daily steps is followed by gradual blocking of the cannula for 12 hours during daytime and for 24 hours the next day and night. Only if the child tolerates this procedure well (normal daily activity) can the cannula be taken out. Further hospital observation for 2 more days is necessary.<sup>2,39</sup>

In case of significant suprastomal collapse surgical decannulation with or without a cartilage graft is performed. The patient is kept intubated for 24 to 48 hours postoperatively.<sup>2</sup>

If decannulation is not tolerated check the upper airway again. Tracheomalacia, suprastomal collapse or vocal cord mobility problems can too easily be missed or not adequately appreciated unless a specific search is carried out.<sup>2,39</sup>

### Levels of evidence

Apart from some publications on the treatment of croup,<sup>14</sup> which are of level II, all scientific data on diagnostic evaluation and treatment of stridor in neonates and infants are classified as level III evidence.

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**CME questions**

1. Which of the following clinical situations is unlikely to cause inspiratory stridor?
  - A – Epiglottitis
  - B – Laryngomalacia
  - C – Aspiration of a pistachio nut
  - D – Croup
  - E – Bilateral choanal atresia
  
2. The most common cause of congenital, chronic stridor is
  - A – Vocal cord paralysis
  - B – Congenital subglottic stenosis
  - C – Laryngomalacia
  - D – Tracheo-oesophageal fistula
  - E – Treacher Collins syndrome
  
3. The diagnostic work-up of chronic, congenital stridor should at least include the following:
  - A – History and clinical examination of nose, pharynx and neck
  - B – History, clinical examination of nose, pharynx and neck, lateral neck X-ray
  - C – History, clinical examination of nose, pharynx and neck, flexible endoscopy at the ward to assess the glottis and supraglottic structures
  - D – History, clinical examination of nose, pharynx and neck, laryngotracheobronchoscopy under general anaesthesia
  - E – History, clinical examination of nose, pharynx and neck, arterial blood gas analysis
  
4. Respiratory distress develops more frequently in neonates and small children compared to adults for the following reasons:
  - A – The neonate/small child larynx has a higher position in the neck
  - B – The neonate is a preferential nose breather
  - C – The neonate's smaller dimensions of trachea and subglottis are more critical
  - D – Neonates and small children have more infections that can cause stridor
  - E – The cartilages are less rigid in the neonate/small child
  
5. When evaluating chronic stridor in a neonate or small child, which of the following is least important?
  - A – Plain radiography of lungs and neck
  - B – Anamnesis related to time and conditions when stridor occurs
  - C – Clinical examination of the child's head and neck.
  - D – Auscultation
  - E – CT or MRI imaging
  
6. Clinical examination of stridor: indicate the false statement:
  - A – Isolated inspiratory stridor is caused by an intrathoracic obstruction
  - B – Isolated expiratory stridor is caused by an intrathoracic obstruction
  - C – Isolated inspiratory stridor is caused by an extrathoracic obstruction
  - D – Combined inspiratory – expiratory stridor is caused by a subglottic obstruction

7. When should diagnostic laryngotracheobronchoscopy be considered? Indicate the false statement.
- A – Every case of stridor lasting longer than 48 hours
  - B – Severe stridor
  - C – Progressive stridor
  - D – Stridor associated with unusual features, such as cyanotic attacks, apnoeic attacks, dysphagia, aspiration, failure to thrive, or a radiological abnormality
  - E – Stridor causing undue parental anxiety
8. Which of the following strategic choices for endoscopy is wrong?
- A – Flexible endoscopy is not sufficient for neonatal stridor
  - B – All endoscopic procedures should be kept as short as possible and should be stopped as soon as a single cause for stridor is found
  - C – Laryngotracheobronchoscopy should not be planned when a bed in the ICU is not available
  - D – It is best to record or digitalize the endoscopic procedure
  - E – The procedure should be done by a team involving anaesthetist, ENT specialist and nursing staff who are experienced with the procedure
9. Foreign body aspiration: the foreign body causing respiratory distress is most of the time found to be located at:
- A – The supraglottic level
  - B – The carina
  - C – The oesophagus
  - D – The right main bronchus
  - E – The left main bronchus
10. In chronic laryngotracheal stenosis where an aetiological treatment is not possible, which of the following techniques are nowadays rarely performed?
- A – Tracheostomy
  - B – Anterior cricoid splitting
  - C – Evans castellation laryngotracheoplasty
  - D – Partial cricotracheal resection
  - E – Laryngotracheal reconstruction
11. In neonatal and paediatric tracheostomy, the first changing of the cannula is recommended to be performed:
- A – The day following the surgical procedure
  - B – At 48 hours
  - C – At 5 days
  - D – At 2 weeks
12. In neonatal and paediatric tracheostomy, subcutaneous fat is removed as a first step of the procedure (give the wrong statement)
- A – To reduce secondary wound infection
  - B – To facilitate spontaneous closure of the wound following decannulation
  - C – To improve visualisation and control of the surgical procedure
  - D – To facilitate cannula replacement following accidental decannulation during the first 24 hours.

13. Which statement concerning paediatric tracheostomy is correct?
- A – Traction sutures on both sides of the tracheal incision are useless in case of accidental early decannulation.
  - B – The level of incision of the trachea is not important.
  - C – The incision and further manipulations to the trachea should be performed with great care.
  - D – As in paediatric tracheostomy no additional airway equipment is needed, the procedure can easily be performed as a bedside procedure in the intensive care unit.
14. When it is decided to decannulate a patient and the procedure is not well tolerated by the patient, you should specifically look for:
- A – Tracheomalacia, suprastomal collapse, vocal cord immobility.
  - B – Tracheomalacia, suprastomal collapse, adeno/tonsillar hypertrophy.
  - C – Suprastomal collapse, adeno/tonsillar hypertrophy, asthma.
  - D – Suprastomal collapse, vocal cord immobility, nasal obstruction.

**Answers:** 1C; 2C; 3D; 4A; 5E; 6A; 7A; 8B; 9D; 10C; 11C; 12B; 13C; 14A