# Laryngotracheal Clefts

# 10

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## 10.1 Introduction

Laryngotracheal clefts (LTC) constitute a rare cause of congenital upper airway malformation. First described in a doctoral dissertation on unavoidable causes of neonatal death in the eighteenth century by Richter, LTC incidence is estimated to be around 1 in 10,000–20,000 live births, accounting for 0.2–1.5 % of all congenital laryngeal anomalies [1, 2].

The presenting signs and symptoms as well as the age of diagnosis largely depend on the type of cleft (Table 10.1) as well as the eventual presence of associated malformations. Moreover, it has been previously pointed out that the actual incidence of LTC may be highly underestimated [3]. Potential reasons for this include asymptomatic or pauci-symptomatic disease especially in type I clefts, difficulties in endoscopic diagnosis mostly related to physician's unawareness, and immediate mortality in high-grade clefts without postmortem diagnosis. Furthermore, since LTC often occurs in a context of polymalformative disorders, endoscopic diagnosis may not be the priority in many cases [4, 5].

From an embryologic perspective, the larynx develops from endodermic tissues (which in turn derive from the primitive gut) as well as mesenchymal tissues derived from the IV–VI branchial arches. Toward the fourth week of development begins the midline fusion which is meant to lead to the separation of the digestive (esophageal) and respiratory (tracheal) axes. This fusion process takes place in the caudal to cranial direction [3]. The esophagus elongates and reaches its final relative length toward the seventh week. LTCs are pathological clefts thought to be the result of a closure failure between the tracheal and esophageal axes during embryogenesis, but despite the explanatory power of this theory, it is important to point out that it has been challenged as it does not allow explaining the origin of other pathological entities such as isolated tracheoesophageal fistulae.

## 10.2 Diagnostic Aspects

### 10.2.1 Grading

Several grading classifications for LTCs have been proposed, primarily based on the craniocaudal extent of the cleft. Such classifications are mainly descriptive, help in therapeutic choices, and have to some degree prognostic value. The

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**Table 10.1** Classification of laryngotracheal clefts

| Benjamin and Inglis (1989) | | Sandu and Monnier (2006) | |
|---|---|---|---|
| Type | Description | Type | Description |
| I | Supraglottic cleft | 0 | Submucosal cleft |
| | | I | Interarytenoid cleft with the absence of the interarytenoid muscle |
| II | Cleft with partial cricoid involvement | II | Posterior cleft extending partially through the cricoid plate |
| III | Cleft beyond the cricoid cartilage with involvement of the cervical trachea | IIIa | Posterior cleft extending down to the inferior border of the cricoid plate |
| | | IIIb | Posterior cleft extending into the cervical trachea, but not beyond the sternal notch |
| IV | Involvement of thoracic trachea | IVa | Laryngotracheal cleft extending into the intrathoracic trachea to the carina |
| | | IVb | Intrathoracic extension of the cleft involving one main bronchus |

most widely used classification is the one proposed by Benjamin and Inglis in 1989 [3]. Benjamin's classification was modified and updated by Sandu and Monnier [2] in 2006, in order to introduce parameters with therapeutic and prognostic implications previously overlooked (Table 10.1).

Unlike previous classifications, Sandu and Monnier introduce type 0 clefts ("occult" or more accurately submucosal), which appear as a consequence of a posteriorly defective cricoid cartilage accompanied by the absence of transverse interarytenoid muscles [6]. Type I clefts are manifest interarytenoid clefts with the absence of interarytenoid muscles which do not involve at all the cricoid cartilage. Type II clefts extend partially through the cricoid cartilage. By definition, type III and type IV clefts extend above and below the thoracic inlet, respectively. Sandu and Monnier's classification pays particular attention to two specific features in these severe forms of disease: (1) whether the cricoid plate is partially or completely involved by the LTC in type III clefts and (2) whether the cleft is supra- or infracarinal in type IV LTCs (Fig. 10.1).

### 10.2.2 Clinical Presentation

The classical diagnostic triad of LTC consists of husky cry, aspiration pneumonia, and swallowing disorders in a newborn child with associated congenital anomalies [7]. While such scenario should immediately prompt active search for LTC, disease spectrum can often complicate and delay LTC diagnosis. Indeed, LTCs are congenital laryngotracheal disorders which can present as submucosal closure defect in the mildest cases versus complete laryngotracheoesophageal clefts (LTEC) in some other severe cases. Breathing difficulties are typically due to prolapsing of retroarytenoid mucosa caused by the absence of the aerodigestive party wall and obstruction of the posterior respiratory glottis (Fig. 10.2). Therefore, presenting signs and symptoms related to LTC may vary depending on disease severity [7, 8].

LTCs without involvement of the posterior cricoid plate (type 0 and I) may be asymptomatic or in the case of type I clefts present with mild occasional episodes of aspiration, hoarse cry, aspiration, cough, or in some cases dyspnea or cyanosis during feeding [8–10]. Due to their rarity, diagnosis of type 0 and I clefts requires a high-suspicion index.

Regarding clefts with cricoid (with or without tracheal involvement), the outstanding issue is severe aspiration and subsequent lower pulmonary tract infection, as well as respiratory problems in some cases [9, 11]. Finally, type IV LTCs have a dim prognosis due to respiratory distress, poor airway tone, and difficulties to maintain a patent airway even when using invasive procedures [11–13]. Table 10.2 summarizes the clinical presentation of LTCs [14].

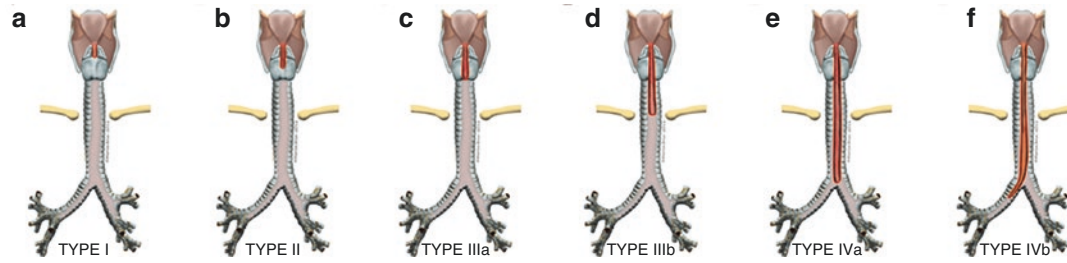


**Fig. 10.1** LTC classification according to Sandu and Monnier

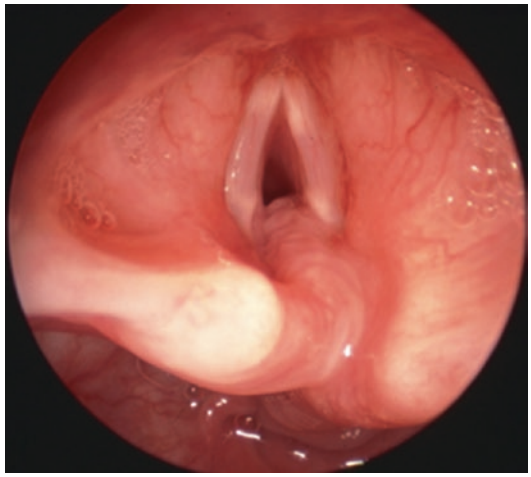

**Fig. 10.2** Airway obstruction secondary to arytenoid prolapse and mucosal invagination into the trachea

### 10.2.3 Radiologic Studies

While the mainstay of LTC diagnosis is endoscopic (discussed below), some imaging exams may be contributive. Plain chest X-ray is often performed in children with persistent respiratory symptoms, whether or not such symptoms are secondary to an underlying LTC. Patients with LTC may present aspiration pneumonia or peribronchial cuffing, but chest X-ray is normal in up to 25% of type I and around 10% of type II LTCs [15].

Modified barium swallow (MBS) performed under the supervision of a speech and swallowing pathologist, testing diverse food consistencies, may help identifying patients with discoordinated swallow or aspiration. It has been previously pointed out that aspiration in an otherwise healthy child most often correlates with an underlying anatomic abnormality [16].

**Table 10.2** Clinical presentation of LTCs

| Feature | Incidence (%) |
|---|---|
| Aspiration | 53–80 |
| Chronic cough | 27–35 |
| Stridor | 10–60 |
| Weak voice/cry | 16 |
| Salivary stasis in the pharynx | 10–23 |
| Aspiration pneumonia | 16–54 |
| Neonatal asphyxiation | 33 |

It is important to keep in mind that children with an undiagnosed LTC presenting with swallowing disorders may have undergone several MBSs in the past, and the cumulative radiation dose can be substantial.

### 10.2.4 Functional Endoscopic Evaluation of Swallowing (FEES)

FEES provides an extremely accurate dynamic assessment of the laryngeal function during swallowing. FEES is usually possible in children older than 1 year but is otherwise difficult to perform in younger children. During FEES, several food consistencies should be tested. Hypopharyngeal stasis and/or laryngeal penetration may be visualized in cases of early-stage LTCs [17].

Like MBS, FEES can be normal in children with grade I cleft with only intermittent aspiration.

### 10.2.5 Endoscopic Diagnosis

Definitive LTC diagnosis relies on endoscopic examination. Due to the high incidence of associated malformations, complete endoscopy of the upper aerodigestive tract is mandatory, and should include:

- Transnasal fiberoptic laryngoscopy, performed with the patient under spontaneous breathing. The entire airway is examined dynamically from the nostrils to the bronchi. The glottis ought to be sprayed with local anesthesia in order to examine the lower airway. In case the child bears a tracheotomy, the canula is removed intermittently during ventilation to facilitate comprehensive dynamic airway examination. Typically, LTC is associated with varying degrees of tracheobronchomalacia due to the absence of the trachealis muscle.
- Direct laryngoscopy and esophagoscopy:
  - Laryngoscopy must be performed using an anesthesia Macintosh spatula as well as 0°, 30°, and 70° telescopes, prior to airway intubation.
  - Suspension laryngoscopy in order to evaluate the morphology of the glottis and assess the posterior commissure using a Lindholm vocal fold retractor allows the diagnosis of a submucosal or small type I cleft. Palpation of the interarytenoid region using a blunt probe is the hallmark to the diagnosis of an LTC (Fig. 10.3). The probe dips down into the posterior commissure in the presence of a cleft. The lower limit of the probe and simultaneous visualization by a telescope gives an idea regarding the cleft depth and thus its type.
  - The esophagus and stomach are equally examined, paying particular attention to the presence of an eventual tracheoesophageal fistula [4, 18], GER, and microgastria (may be associated in type IV clefts).

Associated airway findings in LTC include:

- Narrowed interarytenoid distance with prolapsing retroarytenoid mucosa that blocks the posterior respiratory glottis

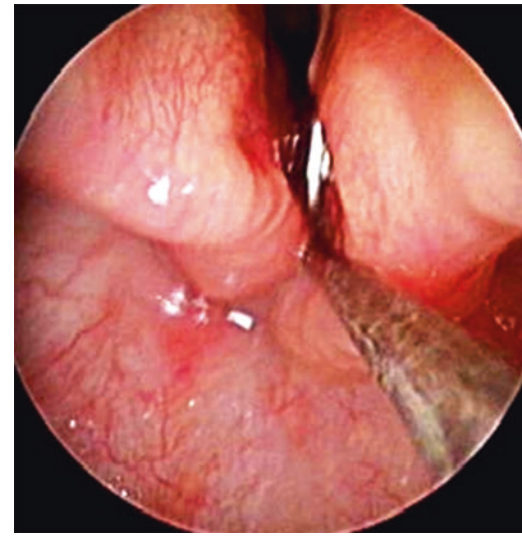

**Fig. 10.3** The use of a blunt-angled probe to diagnose an LTC

- Paramedian position of the vocal folds
- Interarytenoid erythema
- Mucosal cobblestoning (secondary to gastroesophageal reflux)

## 10.3 Management

The therapeutic approach to the child with LTC largely depends on the extent of the cleft, severity of clinical manifestations, and associated or underlying disorders. All decisions should be taken in consultation with the parents and in the context of a multidisciplinary team, especially in children with severe comorbidities (such as cardiac, respiratory, etc.). In symptomatic patients, within the period between diagnosis and eventual surgical management, two issues stand out: respiratory and swallowing disorders.

Type 0 and I LTCs without respiratory symptoms may benefit of regular follow-up and eventually prophylactic-intermittent antibiotherapy in order to prevent pulmonary complications of recurrent pneumonia. Most of these patients are likely to require surgery at some point in their lives for chronic intractable aspiration. Type II clefts present with feeding difficulties and quasi-systematically require surgical management.

Type III and type IV LTCs may quickly degrade from a respiratory point of view, imposing oxygen therapy and pharyngeal aspiration tubes. The degree of invasiveness of oxygenotherapy may vary from nasal administration of oxygen to invasive ventilation (i.e., endotracheal tubes and even tracheostomy).

With respect to swallowing disorders, measures to avoid tracheobronchial aspiration and GERD must be considered a priority. Tracheobronchial aspiration in advanced-type LTCs represents a serious and immediate threat to the vital prognosis and consequently requires aggressive management approaches. More specifically, LTCs of type I and II require medical anti-GERD treatment and thickened food. More severe cases may impose suspension of oral feeding and enteral administration through a nasal or a gastrostomy tube, or even parenteral nutrition in case of longer clefts.

In summary, even though the management of LTCs is far from being consensual, there is some agreement that children with type I clefts may benefit from a trial of medical management and regular follow-up, while longer clefts (with cricoid involvement) require early surgical approaches to avoid the pulmonary complications of aspiration [7, 15, 17, 19]. Furthermore, a minimally invasive endoscopic approach has become standard for the management of types I, II, and IIIa (and even selected IIIb) LTCs, while major types IIIb and IV clefts most often require open approaches [2, 15, 20–22]. Open approaches are equally indicated as salvage for failed attempts of endoscopic repair.

### 10.3.1 Endoscopic Repair

Since the late 1970s, various endoscopic approaches have been successfully used primarily for types I and II clefts [25]. Injection laryngoplasty using different products (Gelfoam, bioplastic) has been reported [26, 27]. Endoscopic repair has progressively become the standard of care for types I and II clefts that remain symptomatic despite adequate medical treatment after an observation period of 4–6 months [14, 16, 17].

Sandu and Monnier reported a small series of patients with types IIIa and IIIb LTCs managed endoscopically [2]. In their reported experience carbon dioxide laser is used in ultrapulse mode to incise the cleft from caudal to cranial direction – cleft apex up to the cuneiform cartilages. The $CO_2$ laser is a precise cutting tool giving a bloodless field and causes no mucosal charring. Two layers of mucosae are created: laryngotracheal and pharyngoesophageal. Starting caudally, a set of inverted Vicryl 5.0 sutures are placed on the tracheal aspect of the cleft and the knots tied facing the pharyngoesophageal side. The second mucosal layer is sutured in similar fashion in distal to proximal direction with knots facing the esophagus while gradually withdrawing the suspension laryngoscope (Fig. 10.4). The procedure is performed under spontaneous respiration and without endotracheal intubation. At the end of the procedure, it is important to maintain an adequate posterior commissure and avoid posterior glottic stenosis (Fig. 10.5). Special endoscopic suturing instruments are a must and their role needs emphasis. The details of such instruments are described in related articles [2].

The advantages of the endoscopic repair are:

- The surgeon is axial to the larynx and gets the best view of the cleft. This is important so as to achieve a meticulous closure of the cleft and avoid excessive crowding of mucosa into the airway lumen and thus its obstruction.
- Maintains stability of the laryngotracheal framework.

### 10.3.2 Open Surgical Approaches

Several open surgical techniques for the repair of LTCs exist, depending on their extent. All approaches require a layer-by-layer closure of the cleft (esophageal and tracheal). The use of interposition material to reinforce the closure has been suggested by some authors (tibial periosteum, auricular cartilage, sternocleidomastoid muscle flap, fascia temporalis, or costal cartilage) [20, 22–24].

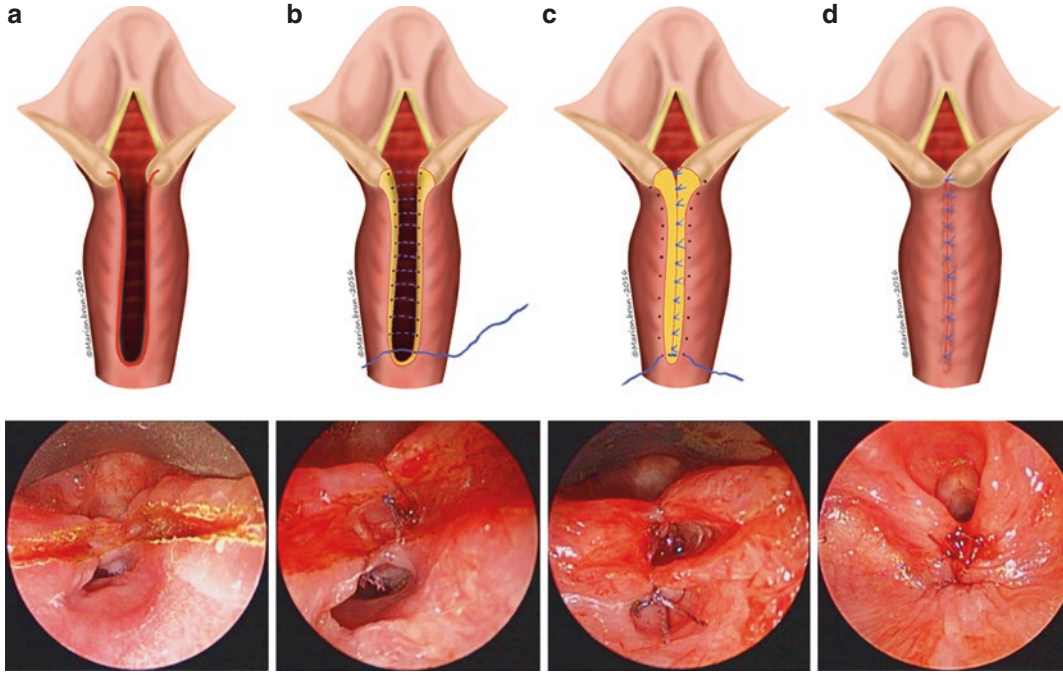


**Fig. 10.4** Endoscopic closure of LTC: (**a**) Type IIIb LTC and the use of $CO_2$ laser to create two mucosal layers, laryngotracheal and pharyngoesophageal. (**b**) Vicryl 5.0 is used to suture the inner laryngotracheal layer from caudal to cranial, with the knots tied toward the esophageal side. (**c**) Caudo-cranial suturing of the pharyngoesophageal layer. (**d**) End of the two-layer LTC closure. Note that in the end, we should have an adequate posterior commissure and avoid a posterior glottic stenosis

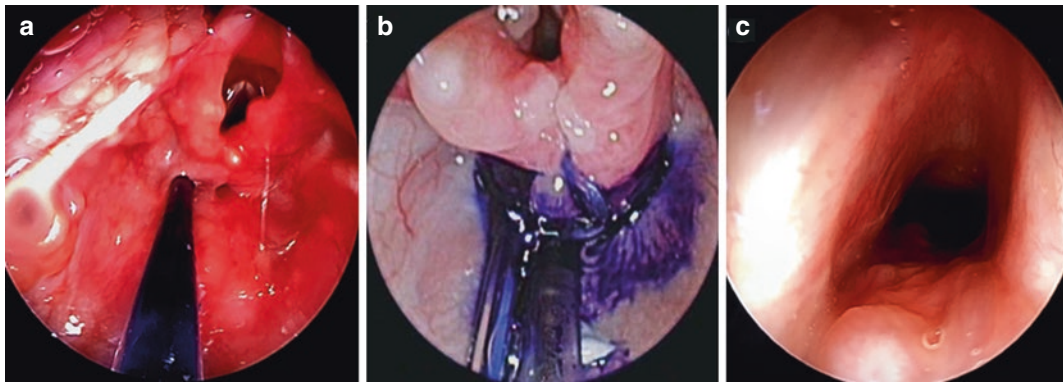


**Fig. 10.5** Endoscopic closure of LTC – post-LTC repair endoscopy: (**a**) Placement of naso-esophageal suction catheter and injection of dilute methylene blue. (**b**) Avoid excessive spillage of the colored solution and airway soiling. (**c**) Simultaneous endoscopic visualization of the larynx and trachea shows complete healing of the LTC and no residual fistula

Cervical approaches differ in cases of extra- and intrathoracic LTCs and can be summarized as follows:

1. Extrathoracic LTEC

   The surgery begins with a nasotracheal intubation using a soft Portex blue line ET tube. Surgery begins with a horizontal neck incision and separating the pre-laryngeal strap muscles in the midline. The thyroid isthmus is divided and retracted. An extended laryngotracheofissure is performed dividing the anterior commissure exactly in the midline (Fig. 10.6a, b). The posteriorly placed laryngeal cleft is identified. The edges of the cleft are incised to create two layers: pharyngoesophageal and laryngotracheal depending on the cleft extension (Fig. 10.6c). These layers are then sutured independently in between tissue interpositions, using perichondrium, tibial periosteum, or rib cartilage graft that can be sutured to the splayed posterior cricoid plate similar to a posterior cartilage expansion graft (Fig. 10.6d). To calibrate the endolaryngeal reconstruction, Monnier's LT mold stent can be used temporarily.
2. Intrathoracic LTEC

   The repair is done under cardiopulmonary bypass or extracorporeal membrane oxygenation (ECMO) [7]. The long cleft is approached by a longitudinal incision along the right tracheoesophageal groove from the origin of the right main bronchus up to the level of the cricoid cartilage. A long laryngotracheofissure causes severe airway framework instability and is hence avoided. The right lateral esophageal wall is dissected in the groove, and the common tracheoesophageal space is entered. The objective of dissecting and mobilizing the right esophageal wall is to use this mucosa to reconstruct the

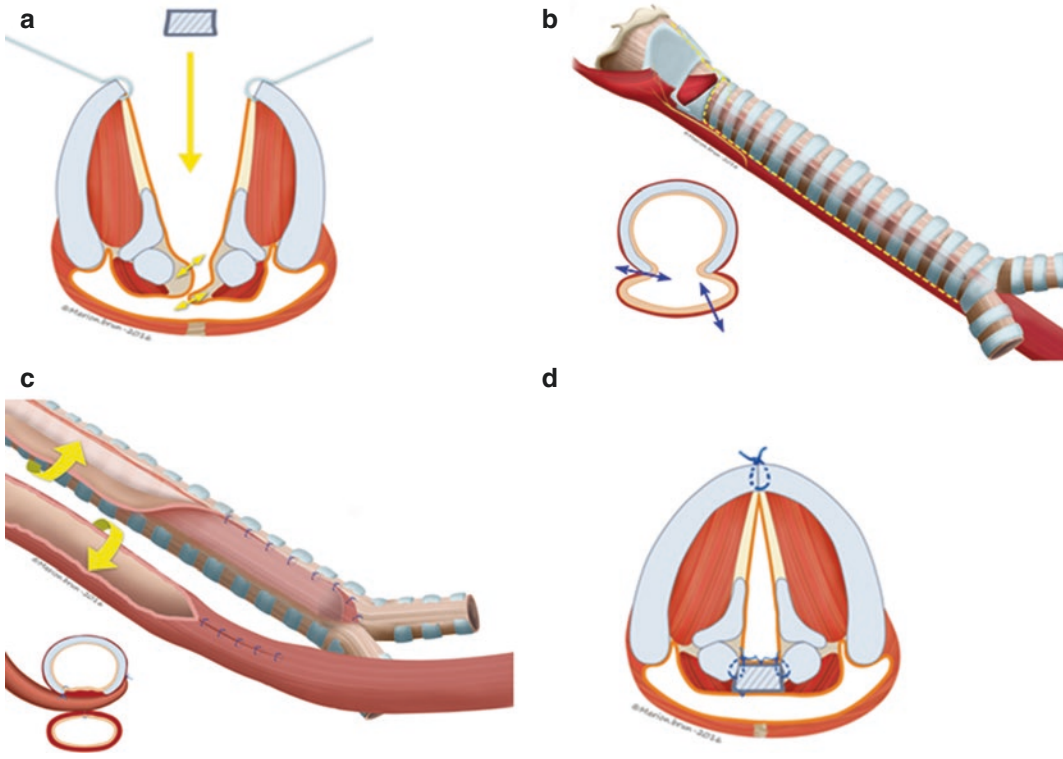


**Fig. 10.6** Open repair of LTC. (**a**) Anterior extended laryngofissure. (**b**) Dissection in the right tracheoesophageal groove and (**c**) esophageal mucosa flipped over to the left that is used to reconstruct the posterior trachea. (**d**) A posterior costal cartilage graft is sutured into the cricoid defect. In addition, a small Monnier's LT mold is inserted for temporary airway calibration

posterior tracheal wall after identifying and incising the left tracheoesophageal cleft mucosa. The rest of the esophageal mucosa is rotated and sutured on to the left side. The trachea and esophagus are sutured longitudinally up to the cricoid. The laryngeal part of the cleft is then approached by laryngofissure. The posterior laryngeal cleft is identified and sutured as described above along with tissue interposition. A small LT mold (avoiding excess contact pressure on the suture line) is inserted to calibrate the airway and the laryngofissure closed. The LT mold is removed endoscopically at a later date.

Ideally and *if possible*, a tracheostomy is to be avoided in LTC management as it destabilizes the tracheal framework, erodes the posterior suture line of repair, and can potentially lead to severe tracheomalacia – albeit to say, it has to be done as per the patient requirements and for oxygenotherapy [19, 22]. In long LTC repairs, a well-fixed nasotracheal tube is preferred during the immediate postoperative period.

### 10.3.3 Transcervical Approach Using Cricotracheal Separation

Propst and Rutter [29] described a technique to repair a type IV LTC. The long cleft is approached transcervically, without sternum split and without deploying ECMO or a cardiopulmonary bypass (Fig. 10.7). The surgery is performed under spontaneous total intravenous anesthesia. An angioplasty balloon catheter is passed into the stomach and inflated, so as to avoid gastric insufflation by anesthesia gases and oxygen during the entire intervention. The surgery begins as described earlier up to exposing the laryngotracheal framework. The cricoid cartilage and the trachea are transected at the first tracheal ring. The dissection is continued posteriorly to identify the long LTC. Recurrent laryngeal nerves are not identified. The trachea is then peeled off from the esophagus up to the lower end of the cleft. A complete laryngofissure is performed, and the splayed cricoid is reconstructed using a rib cartilage. Proximal pharyngoesophageal and laryngotracheal mucosae are flipped across each other to close above the posterior cricoid and reaching the interarytenoid region. The front of the esophagus is sutured in the caudal to cranial direction. The excess of esophageal mucosa is used to reconstruct the posterior tracheal wall. The back of the trachea is then sutured in a similar fashion up to the cricoid cartilage. At this stage, the trachea is reconnected to the cricoid. The authors note the following advantages with this technique – improved visibility, access, airway stability, and coverage of the anastomosis with interpositional sternal periosteum – permitting a three-layer closure.

## 10.4 Associated Disorders

As previously mentioned, LTCs are often a manifestation of a larger spectrum of developmental disease and therefore are often embedded in a context of associated congenital malformations (Table 10.3). The most common comorbidities are esophageal atresia, considered to occur in 20–37 % of cases of LTCs, while various midline malformations including craniofacial and heart malformations would occur in around 10 % and 16–33 % of the cases, respectively [3, 19]. The co-occurrence of LTC and tracheoesophageal fistula is estimated to be between 10 and 15 % [28].

As mentioned above, complete digestive and respiratory examination is essential given the number of cases with associated malformations. LTCs can equally occur in a number of syndromic contexts, especially Opitz-Frias syndrome, Pallister-Hall syndrome, DiGeorge syndrome, CHARGE syndrome, and VACTERL association (Table 10.4).

Genetic counseling is a must in such cases.

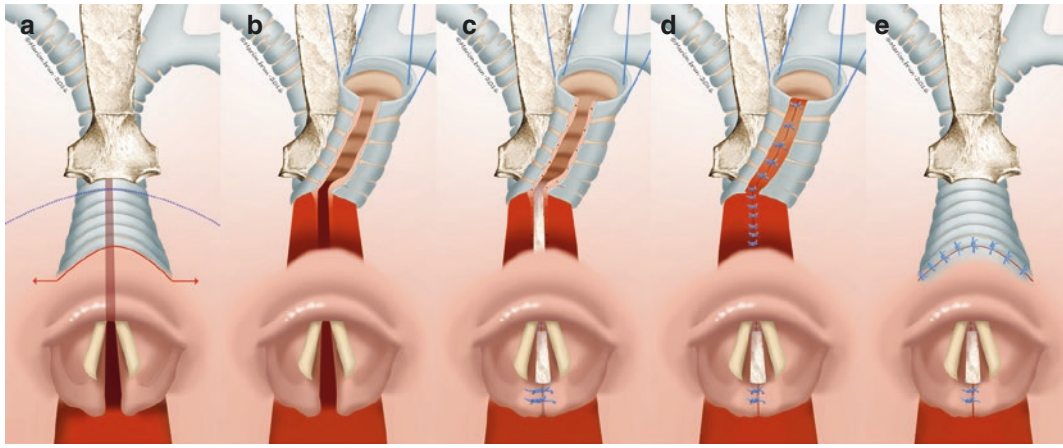


**Fig. 10.7** Open repair (transcervical approach with cricotracheal separation): (**a**) Trachea transected from the cricoid at the first tracheal ring. (**b**) Trachea peeled off from the esophagus. (**c**) Complete laryngofissure and closure of the pharyngeal and laryngeal mucosae. An interpositional costal cartilage graft is used to reconstruct the splayed cricoid defect. (**d**) Repair of the back of the trachea and front of the esophagus. (**e**) Trachea is reconnected with the cricoid

**Table 10.3** Most common associated malformations

| System | Malformations |
|---|---|
| Head and face | Cleft lip and palate, micrognathia, glossoptosis, microtia, hypertelorism, and choanal atresia |
| Respiratory | Tracheoesophageal fistula, tracheomalacia, hyaline membrane disease, irregularities in size and shape of lower airways |
| Gastrointestinal | Esophageal atresia, duodenal atresia, imperforated anus, intestinal malrotation |
| Cardiac | Aortic coarctation, great vessel transposition, double outlet right ventricle, patent ductus arteriosus, and septal defects |
| Genitourinary | Renal malformations, hypospadias, and inguinal hernia |

## 10.5 Prognosis

The outcome of LTCs depends on several factors, mainly cleft extent and associated comorbidities [22]. Early diagnosis and active prevention of lower airway infection and GER equally have an impact on disease progression. Longer clefts are associated with syndromic anomalies and comorbidities and carry a poor prognosis. The mortality rate of LTCs was around 50 % in the 1980s (43 % for types I and II, 42 % for type III, and 93 % for type IV) – but more recent series report rates of 6–25 % [19].

**Table 10.4** Associated syndromes

| Disease | OMIM ID | Chromosomal region | Inheritance mode | Incidence | Phenotypical manifestations |
|---|---|---|---|---|---|
| Opitz-Frias syndrome | 145410 | 22q11.23 | AD<br>X linked | 1:4000–1:10,000 | Hypertelorism, telecanthus, cleft lip, palate, and uvula, hypospadias (male) and splayed labia majora (females), mental retardation, developmental delay, and congenital heart diseases |
| Pallister-Hall syndrome | 146510 | 7p14.1 | AD | Unknown | Extremity anomalies (polydactyly, syndactyly), hypothalamic hamartoma, bifid epiglottis, imperforated anus, and hydronephrosis/hydroureter |
| DiGeorge syndrome | 188400 | 22q11.21 | AD | 1:2000–1:7000 | Variable developmental delay, obesity, ear anomalies, eye anomalies, cleft palate/uvula, tetralogy of Fallot or multiple other heart malformations, endocrine and metabolic disorders<br>*Mnemonic*: CATCH22 – *C*ardiac abnormality, *A*bnormal facies, *T*hymic aplasia, *C*left palate or VPI, *H*ypocalcemia/*h*ypoparathyroidism, 22q11.2 |
| CHARGE syndrome[a] | 214800 | 7q21.11<br>8q12.2 | AD | 1:8500–12,000 | Variable phenotype<br>Mnemonic: CHARGE – *C*oloboma, *H*eart defect, *A*tresia choanae, *R*etarded growth and development, *E*xtremity abnormalities |
| VACTERL association | 192350 | | | 1.6 cases/10,000 live births | Variable phenotype<br>Mnemonic: VACTERL – *V*ertebral anomalies, *A*nal atresia, *C*ardiovascular anomalies, *T*racheoesophageal fistula, *E*sophageal atresia, *R*enal anomalies, preaxial *L*imb anomalies |

*Source*: Online Mendelian Inheritance in Man (www.omim.org)
*Abbreviations*: *AD* autosomal dominant
[a]Two different genetic lesions have been identified, and CHARGE syndrome overlaps with Kallmann syndrome

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