

Review

Advances in the Treatment of Laryngotracheal Stenosis

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Abstract: Laryngotracheal stenosis (LTS) is a refractory airway disorder characterised by pathological scar formation and airway luminal narrowing, which severely impairs respiratory function and quality of life. This review systematically summarises the pathological mechanisms, anatomical characteristics and latest therapeutic progress of LTS, including iatrogenic and idiopathic subtypes. The core pathogenesis of LTS lies in disordered wound healing, persistent chronic inflammation and excessive extracellular matrix deposition, driven by key fibrotic mediators such as transforming growth factor- β 1. Current interventions cover endoscopic techniques such as laser and cold-knife resection, balloon dilation, as well as open procedures including tracheal resection and anastomosis, and graft reconstruction. For lesions beyond the safe limits of conventional resection (> 6 cm), graft-based reconstruction using autologous tissues, allografts, or tissue-engineered constructs becomes necessary. Each therapy has distinct indications, curative effects and limitations. Multimodal therapy, individualised regimens and multidisciplinary collaboration have become the core principles to improve clinical outcomes. Adjunctive pharmacologic therapies, including topical mitomycin C and corticosteroids, are increasingly integrated to modulate wound healing and reduce recurrence risk. For refractory long-segment stenosis and recurrent cases, biomaterials, tissue engineering and precision medicine represent the key research directions for future LTS treatment.

Keywords: Laryngotracheal Stenosis; Subglottic Stenosis; Endoscopic Surgery; Balloon Dilation; Tracheal Resection; Graft Reconstruction; Multidisciplinary Management

1. Introduction

Laryngotracheal stenosis (LTS) is a narrowing of the larynx and trachea that can be fatal. It can be caused by iatrogenic injury, such as a prolonged endotracheal tube, or by idiopathic and systemic inflammatory diseases. Although good diagnostic tools and treatments have been developed recently, recurrence and complex anatomy remain problems for LTS to this day; thus, it still causes significant harm to patients' lives. Traditional management went from just mechanical stretching to target tissue removal and rebuilding. Still, the best way would need knowledge of both the molecular pathophysiology as well as the surgical geometry. This review seeks to gather evidence around the mechanism, anatomical basis and modern treatments of LTS so as to develop an all-in-one personalized & multi-disciplinary form of treatment. It also discusses some emerging technology and the future direction of study.

2. Mechanism

In other words, LTS is a spectrum of diseases caused by abnormal scar formation within the upper airway, resulting in a narrowed lumen. It may involve the glottis, supraglottis, subglottis and trachea, leading to severe respiratory dysfunction and abnormal voice [1]. By cause, LTS is mainly divided into iatrogenic LTS (iLTS) and idiopathic subglottic stenosis (iSGS). Although these two entities are very different in terms of their predisposing factors and clinical presentations, it is currently held that they have a common final pathway: an abnormal, uncontrolled and exaggerated wound healing process results in the formation of pathological scar tissue inside the airway, leading to narrowing [2].

2.1. Imbalance of Physiological and Pathological Wound Healing

Classically speaking, physiological wound healing in the airway is a very specific and controllable series of events like hematological/inflammatory phase → proliferation phase → remodeling/maturity phase [3,4], but its goal is to repair injury, create new barriers & recreate tissue structure with minimal scarring, if any at all. However, in the pathogenesis of LTS, this orderly progress is out of order, transforming into a fibrotic process with persistent chronic inflammation, abnormal activation of fibroblasts and pathological over-deposition of the extracellular matrix (ECM). In the clinic, ILTS patients' symptoms often appear late, 4–6 weeks after an acute injury (like extubation); this corresponds exactly to when a mature scar would form, thus directly proving that it is a disease of dysregulated wound healing (**Figure 1**) [2].

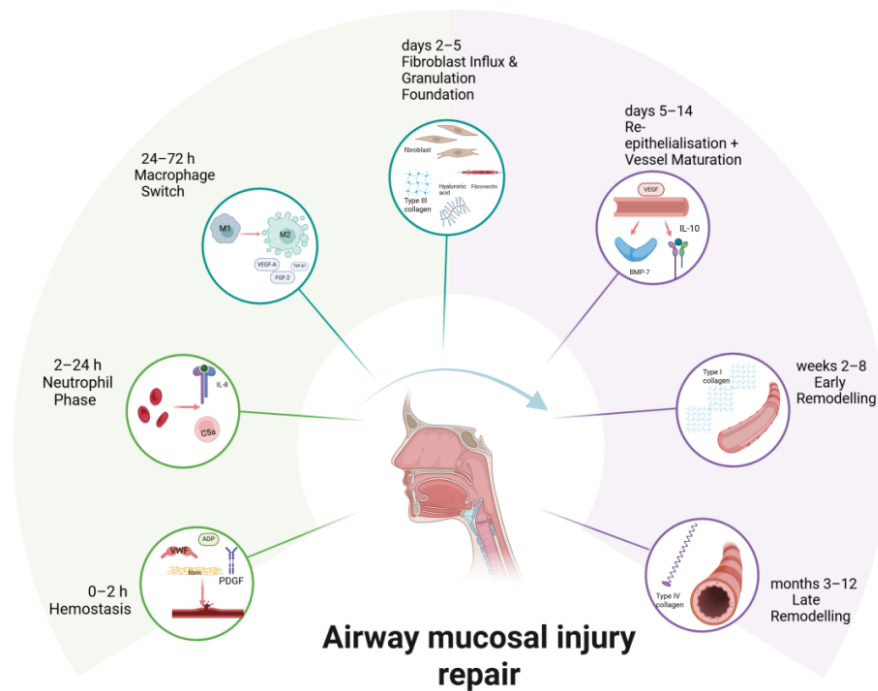


Figure 1. Pathophysiological timeline of laryngotracheal stenosis (LTS).

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Time 0–2 h: Delayed hemostasis (anticoagulants, uremia) → expanding hematoma → tension scar & cartilage ischemia

Time 2–24 h: Excessive or lingering neutrophils (hyperglycaemia, smoking) → autolysis of viable matrix → ulcer deepening

Time 24–72 h: Persistent M1 (obesity, OSA, low-IL-10 genotype) → non-stop inflammation → later myofibroblast drive

Time days 2–5: Ongoing hypoxia (anaemia, poor perfusion) → HIF-1 α sustained → fibroblast → myofibroblast transition & early contraction

Time days 5–14: Epithelial bridge fails (deep ulcer, infection, acid-pepsin) → persistent granulation → myofibroblast unchecked → collagen ↑↑

Time weeks 2–8: MMP/TIMP imbalance (high TGF- β_1 , low MMP-1 SNP) → over-cross-linking → rigid scar ring

Time months 3–12: Myofibroblast apoptosis-resistant (IL-4/IL-17 high, oestrogen dysregulation) → continuous contraction → posterior glottis/cricoid fixation → clinical stenosis

iLTS mostly happens after a long time of endotracheal intubation, and its histopathological change can clearly show the process of abnormal repair. The injury starts with mucosal ulceration due to pressure from the tracheal tube, and in severe cases can extend deep to the cartilage [5]; In the following healing process, when the injury reaches a full thickness, granulation tissue rich in capillaries, inflammatory cells, and fibroblasts forms [5,6]; If there is dysregulation, this hyperplastic granulation tissue cannot be fully covered by epithelium and finally turns into dense, poorly vascularized fibrous scar tissue. Within this scar tissue, normal glandular structure is replaced by cystic dilation, and even ectopic bone or islands of cartilage may appear [5], usually taking about 6–8 weeks for the maturation process of the scar after the primary injury occurs, which coincides with the time of clinical symptoms appearing [5,6].

Molecularly, there is a characteristic pro-fibrotic inflammatory profile of the scar microenvironment in iLTS. Key mediators, such as transforming growth factor-beta 1 (TGF- β_1), are significantly upregulated in iLTS tissue. This will increase the expression of elastin and procollagen in fibroblasts from scar tissue. It also induces enzymes that cross-link these proteins to help stabilise and organise the extracellular matrix [7]. A hypoxic microenvironment has been identified as a significant driver for the transformation of normal airway fibroblasts into a pro-fibrotic, highly contractile myofibroblast phenotype, a process accompanied by the upregulation of markers like interleukin-6 (IL-6) and alpha-smooth muscle actin (α -SMA) [8]. Compared to normal fibroblasts, fibroblasts derived from iLTS proliferate more rapidly, exhibit enhanced migratory capacity, and show partial resistance to the inhibitory effects of antifibrotic signals such as prostaglandin E2 [9,10]. Genetic studies suggest that specific single-nucleotide polymorphisms (SNPs) in the TGF- β_1 gene and the matrix metalloproteinase-1 (MMP-1) gene may be associated with an individual's susceptibility to developing iLTS [11,12]. Furthermore, microbiome analysis indicates a reduced microbial diversity at the airway scar sites in iLTS patients and a higher detection rate of *Acinetobacter baumannii*, a pathogen associated with ICU-acquired infections. This suggests that microbial dysbiosis may contribute to shaping the local inflammatory microenvironment [12,13].

2.2. Hypothesised Mechanisms of Idiopathic Subglottic Stenosis (iSGS)

iSGS is a chronic fibroinflammatory disease which is almost all middle-aged and older women and has no clearly identified cause [2,14]. Its fundamental pathophysiology is likewise abnormal wound healing, but there is a much more complicated group of triggers.

The effect of estrogen has been widely known. Recent studies have detected upregulated expression of estrogen receptor alpha (ER- α) within the stenotic tissue of iSGS, while the expression of estrogen receptor beta (ER- β) and progesterone receptors may be decreased [15,16]. Given the established role of estrogen in regulating inflammatory responses and epithelialization in skin wound healing, its differential expression within the local airway microenvironment is hypothesised to contribute to the female predilection of iSGS [17,18].

In the field of inflammatory pathologies, iSGS is a functional pattern. Research has confirmed that there are significant activations in the il-23/il-17a axis in iSGS tissue, and $\gamma\delta$ T cells producing il-17 possibly serve as key effector cells [19]. il-17 can not only synergize with tgf- β_1 to directly promote proliferation of iSGS fibroblasts and ECM production, but also stimulate these cell to secrete chemo attractant so it further amplifies local inflammation and creates a self-sustainment loop [20], and expression of both TH2 (IL-4) & TH1 (IFNG- γ) cytokine increases, shows it have Mixed profile instead of purely pure Th2 response [21].

Laryngopharyngeal reflux has been regarded as a possible incitement to iSGS for quite some time; however, the evidence is conflicting. Pepsin has been detected in biopsy specimens from some patients [22]; there is a high comorbidity rate in the clinic [23,24], but recent in vitro experiments have shown that exposing fibroblasts to pepsin did not significantly trigger a fibrotic response. According to the above clinical studies, anti-reflux surgery or therapy does not reduce the degree of stenosis further [25]. Therefore, reflux is more likely to be one of the reasons than the sole reason.

Patients with iSGS have an increased abundance of Moraxellaceae, such as *Moraxella* and *Acinetobacter*, and a

reduced quantity of the beneficial commensal *Prevotella* [26]. The above microbes can prolong chronic inflammation of the airways.

3. Surgical Airway Anatomy for Laryngotracheal Stenosis

Surgical treatment for laryngotracheal stenosis (LTS) includes both endoscopic minimally invasive procedures and open reconstructive surgery. A thorough understanding of airway surgical anatomy is essential to minimise perioperative complications and optimise surgical success. The following sections will focus on the key anatomical structures and surgical procedures from the glottis to the main bronchi that are relevant to the diagnosis and treatment of LTS [27].

3.1. Overall Anatomy and Segments of the Trachea

The lower limit of the thyroid cartilage is adjacent to the beginning of the trachea. It is about 9–11 cm long in adults, and for very tall people, it can be as large as 12–13 cm [28,29]. The upper part is in the neck at the C6–C7 level, and the lower part extends to the chest, running in front of the spine and ending at T4–T5 [30]. The two types mentioned above have different clinical applications; the cervical trachea can be accessed through a neck incision, but exposing the intrathoracic trachea and carina generally requires a median sternotomy or a right thoracotomy [31]. There are 15 to 22 C-shaped cartilage rings and a membranous posterior wall in the framework of the trachea. With age, the cartilage is more likely to calcify and thus more easily fragment during suturing [28].

3.2. Surgical Anatomy of the Cervical Trachea

The most commonly damaged part of the cervical trachea in LTS surgery is the subglottic area, and frequently, iatrogenic stenosis occurs here [32].

The subglottic region is anteriorly limited by the cricothyroid membrane and posteriorly by the cricoid lamina, and it is used to perform emergency cricothyrotomy [33]. The trachea is supplied by branches of the inferior thyroid arteries in the neck and intercostal arteries in the thorax. These vessels enter the tracheal wall laterally at the 3- and 9-o'clock positions [28]. During circumferential mobilisation of the trachea, dissection must be close to the tracheal wall to preserve this lateral blood supply; otherwise, there will be anastomotic ischaemia, poor healing, or even necrosis [34].

The recurrent laryngeal nerves need to be handled carefully during dissection in this area [35]. On the right is the nerve loops under the subclavian artery, and on the left, it loops under the aortic arch. Both nerves then rise in the tracheoesophageal groove to supply the vocal cords. When dissecting the lateral aspects of the cervical trachea, especially while handling the thyroid and paratracheal tissues, it is imperative to stay close to the tracheal wall to avoid entering the plane where the nerves course. The use of bipolar electrocautery is recommended to minimise the risk of thermal injury [28,36].

When an elective tracheostomy is performed, the stoma is typically placed at the level of the second or third tracheal ring. A position too high (near the cricoid cartilage) increases the risk of subglottic stenosis; a position too low may cause the tip of the cannula to erode the right brachiocephalic (innominate) artery anteriorly, potentially leading to a life-threatening trachea-innominate artery fistula [28].

3.3. Exposure of the Intrathoracic Trachea and Carina

Surgical exposure is more challenging for complex stenoses involving the intrathoracic trachea or carina.

Retrosternal Area: The trachea in the upper mediastinum has a close relationship anteriorly with the right brachiocephalic artery. After tracheal anastomosis in this area, the anastomosis must be separated from the artery using a pedicled tissue flap (e.g., thymic flap) to prevent the formation of a delayed fistula [28].

The carina lies approximately 2–3 cm inferior to the sternal angle, at the T5–6 vertebral level. Exposure via a median approach requires opening the pericardium and may necessitate downward retraction of the right pulmonary artery to gain space. Exposure via a right posterolateral thoracotomy often requires division of the azygos vein arch, which crosses over the right main bronchus and lies adjacent to the right lateral wall of the trachea, making it a structure also susceptible to injury during mediastinoscopy [28,34].

Lymph-node biopsy at station 7 should be performed last, as these nodes are supplied primarily by the bronchial

arteries [37], and early biopsy may provoke significant bleeding.

3.4. Brief Anatomy of the Main Bronchi

Knowledge of main bronchial anatomy is relevant in the rare LTS surgeries involving the carina or proximal bronchi. The right main bronchus is shorter, wider, and more vertical (approximately 2 cm long), while the left main bronchus is longer and more horizontal (approximately 4–5 cm long) [38]. The right main bronchus is commonly exposed via a right thoracotomy. The entire length of the left main bronchus can be exposed through a median sternotomy, making it more convenient than the right bronchus for intraoperative cross-field ventilation when needed [28].

4. Laryngotracheal Stenosis: Advances in Tissue-Resection Techniques

At present, for laryngotracheal stenosis (LTS), with the deepening of research on this disease, its historical reliance on mechanical dilation has gradually become outdated and has shifted to precision scar removal. The deficiency of dilation is that it does not address the fibrotic tissue itself, which often results in a cycle of recurrence. To break the loop, currently, endoscopic lesion resection using lasers, cold knives and other instruments has become the mainstream minimally invasive therapy for LTS. These methods provide a more certain way to remove the obstacle, which is not only aimed at saving the airway lumen but also at reducing the frequency of re-intervention and treating the cause at the source. **Figure 2** illustrates the spectrum of currently available surgical techniques for LTS, ranging from endoscopic to open approaches.

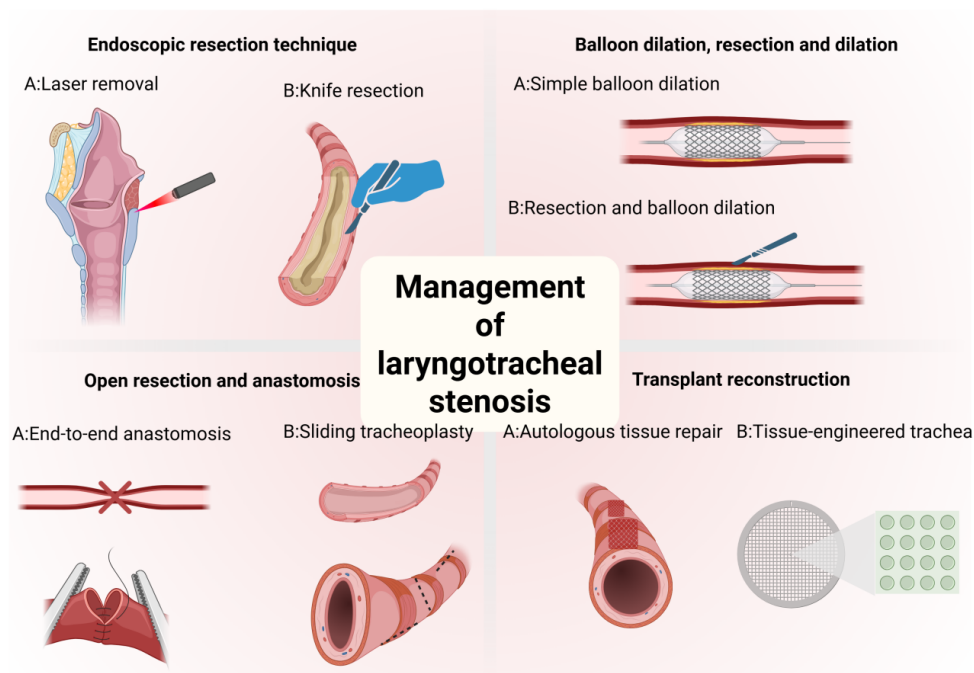


Figure 2. Spectrum of surgical techniques for laryngotracheal stenosis.

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These four sets of diagrams, ranging from endoscopic minimally invasive procedures (Panels 1 and 2) to open radical reconstructive surgery (Panel 3), and further to advanced reconstruction for complex defects (Panel 4), systematically and vividly illustrate the stepwise, individualised treatment technical system for laryngotracheal stenosis.

4.1. The Respective Philosophy: Laser and Cold-Knife Excision

Laser technique and cold-knife procedure are both based on a subtractive way of surgery to physically remove the fibrotic scar causing narrowing of the airway. This is a good deal more conceptually than merely dilative ones.

Techniques evolving: The lasers that we have nowadays are not the simple radial incisions which would dilate into a somewhat precisely wedged or segmental excision. For example, in the case of endoscopic CO₂ laser wedge excision, a wedge of scarred tissue is resected en bloc; Mucosal bridges left untouched between resected zones are meant to ease re-epithelialization and limit circumferential scarring [39]. Likewise, cold-knife surgery has progressed from basic lysis to precise microdissection & excision of fibrotic bands & webs with sharp blades under high-magnification endoscopy [40]. This move towards complete removal is with the aim of dealing directly with the cause of narrowing.

4.2. Mechanisms, Advantages, and Comparative Profiles

Although both laser and cold-knife excision have the same aim to remove the tissue, their operation is based on fundamentally different mechanisms, and this fact brings about various benefits and aspects to consider.

4.2.1. Laser Excision (Primarily CO₂ Laser)

The mechanism uses photothermal energy. CO₂ laser wavelength (10.6 μm) is highly absorbed by the tissue water and instantly vaporised, cutting with a very narrow area of thermal change (about 0.1 mm) [41]. Precision and Hemostasis: It enables precise, hemostatic microdissection under microscopic visualisation, and it provides reliable hemostasis for delicate vascular tissues in the airway. Good for posterior glottic and subglottic areas that are hard to place with mechanical instruments. Its non-contact quality ought to also make adjacent tissue less damaged.

4.2.2. Cold-Knife Excision

Mechanism: Mechanical shear based on sharp blades to completely avoid any heat energy transfer. Absence of Thermal Injury: No risk from heat, such as charring, delayed thermal necrosis, and even stimulation of surrounding tissues resulting in fibrosis. This is a principal advantage of mechanical cutting. Direct tactile feedback to the user, who can use this to help distinguish scarred tissue from flexible mucosa.

Generally, it requires less investment in equipment and is common. Among them, the options are closely weighed. In a single-centre retrospective comparison, the two-year treatment-free survival favoured lasers (50.2% vs. 31.9%), though not significantly ($p = 0.07$); Larger cohorts are needed [42]. In people who hadn't undergone airway surgery, results were about the same, but lasers were clearly better than revisions [42]. But it seemed to be a trend of longer-lasting effects with lasers. The same result as Hsieh et al found, cold-knife therapy would have a higher risk of having to perform another operation compared to the laser group about 2.3 times (Risk ratio (OR) = 2.31, $p = 0.037$) [42], this shows that cold-knife excision can be still one of the very effective first choice of resection tool for primary, clean cut stenosis while laser may be better applied in complicated situation or revision surgeries when more blood needs stopping.

4.3. Indications, Efficacy, and Role in Multimodal Therapy

Both techniques are most appropriate for short-segment, focal stenoses with little cartilaginous loss. They are the first choice in case of iSGS or post-intubation membranous stenosis [43,44]. Studies show that ESDCO₂ laser wedge excision with medical treatment has low recurrence rates (e.g., 12.4% in one iSGS study) and better results for voice [45].

Notably, neither endoscopic resection is curative for > 1–1.5 cm length stenosis; those with marked chondral framework collapse and/or complex multilevel obstruction (e.g., some post-COVID-19 stenoses) require open surgical reconstruction [27,46].

In current practice, the results of endoscopic resection are often optimised within a multimodal treatment framework. Adjuvant therapies are frequently employed, and they are commonly applied immediately after resection to help maximise luminal patency. Among these, topical mitomycin C, an antifibrotic agent, and intralesional corticosteroid injections are widely used as local adjuncts to modulate wound healing and reduce the risk of recurrent scarring [47].

Although laser and cold-knife excision are effective for focal fibrous stenoses, the residual lumen often requires further enlargement to maintain patency. Balloon dilation meets this need, whether as an immediate adjunct after resection or as a standalone procedure for simple membranous stenoses.

5. Bronchoscopic Balloon Dilation (BBD)

Balloon dilatation of bronchoscopy (BBD) is the most commonly needed operation in interventional pulmonology. Place balloons of different specifications of balloon catheters in the stenotic part of the airway under bronchoscopic guidance. The physical dilation force of the balloon mechanically disrupts the hyperplastic scar and fibrous tissue to restore airway patency. Since it was first used clinically in the 1980s to deal with post-tuberculosis stenosis, it has evolved into the first choice of therapy for dealing with benign airway stenosis. The development process is not only along with the advancement of interventional devices, but more importantly, it also reflects a change in the concept of surgery for bolder resections to be less invasive and preserve function.

5.1. Technical Principle and Core Advantages

To treat benign laryngotracheal stenosis, Balloon Dilation which is simple and basic Endoscopic Mechanical Dilation Method. By applying a radially outward force on the stenosis site with a balloon catheter and expanding the lumen in a non-cutting way. It is reported to carry a relatively low risk of mucosal injury, which may help preserve mucosal integrity and lower the potential of secondary scar formation caused by excessive tissue damage [48].

5.2. Clinical Applications and Indications

BBD is primarily employed to manage benign airway stenoses, such as post-intubation/tracheostomy stenosis, post-infectious cicatricial stenosis (e.g., tuberculosis-related), and stenoses caused by inflammatory diseases like granulomatosis with polyangiitis.

For newly diagnosed, focal, and simple subglottic or tracheal stenoses, balloon dilation is considered a first-line option. It is particularly indicated for short-segment (typically < 1 cm), circumferential, or web-like stenoses [48]. Endoscopic balloon dilation is also a common option for mild pediatric LTS in clinical practice [49]. Furthermore, in the early management of acute post-intubation laryngeal injury, the timely use of balloon dilation combined with intralesional steroid injection and granulation tissue debridement has been proven effective in addressing early fibroinflammatory lesions, reducing the total number of future interventions required, and holding the potential to avert subsequent open reconstruction surgery [50].

5.3. Efficacy Evaluation

For pediatric subglottic stenosis (SGS), a meta-analysis encompassing 14 studies and 473 patients demonstrated that BBD, as a minimally invasive intervention, achieved an overall success rate of 76% (95% CI: 0.65–0.86), a result of high statistical significance ($p < 0.001$) [51].

BBD shows significant value in treating tuberculosis patients. Case reports indicate that multiple, staged balloon dilations can achieve airway patency with no observed recurrence during a one-year follow-up period. However, its long-term efficacy for patients with prolonged disease courses requires further investigation [52].

And prospective studies prove to us that BBD brings immediate Airway Function Improvements. For example, a study conducted with 50 SGS patients found that the spirometry parameters included FVC, FEV1, and PEF and showed statistical improvements after 30 days post-BBD [53].

5.4. Adjunctive Therapies and Integrated Strategies

Clinical use of BBD is nearly never as monotherapy. Its effects are associated with adjunctive therapies, technical details and holistic therapeutic schemes. To avoid restenosis, topical application of mitomycin C (MMC) and postoperative administration of inhaled corticosteroids (ICS) are the most frequently used auxiliary approaches; however, neither has been fully supported by strong evidence.

MMC: A prospective study on post-intubation tracheal stenosis showed that all 7 patients receiving rigid bronchoscopic dilatation plus a single application of MMC at the site of injury had 100% restenosis within an average period of 27 days [54].

ICS: A randomised controlled trial found no added benefit of post-operative inhalation of fluticasone on lung function when compared with controls [55].

Studies show that the routine use of single-modality adjunctive pharmacotherapy is uncertain in terms of cost-effectiveness and reveals the complicated pathophysiological mechanisms of restenosis.

Multimodal combined therapy: Clinical practices tend more towards multimodal combined treatments. Large-sample retrospective studies involving 80 patients with an average follow-up of 2.9 years confirmed that such all-encompassing measures were generally safe and operable. However, patients needed a mean number of 2.8 treatments, suggesting many cases require repeating [56].

5.5. Technical Refinements and Safety

Traditional BBD operation needs to fully block the airway by balloon inflation, which makes it hard for patients to breathe. Research directions have included the following refined contents:

Shorten inflation time: by significantly lowering (to 10–60 s) the length of a single inflation, ventilatory recovery was accomplished in the interval between inflations, thus striking a balance between the demands for dilation and oxygenation [52].

Maintaining Auxiliary Ventilation: Studies have confirmed that maintaining auxiliary ventilation during the process, using an ultrathin catheter or LMA (laryngeal mask airway), can maintain the patient's partial pressure of oxygen (PaO₂). This method only has temporary CO₂ retention, but the airway lumen can be effectively enlarged [41,57].

New technologies have changed the BBD model from "dilation only" to a combined approach of "dilation plus ventilatory support", and this method is safer and has expanded the application scope of BBD to critically ill patients.

5.6. Long-Term Efficacy and Limitations

A single-centre, medium-term retrospective study of 35 patients over an average period of about 33 months employed BBD as the primary therapy. Both the short-term safety and efficacy of the procedure were good; immediately after dilation, all patients showed an expansion of the airway diameter, symptomatic relief, and an improvement of 10.5% in FEV1 that persisted for over a month. However, by the end of the follow-up period, 71% of the patients (25/35) had not been resolved and still required a second final operation due to restenosis. Ten patients (10/35) died from the progression of their underlying diseases during the study period, and none of these deaths was due to BBD [58].

5.7. Comparative Efficacy of Dilation Techniques

Many LTS studies have compared balloon dilation with rigid dilation. A large retrospective pediatric study of the results of 156 rigid dilation procedures on 62 children (68 treatment courses) showed a total success rate of 70.6% without procedure-related adverse events. Rigid dilators are relatively inexpensive, provide good tactile feedback, and have achieved the same success rates as those reported for balloon dilation [54]. Another retrospective study that directly compared the two ways found that both were safe and effective, and the complication rates were 2.9% for balloon dilation and 4.2% for rigid dilation. Balloon dilation had better long-term outcomes, and a larger proportion of patients achieved extended symptom relief for more than 8 weeks (71% compared with 31.2% for others) and had a longer mean time to recurrence (27.9 vs. 19.6 weeks). The effects of balloon dilation at different sites were not the same. Subglottic stenosis had the highest rate of symptom relief after more than 8 weeks at 92%, significantly higher than for upper tracheal (62%) and mid-tracheal (20%) stenoses [59].

A 2-year retrospective cohort study on balloon dilation and tracheal stent placement. For the endpoint of sustained airway patency at 2 years, stents were significantly better than balloon dilation (risk ratio 3.9). The above reasons were more prominent in cases of severe stenosis, idiopathic causes or stenoses exceeding 70%. However, the increase in patency came at the cost of a rise in adverse events [60]. Therefore, stenting may be more beneficial for complex benign stenoses in the long term and is more stable, but it also has a relatively higher short-term risk; on the other hand, balloon dilation has a lower complication rate but may not provide the same extended stability.

Bronchoscopic balloon dilation is a relatively simple method for treating benign stenosis of the airways, and it is more suitable for early-stage, short-segmental and morphologically simple lesions. It is relatively safe in the short run and effective; both the symptoms and functions improve quickly. At some places in the human body, such as the subglottis, a balloon dilator can be used to extend the effect of symptom relief after rigid dilation for a longer period of time. Stents have a lower complication rate after placement, but they do not offer extended support for the blood vessel. The restenosis rate after balloon dilation alone is relatively high in a clinical setting; therefore, many more patients need repeated surgery or combination therapy with other kinds of treatment [61,62]. Adjunctive pharmacotherapy, such as topical mitomycin C or inhaled corticosteroids, is still debated, although some studies

have reported a positive effect of topical MMC [63]. Adjuvant pharmacotherapy, such as topical mitomycin C or inhaled corticosteroids, is not yet standardised, although some studies have reported a positive effect of topical MMC.

Whether tissue resection or balloon dilation is employed, mechanical intervention alone cannot fundamentally interrupt the dysregulated wound-healing cascade, which accounts for the persistently high restenosis rates. Pharmacologic adjuncts targeting the wound-healing process itself, therefore, constitute an indispensable third component of management.

6. Adjunctive Therapies for Laryngotracheal Stenosis: An Integrated Perspective

Management of LTS is no longer solely through a single surgery. To enhance the main treatment effects, reduce recurrence and minimize/minor/decrease complications, will use adjunct therapy.

Local Pharmacologic Adjuncts

Endoscopic/open procedure (stenotic site) → local drug delivery (to the stenotic site, before/after); this is a well-acknowledged way of interfering with abnormal wound healing.

MMC, a mitomycin C alkylating agent, can inhibit the proliferation of fibroblasts and collagen deposition. Clinical evidence is lacking. Although both retrospective and prospective cohort studies have shown that MMC can significantly extend the asymptomatic period after endoscopic surgery relative to just surgical intervention [64,65], a small-sized prospective randomised trial of n = 7 did not find any significant improvement [66]. It shows the effects, maybe differences in different patients, stenosis situations and how we apply it.

Corticosteroids: Strong Anti-inflammatory effects, reduce oedema, and inflammatory cell infiltration. Injections include intra-lesional (for instance, serial in office for subglottic stenosis [67,68]), topically after resection, often included within maximal medical therapies [39]. A Meta-analysis showed local steroid use decreased restenosis after endo- procedure significantly, but without adding complications [47].

Novel Formulations & Targeted Delivery: Advancements focus on improving drug bioavailability and reducing systemic effects. Include injectable hydrogel; Thermosensitive polymers for release [69]; Nanoparticle carrier to deliver anti-inflammatory and anti-fibrotic agents.

For its secondary to systemic diseases such as GPA, it is important to control the underlying disease. Immunosuppressants like corticosteroids, cyclophosphamide, rituximab, etc., have the necessity of use for controlling disease activity so that lasting local interventive success can be achieved [70]. Systemic corticosteroids show an extension of the intervention-free interval in the animal model, but human clinical data is not available [71].

In recent years, two classic anti-fibrotic agents, pirfenidone and nintedanib, have been explored for the treatment of LTS. Related preclinical studies suggest that these drugs may inhibit the activation of fibroblasts and block profibrotic signalling pathways, thereby slowing the progression of tracheal fibrosis [72]. At present, pirfenidone has been reported to combine with endoscopic interventions to reduce the recurrence risk of scarring airway stenosis in individual clinical cases [73], while nintedanib exerts dual effects of anti-inflammation and anti-fibrosis in tracheal injury models [74]. However, large-sample clinical trials of the two drugs for LTS are still lacking.

The combined application of local drugs and systemic immunomodulatory therapy has gradually become a routine part of multimodal treatment. Novel drug delivery systems such as hydrogels and nanoparticle carriers also show promising application prospects in reducing scar formation.

7. Resection and Anastomosis

Resection and anastomosis are still a basic procedure in the definitive surgical management of LTS. And this way has some significant procedures according to Location & Extent of the stenosis.

7.1. Primary Resection and Anastomosis Techniques

7.1.1. Cricotracheal Resection (CTR)

This procedure is mostly suitable for subglottic and upper tracheal stenosis. Considered the standard surgical intervention for symptomatic subglottic stenosis in pediatric patients with decannulation rates near 90% [75]. In adults, CTR is also a safe and viable option for managing similar stenoses accessible via a cervical approach [76].

For complex benign tracheal stenosis, tracheal resection and anastomosis achieve a significantly higher long-term clinical resolution rate than tracheal stenting, especially for low-risk patients [77]. Due to its proximity to the glottis, the risk of postoperative complications such as laryngeal oedema is relatively higher, often necessitating a protective tracheostomy [78,79].

7.1.2. Tracheal Segment Resection and End-to-End Anastomosis

This is the preferred curative intervention for localised tracheal stenosis, particularly post-intubation tracheal stenosis (PITS) [80,81]. For post-intubation tracheal stenosis in adults, early success rates exceed 95%; outcomes for radiation-induced or post-COVID-19 stenoses are somewhat lower (86–90%) [78,82]. Álvarez-Maldonado, in a systematic review, supported its favourable outcomes and safety profile in both the short and medium term [83].

7.1.3. Slide Tracheoplasty

This technique is mainly employed for long-segment tracheal stenosis, especially in pediatric cases involving congenital complete tracheal rings, which are not amenable to traditional resection and anastomosis [84]. By sliding and overlapping the tracheal segments to enlarge the lumen, it circumvents the challenge of extensive resection. It may also require fewer postoperative interventions for granulation tissue compared to graft-based reconstruction [84]. A 28-year single-centre study involving 210 pediatric patients reported an overall survival rate of 90.5%, establishing it as the standard procedure for such complex stenoses [85]. In a small subset with tracheal trifurcation (n = 9), postoperative mortality reached 23% (2/9) [85].

The fundamental principle of resection and anastomosis is the complete excision of the stenotic segment followed by a tension-free primary anastomosis to restore airway continuity. While more invasive than tracheal dilation procedures, and compared to repeated endoscopic interventions like balloon dilation, open resection and anastomosis provides a permanent anatomical solution that significantly reduces the need for additional surgeries due to restenosis [46]. For severe stenoses (Cotton-Myer grades III–IV), this approach is a key option for achieving definitive airway reconstruction [86].

When performed in experienced centres by multidisciplinary teams, resection and anastomosis demonstrate excellent therapeutic outcomes. For acquired conditions like PITS, single-stage resection with primary anastomosis as a first-line treatment achieves an early success rate of 96.7%, with long-term follow-up (average > 4 years) showing stable anatomical and functional success rates between 86.3% and 95% [78,79,82,87].

7.2. Management of Special and Complex Cases

7.2.1. Long-Segment Stenosis

For lesions exceeding the traditional safe resection length (typically > 4 cm), slide tracheoplasty is employed to address pediatric congenital long-segment stenosis (e.g., complete tracheal rings) by overlapping the tracheal walls to enlarge the lumen, thereby avoiding the tension issues associated with ultra-long segment resection [84].

7.2.2. Complex and High-Risk Cases

This approach remains a safe and effective option in specialised centres for patients with a history of multiple prior surgical failures, idiopathic stenosis, post-tuberculosis stenosis, and even post-radiation stenosis [88–90]. Revision surgery presents significant technical challenges but remains feasible with meticulous technique [91].

7.2.3. Emerging Etiologies

Studies confirm that for post-COVID-19 tracheal stenosis, tracheal resection and anastomosis, when performed with strict adherence to surgical indications, exhibit safety and efficacy comparable to that in non-COVID-19 patients [92].

7.3. Integrated Perioperative Management

7.3.1. Multidisciplinary Team (MDT) Collaboration

Close collaboration between thoracic surgeons, anesthesiologists, intensive care physicians and speech therapists is essential to guarantee the safety and efficacy of LTS surgery [93]. Given the grounds for dealing with

complicated airway disease [94]. A study covering both benign and malignant stenosis also stresses the need for treatment in high-volume speciality centres [87].

7.3.2. Intraoperative Support Technology

Distal trachea or long-segment resections may be able to use ECMO for intraprocedural oxygenation and creation of a bloodless field. Research shows that the integration improves the overall safety of complex procedures [95]. Cardiopulmonary bypass (CPB) is usually used for pediatric long-segment stenosis surgery [84].

7.4. Technical Challenges and Evolving Strategies

Although the general rate is high, solving anastomotic tension, keeping good intraoperative ventilation and protecting the larynx and swallow function are all very difficult. In recent times, there has been some progress in precisely dealing with the aforementioned problems.

7.4.1. Minimally Invasive and Precise Tension Control

Anastomotic tension is an important risk factor for postoperative dehiscence, granulation tissue and restenosis [79–81, 90]. Traditional laryngeal release moves are able to decrease tension but increase the risk of postoperative dysphagia [79, 96]. Video-mediated mediastinoscopy allows for safe and accurate mobilisation of the mediastinal trachea, which extends the reach possible without tension substantially [79]. U-shaped tracheal retention suture is also a local tension-reducing method; it can add protection for the anastomosis by longitudinally spreading the stress [97]. In terms of suture technique, absorbable sutures (e.g., PDS) are commonly used now to reduce suture-related granulation [80, 81]; A running suture of the posterior wall may provide better mucosal apposition than interrupted sutures [79].

7.4.2. Refined Techniques for Functional Preservation

The first purpose of the current LTS approach is to maximise function preservation and open the airway. For high-grade stenosis next to the glottis, one must carefully balance the requirement for sufficient resection with the risk of recurrent laryngeal nerve injury or laryngeal oedema, and in some cases, a protective tracheostomy is required [86].

Protect the Swallowing Function at the same time. Intraoperative application of a limited infrahyoid release, with preservation of the superior horn of the thyroid cartilage, has been shown to significantly improve postoperative swallowing outcomes in older people compared with a full release [96].

7.4.3. Innovations in Intraoperative Support and Ventilation Strategies

ECMO provides the necessary protection with its oxygenation and makes the surgical area clearer by resecting the distal trachea or long-segment lesions, making complex surgeries safer as discussed previously [95]. CPB is generally used in paediatrics [84].

7.4.4. Timing and Biological Factors

Experimental studies suggest that the local inflammatory state (e.g., TNF- α levels) significantly impacts healing, and surgery during specific post-injury periods (weeks 4–6) may increase the risk of restenosis [98]. This shows the prospect of biomarker research for surgery time optimisation.

7.4.5. Limitations in Ultra-Long Segment Defects

As for ultralong segment defects, there are still a lot of breakthroughs needed to maintain good long-term stability and less need for interventions [84].

7.5. Discussion and Future Directions

Resection and anastomosis have developed into a mature, highly efficient, and constantly improving surgical system. Progress at present mainly includes the minimisation and refinement of techniques (such as video-mediastinoscopy, precise functional protection), systematisation of management (such as ECMO support, early en-

doscopy scoring), and comprehensiveness of evaluation (patient-centred functional outcomes). Future efforts will aim to minimise the occurrence of complications and achieve more targeted individualised treatment through the application of digital surgical technology (3D printing, simulation) [99]; Inflammatory biomarker-guided surgery timing [98] and the cutting-edge interdisciplinary areas of tissue engineering and regenerative medicine. These developments seek to offer more patients who have a better quality of life, as well as a curative treatment option.

8. Autologous Tissue Grafting: Functional Repair and Augmentation

In the Stepwise approach to LTS, resection with primary AO remains the most preferred & “gold-standard” of therapy for localised stenosis (typically $\leq 4\text{--}6$ cm), as it achieves excellent long-term patency and function via definitive lesion removal and direct repair [78, 79]. However, in the case of ultralong segment stenosis (> 6 cm) or a complicated circumferential defect, extending the extent of resection towards anastomosis is constrained by the safe mobilisation length of the trachea, and the anastomosis becomes tense. So, this would basically be why severe post-op complications happen, like anastomotic dehiscence, restenosis, etc. [90]. As a consequence, for lesions that are beyond the safety limits of conventional excision and anastomosis, graft reconstruction methods will be mainly employed in airway reconstruction. They developed from using their own tissues to repair, then began to explore using allografts for anatomical replacement, and finally moved towards the ideal target of tissue engineering construction.

8.1. Autologous Tissue Grafting

Autologous Tissue Grafting can't be replaced in complex airway management when allotransplantation is out of the question or a particular site requires special Structural Improvement. Mostly, this will have to be functionally repaired.

8.1.1. Autologous Costal Cartilage Grafts

Autologous costal cartilage grafts are commonly used for segmental stenosis resulting from cartilage loss or tracheomalacia, particularly when the disease process has weakened the anterior and lateral cartilaginous framework while the posterior mucosal wall remains relatively intact. In such patients, circumferential resection with end-to-end anastomosis often carries a high risk. Rather than enlarging or reconstructing the airway, surgeons may use carved costal cartilage grafts to restore structural support. However, because costal cartilage is essentially a heterotopically placed foreign biomaterial, it elicits a significant local inflammatory reaction. Compared with native-tissue procedures such as slide tracheoplasty, costal grafts are more prone to the formation of postoperative granulation tissue, a complication that often necessitates repeated bronchoscopic interventions [84].

8.1.2. Vascularized Composite Tissue Flaps

Extensive, irregular defects involving the larynx, trachea, and cervical soft tissues typically result from aggressive oncologic resections in which direct anastomosis is not feasible. To bridge these complex gaps, surgeons may employ vascularized composite tissue flaps, such as the radial forearm free flap. Although the robust blood supply of such flaps ensures graft viability, the functional outcomes are suboptimal: mucociliary clearance is completely absent. Moreover, because the biomechanical properties of flap tissue differ substantially from those of native tracheal cartilage rings, the reconstructed airway remains at risk for late softening and collapse [100].

8.2. Allogeneic Tracheal Transplantation

For patients with a circular, ultra-long segment (> 6 cm) of the tracheal defect (such as congenital long-segment agenesis, severe post-traumatic loss) that is difficult to repair using autologous tissue, allogeneic tracheal transplantation provides the most similar approximation of a physiological anatomical support framework. Its successful application has taken repair to the level of organ replacement.

8.2.1. Vascularisation: The Cornerstone of Success

The blood supply to the area of the Trachea repair operation must be rich. Resection with primary anastomosis uses perfusion from the native tracheal stumps; free grafts are entirely dependent on neovascularisation.

Techniques of surgery have changed significantly; previously, indirect ways were used to wrap the graft with vascularised tissue for slow revascularisation, and now single-stage microvascular transplantation is employed. The latter method promptly restores arterial blood supply by fine microvascular anastomosis. At 20 months post-transplantation, this method has maintained good mucosal blood supply and normal ciliated epithelial function in the graft, thus showing clinical feasibility [101].

8.2.2. Innovations in Immunological Management

Due to the adverse effects of prolonged systemic immunosuppression, some new immunological strategies for tracheal transplantation have been proposed.

A typical case is the phenomenon of tissue chimerism. Research has shown that the luminal epithelium of a successfully transplanted trachea gradually changes to include recipient-derived epithelial cells and forms a chimeric epithelial layer. The above may be the reasons for the relatively low rejection rates of tracheal allografts and provide new ideas for the induction of local immune tolerance [102].

Another approach focuses on the creation of de-epithelialized grafts with immunoprivileged properties. These grafts exhibit reduced HLA-DR expression and, when pre-vascularized before transplantation, have been reported to remain patent and functional for more than 5 years in clinical cases without the need for systemic immunosuppression [103]. Collectively, these immunomodulatory strategies hold considerable promise for improving the safety and long-term efficacy of allogeneic tracheal transplantation.

8.3. Tissue-Engineered Trachea: Regenerative Medicine's Blueprint for the Future

Tissue engineering is intended to make a bioactive tracheal substitute with materials, cells and bioactive substances. It is a basic direction to solve the problem of donor shortage and realise personalised repair, but still in the exploration front-line stage. Tissue engineering combining biomaterials, stem cells and bioactive molecules has become a promising direction for repairing long-segment tracheal defects that cannot be treated by conventional resection and anastomosis [104]. However, incomplete epithelialization and mechanical instability remain major obstacles to clinical translation.

Insufficient revascularisation and delayed re-epithelialization are two key bottlenecks restricting the large-scale clinical application of tissue-engineered trachea [105]. Early clinical attempts using tissue-engineered tracheal substitutes could only achieve temporary ventilatory function, and long-term follow-up (> 1 year) in humans has always resulted in graft failure due to collapse or infection; Can offer basic ventilatory function. However, because there is no complete neural and vascular network and glandular structure, they do not form a physiologically complete organ [106]. The bottleneck of clinical translation: (1) Lack of ideal scaffold material combining biocompatibility, mechanical properties and degradable profile; (2) Insufficient efficacy for vascularisation, thus unable to treat long-segment defects; (3) Insufficient long-term stabilisation ability, as seen in an animal study showing softening, stenosis or collapse after operation. Existing research confirms that anastomotic stenosis of bioengineered tracheal grafts is primarily driven by TGF- β 1 signalling pathways, persistent proinflammatory macrophages and delayed re-epithelialization [107].

Advancement of this area requires integrating the 3D bioprinting technique towards personalised fabrications [99], also in combination with seeding cells, e.g., 3D bio-printed scaffolds loaded with mesenchymal stem cells can form cartilage-like tissue in vitro and achieve satisfactory airway repair in animal models [108]. Mesenchymal stem cells have the property of multi-potent differentiation and immunoregulation [109,110]. The end aim is to fabricate a "ready-to-use", flawless substitute trachea capable of attaining bio-integration and enduring stability.

9. Integrated, Personalised and Multidisciplinary Management for Laryngotracheal Stenosis (LTS)

In the present era, Laryngotracheal Stenosis (LTS) is moving away from a single-procedure-based approach towards an overall systemic framework comprising 3 I's—Integration, Individualisation, Interdisciplinary. In particular, the cause of LTS has many etiologies such as iatrogenic, idiopathic, radiation-induced, and congenital, and also involves diverse patient groups like pregnant women or those with severe comorbidities [111–113].

9.1. The Cornerstone of Multidisciplinary Teams (MDT)

Effectively dealing with the complex LTS needs a multi-professional coordinated group. A group that includes otolaryngology-head & neck surgery, thoracic surgery, pulmonology, anesthesiology, radiology, and often specialists in obstetrics, rheumatology, pediatric depends on the case [49,112]. MDTs' cycle begins with joint assessment: pulmonologists evaluate breathing, radiologists employ advanced imaging like thin-slice CT plus multiplanar reconstruction, virtual endoscopy for precise structure; surgeons do endoscopy, scoring stenosis scale and looking over intestinal lining continuity [44], then this team investigation takes us on to personal choice, formulating one's plan. For example, a patient with GPA has immunomodulatory therapy from a rheumatologist before or alongside surgery for the airway, as this will help manage systemic disease activity [112]. Similarly, a pregnant patient's MDT (obstetricians, anesthesiologists, otolaryngologists) prioritises fetal safety by choosing awake, least invasive procedures during pregnancy and planning definitive surgery after delivery [111,113].

9.2. Personalization of Treatment Strategies

Therapeutic Personalisation is stenoses' aetiology, localisation, length, and intensity (Myers-Cotton scale, for instance), combined with individual factors. Minimally invasive endoscopic methods are preferred for small segments and soft stenosis, usually combined with local add-ons such as intraluminal steroid injection, mitomycin C, etc., to reduce recurrence [43,47,114]. Conversely, open surgical reconstruction (e.g., laryngotracheal resection and end-to-end anastomosis, partial cricotracheal resection) is still necessary for long-segment, cartilaginous, or high-grade stenoses [115,116]. Choice is not fixed; a personalized strategy may involve a staged approach, from the beginning of endoscopic management, and then, if necessary, to open surgery [112].

Personalisation extends to the special population. Pregnant patients have physiological changes (mucosal swelling, decreased functional residual capacity), which intensify airway impairment; Fetal safety constraints diagnostic and therapeutic interventions. Management prefers conservative actions, non-radiative imaging (ultrasound, MRI), and timing perioperatively if necessary in the 2nd trimester if a procedure is imperative [117-119]. For those with post-COVID-19 LTS patient that has multi-level complex stenoses and poor pulmonary reserve, planning would take into account of increased risk of hoarseness, difficulty swallowing and restenosis, so careful perioperative care and rehabilitation are needed [27,120].

9.3. Integration of Adjunct and Emerging Therapies

Modern LTS management introduces new additions to the therapeutic array. More and more pharmacologic adjuncts are targeted. Sirolimus-eluting stents try to lessen Fibrosis through immune Modulation [121]. Gas Dynamics supported by Heliox, reducing the resistance of the airways, can be used temporarily for Congenital or Acute Stenosis [122,123].

Various surgical and reconstructive techniques have their own applicable scenarios. Formulating individualised strategies based on patient conditions relies on standardised multidisciplinary management. A combination of exact diagnosis, a wide range of treatments including endoscopic operation, open surgery and various systems for medical therapy, as well as novel auxiliary items. Future efforts should focus on standardising MDT procedures, validating bio-signals for disease condition observation, and introducing new technologies through in-depth clinical studies to further perfect this all-encompassing, customised mode [124-126].

Future research should focus on molecular subtyping of LTS and the application of artificial intelligence. Parallel efforts in LTS could identify gene signatures associated with fibrosis progression and treatment response. Moreover, deep learning models that integrate imaging, voice, and clinical data [127] could enable real-time intraoperative decision-making and personalised follow-up.

Table 1 is a comprehensive overview of currently available therapeutic modalities for LTS, categorised by technique type (endoscopic resection, endoscopic dilation, open reconstruction, and adjunctive therapy). For each technique, the table summarises the mechanism of action, primary indications, core advantages, major limitations, and clinical selection considerations to guide individualised treatment decision-making.

Table 1. Comparison of Major Treatment Techniques for Laryngotracheal Stenosis (LTS).

Technique Category	Technique Name	Mechanism	Primary Indications	Core Advantages	Major Limitations	Remarks (Selection Advice & Special Considerations)
Endoscopic Tissue Resection	CO ₂ Laser Excision	Photothermal vaporisation of tissue water.	Short-segment (< 1.5 cm), focal fibrotic stenosis (e.g., iSGS).	High precision, excellent hemostasis, non-contact.	Thermal injury risk, high equipment cost.	Preferred for vascular areas, complex/revision cases. Avoid cartilage exposure.
Endoscopic Tissue Resection	Cold-Knife Excision	Mechanical shearing with sharp blades.	Short-segment, well-defined fibrous bands/webs.	No thermal injury, provides haptic feedback, cost-effective.	Less ideal hemostasis requires high operator skill.	First-line for primary fibrous stenosis. Suitable for resource-limited settings.
Endoscopic Dilation	Bronchoscopic Balloon Dilation (BBD)	Radial force to cause microfractures in scar tissue.	Short-segment (< 1 cm), circumferential or web-like stenosis.	Minimal mucosal trauma, simple, repeatable.	High long-term restenosis rate, limited by dense scar.	First-line for simple stenosis or adjuvant post-resection. Bridge/palliative therapy for high-risk patients.
Open Surgical Reconstruction	Cricotracheal Resection (CTR)	Resection of anterior cricoid/stenotic segment; cricoid lamina to trachea anastomosis.	Fixed subglottic/upper tracheal stenosis.	Curative for subglottic stenosis, high long-term patency.	Risk of laryngeal oedema (may need tracheostomy), affects voice.	Standard for symptomatic subglottic stenosis. Protect recurrent laryngeal nerves.
Open Surgical Reconstruction	Tracheal Segment Resection & Anastomosis	Complete resection of stenotic segment; end-to-end anastomosis.	Localised tracheal stenosis (length ≤ 4–6 cm), e.g., PITS.	Definitive, permanent anatomical solution.	Length limitation due to tension, invasive.	First-choice curative surgery for localised stenosis. Safe in post-COVID-19 with strict selection.
Open Surgical Reconstruction	Slide Tracheoplasty	Longitudinal incision with transverse sliding/overlap to enlarge the lumen.	Long-segment stenosis (e.g., congenital complete rings).	Addresses ultra-long segments, uses native tissue.	Technically complex granulation tissue formation.	Standard for pediatric congenital long-segment stenosis. Requires CPB/ECMO support.
Adjunctive Therapy	Local Pharmacologic Agents	MMC inhibits fibroblasts; steroids reduce inflammation.	Post-resection/dilation to inhibit scar reformation.	Prolongs asymptomatic interval, reduces restenosis.	Unstandardized protocols, varying evidence.	Use within multimodal therapy. Avoid MMC leakage; monitor steroid absorption.

10. Conclusion

Management of Laryngotracheal stenosis has gradually moved away from single-operation surgery to all-encompassing, patient-centred care. At the same time, high spatial accuracy must be maintained in the division of the recurrent laryngeal nerve and segmental tracheal blood supply. Laser resection, cold-knife excision and balloon dilation are endoscopically performed for minimally invasive treatment of focal stenosis, but extensive or complex disease often requires open surgery for primary anastomosis or graft reconstruction. Add-on drugs, advanced ventilation methods and multi-disciplinary teams can all be used to enhance survival and reduce scarring. In the future, tissue engineering will continue to develop; at the same time, biomarker-guided timing of intervention, digital surgical planning and immunomodulation may all be introduced for personalised and effective treatment of LTS. In the end, the development prospect of LTS management is to keep advancing in molecular research, technology improvement and cooperative clinical application.

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References

1. Poetker, D.M.; Ettema, S.L.; Blumin, J.H.; et al. Association of airway abnormalities and risk factors in 37 subglottic stenosis patients. *Otolaryngol. Head Neck Surg.* **2006**, *135*, 434–437.
2. Gnagi, S.H.; Howard, B.E.; Anderson, C.; et al. Idiopathic subglottic and tracheal stenosis: A survey of the patient experience. *Ann. Otol. Rhinol. Laryngol.* **2015**, *124*, 734–739.
3. Branski, R.C.; Verdolini, K.; Sandulache, V.; et al. Vocal fold wound healing: A review for clinicians. *J. Voice* **2006**, *20*, 432–442.
4. Hirshoren, N.; Eliashar, R. Wound-healing modulation in upper airway stenosis—myths and facts. *Head Neck* **2009**, *31*, 111–126.
5. Liu, H.; Chen, J.C.; Holinger, L.D.; et al. Histopathologic fundamentals of acquired laryngeal stenosis. *Pediatr. Pathol. Lab. Med.* **1995**, *15*, 655–677.
6. Duynstee, M.L.G.; De Krijger, R.R.; Monnier, P.; et al. Subglottic stenosis after endolaryngeal intubation in infants and children: Result of wound healing processes. *Int. J. Pediatr. Otorhinolaryngol.* **2002**, *62*, 1–9.
7. Macauley, S.P.; Schultz, G.S.; Bruckner, B.A.; et al. Effects of transforming growth factor- β 1 on extracellular matrix gene expression by human fibroblasts from a laryngeal stenotic lesion. *Wound Repair Regen.* **1996**, *4*, 269–277.
8. Yin, L.X.; Motz, K.M.; Samad, I.; et al. Fibroblasts in hypoxic conditions mimic laryngotracheal stenosis. *Otolaryngol. Head Neck Surg.* **2017**, *156*, 886–892.
9. Sandulache, V.C.; Singh, T.; Li-Korotky, H.S.; et al. Prostaglandin E2 is activated by airway injury and regulates fibroblast cytoskeletal dynamics. *Laryngoscope* **2009**, *119*, 1365–1373.
10. Singh, T.; Sandulache, V.C.; Otteson, T.D.; et al. Subglottic stenosis examined as a fibrotic response to airway injury characterized by altered mucosal fibroblast activity. *Arch. Otolaryngol. Head Neck Surg.* **2010**, *136*, 163–170.
11. Rovó, L.; Széll, M.; Bella, Z.; et al. The -509 C/T genotype of TGF β 1 might contribute to the pathogenesis of benign airway stenosis. *Otolaryngol. Head Neck Surg.* **2010**, *142*, 441–443.
12. Anis, M.M.; Krynetskaia, N.; Zhao, Z.; et al. Determining candidate single nucleotide polymorphisms in acquired laryngotracheal stenosis. *Laryngoscope* **2018**, *128*.
13. Gelbard, A.; Katsantonis, N.G.; Mizuta, M.; et al. Molecular analysis of idiopathic subglottic stenosis for *Mycobacterium* species. *Laryngoscope* **2017**, *127*, 179–185.
14. McCaffrey, T.V. Classification of laryngotracheal stenosis. *Laryngoscope* **1992**, *102*, 1335–1340.
15. Damrose, E.J.; Campbell, R.D.; Darwish, S.; et al. Increased expression of estrogen receptor beta in idiopathic progressive subglottic stenosis. *Laryngoscope* **2020**, *130*, 2186–2191.
16. Fiz, I.; Bittar, Z.; Piazza, C.; et al. Hormone receptors analysis in idiopathic progressive subglottic stenosis. *Laryngoscope* **2018**, *128*.

17. Ashcroft, G.S.; Greenwell-Wild, T.; Horan, M.A.; et al. Topical estrogen accelerates cutaneous wound healing in aged humans associated with an altered inflammatory response. *Am. J. Pathol.* **1999**, *155*, 1137–1146.
18. Horng, H.-C.; Chang, W.-H.; Yeh, C.-C.; et al. Estrogen effects on wound healing. *Int. J. Mol. Sci.* **2017**, *18*, 2325.
19. Gelbard, A.; Katsantonis, N.G.; Mizuta, M.; et al. Idiopathic subglottic stenosis is associated with activation of the inflammatory IL-17A/IL-23 axis. *Laryngoscope* **2016**, *126*.
20. Morrison, R.J.; Katsantonis, N.G.; Motz, K.M.; et al. Pathologic fibroblasts in idiopathic subglottic stenosis amplify local inflammatory signals. *Otolaryngol. Head Neck Surg.* **2019**, *160*, 107–115.
21. Motz, K.M.; Yin, L.X.; Samad, I.; et al. Quantification of inflammatory markers in laryngotracheal stenosis. *Otolaryngol. Head Neck Surg.* **2017**, *157*, 466–472.
22. Blumin, J.H.; Johnston, N. Evidence of extraesophageal reflux in idiopathic subglottic stenosis. *Laryngoscope* **2011**, *121*, 1266–1273.
23. Jindal, J.R.; Milbrath, M.M.; Shaker, R.; et al. Gastroesophageal reflux disease as a likely cause of ‘idiopathic’ subglottic stenosis. *Ann. Otol. Rhinol. Laryngol.* **1994**, *103*, 186–191.
24. Maronian, N.C.; Waugh, P.; Azadeh, H.; et al. Association of laryngopharyngeal reflux disease and subglottic stenosis. *Ann. Otol. Rhinol. Laryngol.* **2001**, *110*, 606–612.
25. Bianchi, E.T.; Guerreiro Cardoso, P.F.; Minamoto, H.; et al. Impact of fundoplication for gastroesophageal reflux in the outcome of benign tracheal stenosis. *J. Thorac. Cardiovasc. Surg.* **2019**, *158*, 1698–1706.
26. Hillel, A.T.; Tang, S.S.; Carlos, C.; et al. Laryngotracheal microbiota in adult laryngotracheal stenosis. *mSphere* **2019**, *4*.
27. Nisa, L.; Leroyer, H.; Sandu, K. Open airway surgery for post-COVID laryngotracheal stenosis. *Eur. Arch. Otorhinolaryngol.* **2024**, *281*, 2531–2538.
28. Allen, M.S. Surgical anatomy of the trachea. *Chest Surg. Clin. N. Am.* **2003**, *13*, 191–199.
29. Yamamoto, T.; Schindler, E. Ideal endotracheal intubation depth at the vocal-cord level to avoid single-lung intubation using the percentage ratio of the tracheal length to body height. *Anaesthesiol. Intensive Ther.* **2023**, *55*, 32–37.
30. Newman, H.; Allen, J.; Narayanan, M.; et al. The challenges of using ultrasound to measure the trachea: A brief report. *Crit. Care* **2025**, *29*, 265.
31. He, J.; Yang, C.; Yang, H.; et al. Resection and reconstruction via median sternotomy incision for tracheal tumors. *Transl. Lung Cancer Res.* **2022**, *11*, 600–606.
32. Feng, Y.G.; Tao, S.L.; Mei, L.Y.; et al. Surgical treatment of severe benign tracheal stenosis. *J. Cardiothorac. Surg.* **2023**, *18*, 293.
33. Kemper, M.; Kleine-Brueggene, M.; Moser, B.; et al. Dimensional compatibility of currently available equipment for cricothyrotomy and adult airway anatomy: An in vitro analysis. *Br. J. Anaesth.* **2021**, *127*, 479–486.
34. Minnich, D.J.; Mathisen, D.J. Anatomy of the trachea, carina, and bronchi. *Thorac. Surg. Clin.* **2007**, *17*, 571–585.
35. Zhao, Z.L.; Wei, Y.; Peng, L.L.; et al. Recurrent laryngeal nerve injury in thermal ablation of thyroid nodules—risk factors and cause analysis. *J. Clin. Endocrinol. Metab.* **2022**, *107*, e2930–e2937.
36. Maddaus, M.A.; Toth, J.L.; Gullane, P.J.; et al. Subglottic tracheal resection and synchronous laryngeal reconstruction. *J. Thorac. Cardiovasc. Surg.* **1992**, *104*, 1443–1450.
37. Naruke, T.; Suemasu, K.; Ishikawa, S. Lymph node mapping and curability at various levels of metastasis in resected lung cancer. *J. Thorac. Cardiovasc. Surg.* **1978**, *76*, 832–839.
38. Drevet, G.; Conti, M.; Deslauriers, J. Surgical anatomy of the tracheobronchial tree. *J. Thorac. Dis.* **2016**, *8*, S121–S129.
39. Ekbom, D.C.; Bayan, S.L.; Goates, A.J.; et al. Endoscopic wedge excisions with CO₂ laser for subglottic stenosis. *Laryngoscope* **2021**, *131*, E1062–E1066.
40. Lavrysen, E.; Hens, G.; Delaere, P.; et al. Endoscopic treatment of idiopathic subglottic stenosis: A systematic review. *Front. Surg.* **2019**, *6*, 75.
41. Deshmukh, A.; Jadhav, S.; Wadgoankar, V.; et al. Airway management and bronchoscopic treatment of subglottic and tracheal stenosis using holmium laser with balloon dilatation. *Indian J. Otolaryngol. Head Neck Surg.* **2019**, *71*, 453–458.
42. Liang, K.Y.; Miller, K.M.; Syed, F.; et al. Laser versus cold steel for endoscopic management of subglottic stenosis. *Otolaryngol. Head Neck Surg.* **2024**, *171*, 471–477.
43. Mesolella, M.; Di Lullo, A.M.; Testa, D.; et al. The CO₂-laser in the treatment of laryngeal and tracheal stenosis: Our personal experiences. *Ann. Ital. Chir.* **2020**, *91*, 239–247.

44. Pandya, A.; Sreevidya, S.R.; Chaudhari, N.; et al. Laryngotracheal stenosis: Our experience in a tertiary care hospital. *Indian J. Otolaryngol. Head Neck Surg.* **2023**, *75*, 39–44.
45. Gelbard, A.; Anderson, C.; Berry, L.D.; et al. Comparative treatment outcomes for patients with idiopathic subglottic stenosis. *JAMA Otolaryngol. Head Neck Surg.* **2020**, *146*, 20–29.
46. Lewis, S.; Earley, M.; Rosenfeld, R.; et al. Systematic review for surgical treatment of adult and adolescent laryngotracheal stenosis. *Laryngoscope* **2017**, *127*, 191–198.
47. Ming, W.; Zuo, J.; Han, J.; et al. Local adjuncts to minimally invasive endoscopic interventions for benign laryngotracheal stenosis: A meta-analysis. *Eur. Arch. Otorhinolaryngol.* **2024**, *281*, 5395–5410.
48. Agrawal, A.; Baird, B.J.; Madariaga, M.L.L.; et al. Multi-disciplinary management of patients with benign airway strictures: A review. *Respir. Med.* **2021**, *187*, 106582.
49. Oscé, H.; Meulemans, J.; Hens, G. Contemporary surgical strategies for pediatric laryngotracheal stenosis: A comprehensive review. *Front. Pediatr.* **2025**, *13*, 1634634.
50. Nouraei, S.A.R.; Ghufloor, K.; Patel, A.; et al. Outcome of endoscopic treatment of adult postintubation tracheal stenosis. *Laryngoscope* **2007**, *117*, 1073–1079.
51. Alamri, A.A.; Alnefaie, M.N.; Alsulami, O.A.; et al. Endoscopic balloon dilatation for pediatric subglottic stenosis: A meta-analysis of successful outcomes. *Eur. Arch. Otorhinolaryngol.* **2024**, *281*, 3977–3984.
52. Fang, Y.; You, X.; Sha, W.; et al. Bronchoscopic balloon dilatation for tuberculosis-associated tracheal stenosis: A two case report and a literature review. *J. Cardiothorac. Surg.* **2016**, *11*, 21.
53. Meo, S.A.; Meo, I.M.U.; Al-Rouq, F.; et al. Lung functions pre- and post-endoscopic balloon dilation treatment among patients with subglottic stenosis. *Eur. Rev. Med. Pharmacol. Sci.* **2023**, *27*, 12021–12028.
54. Madan, K.; Agarwal, R.; Aggarwal, A.N.; et al. Utility of rigid bronchoscopic dilatation and mitomycin C application in the management of postintubation tracheal stenosis: Case series and systematic review of literature. *J. Bronchol. Interv. Pulmonol.* **2012**, *19*, 304–310.
55. Alrabiah, A.; Alsayed, A.; Aljasser, A.; et al. Effect of inhaled fluticasone propionate on laryngotracheal stenosis after balloon dilation: A randomized controlled trial. *Eur. Arch. Otorhinolaryngol.* **2021**, *278*, 1505–1513.
56. Parker, N.P.; Bandyopadhyay, D.; Misono, S.; et al. Endoscopic cold incision, balloon dilation, mitomycin C application, and steroid injection for adult laryngotracheal stenosis. *Laryngoscope* **2013**, *123*, 220–225.
57. Liang, Y.L.; Liu, G.N.; Zheng, H.W.; et al. Management of benign tracheal stenosis by small-diameter tube-assisted bronchoscopic balloon dilatation. *Chin. Med. J.* **2015**, *128*, 1326–1330.
58. Shitrit, D.; Kuchuk, M.; Zismanov, V.; et al. Bronchoscopic balloon dilatation of tracheobronchial stenosis: Long-term follow-up. *Eur. J. Cardiothorac. Surg.* **2010**, *38*, 198–202.
59. Glikson, E.; Abbass, A.; Carmel, E.; et al. Endoscopic management of benign laryngo-tracheal stenosis: Balloon vs. rigid dilatation. *Isr. Med. Assoc. J.* **2021**, *23*, 297–301.
60. Marchioni, A.; Andrisani, D.; Tonelli, R.; et al. Stenting versus balloon dilatation in patients with tracheal benign stenosis: The STROBE trial. *Laryngoscope Investig. Otolaryngol.* **2022**, *7*, 395–403.
61. Alaga, A.; Simhan, V.; Lokeshwaran, S.; et al. Management of postintubation tracheal stenosis with bronchoscope methods—an experience from two centers. *Respirol. Case Rep.* **2024**, *12*, e70014.
62. Alouda, N.; Almujaivel, N.; Alrabiah, A.; et al. Effect of intralesional steroid injections among patients with acquired laryngotracheal stenosis undergoing endoscopic balloon dilation using pulmonary function tests. *Ear Nose Throat J.* **2023**, *105*.
63. Rahbar, R.; Shapshay, S.M.; Healy, G.B. Mitomycin: Effects on laryngeal and tracheal stenosis, benefits, and complications. *Ann. Otol. Rhinol. Laryngol.* **2001**, *110*, 1–6.
64. Simpson, C.B.; James, J.C. The efficacy of mitomycin-C in the treatment of laryngotracheal stenosis. *Laryngoscope* **2006**, *116*, 1923–1925.
65. Cataneo, D.C.; Ximenes, A.M.G.; Cataneo, A.J.M. Mitomycin C in the endoscopic treatment of tracheal stenosis: A prospective cohort study. *J. Bras. Pneumol.* **2018**, *44*, 486–490.
66. Yung, K.C.; Chang, J.; Courey, M.S. A randomized controlled trial of adjuvant mitomycin-C in endoscopic surgery for laryngotracheal stenosis. *Laryngoscope* **2020**, *130*, 706–711.
67. Bertelsen, C.; Shoffel-Havakuk, H.; O'Dell, K.; et al. Serial in-office intralesional steroid injections in airway stenosis. *JAMA Otolaryngol. Head Neck Surg.* **2018**, *144*, 203–210.
68. Song, S.A.; Franco, R.A. Serial intralesional steroid injection for subglottic stenosis. *Laryngoscope* **2020**, *130*, 698–701.
69. John, M.; Nabizath, A.; Krishnakumar, S.; et al. Injectable tissue adhesive microgel composite containing antifibrotic drug for vocal fold scarring. *ACS Appl. Bio Mater.* **2024**, *7*, 5237–5246.
70. Aden, A.A.; Awadallah, A.S.; Xie, K.Z.; et al. Medical maintenance therapy following laser excision in patients

- with granulomatosis with polyangiitis (GPA)-associated subglottic stenosis. *Otolaryngol. Head Neck Surg.* **2024**, *171*, 180–187.
71. Shadmehr, M.B.; Abbasidezfouli, A.; Farzanegan, R.; et al. The role of systemic steroids in postintubation tracheal stenosis: A randomized clinical trial. *Ann. Thorac. Surg.* **2017**, *103*, 246–253.
 72. Fan, Y.; Li, X.; Fang, X.; et al. Antifibrotic role of nintedanib in tracheal stenosis after a tracheal wound. *Laryngoscope* **2021**, *131*, E2496–E2505.
 73. Li, X.; Pan, J.; Qian, H.; et al. Treatment of scarring central airway stenosis with pirfenidone: Case report. *Medicine (Baltimore)* **2022**, *101*, e31354.
 74. Wei, P.; Huang, Z.; Gan, L.; et al. Nintedanib ameliorates tracheal stenosis by activating HDAC2 and suppressing IL-8 and VEGF in rabbit. *Am. J. Transl. Res.* **2020**, *12*, 4739–4748.
 75. Gallagher, T.Q.; Hartnick, C.J. Cricotracheal resection and thyrotracheal anastomosis. *Adv. Otorhinolaryngol.* **2012**, *73*, 42–49.
 76. Stukov, Y.; Bilgili, A.; Redmond, K.; et al. Transcervical Tracheal Resection. *Multimed. Man. Cardiothorac. Surg.* **2025**.
 77. Marchioni, A.; Moretti, A.; Tonelli, R.; et al. Stent and resection anastomosis in patients with complex tracheal stenosis: The STARS retrospective multicenter trial. *Interdiscip. Cardiovasc. Thorac. Surg.* **2025**, *40*, ivaf261.
 78. Nauta, A.; Mitilian, D.; Hanna, A.; et al. Long-term results and functional outcomes after surgical repair of benign laryngotracheal stenosis. *Ann. Thorac. Surg.* **2021**, *111*, 1834–1841.
 79. Rubikas, R.; Matukaitytė, I.; Jelisiejėvas, J.J.; et al. Surgical treatment of non-malignant laryngotracheal stenosis. *Eur. Arch. Otorhinolaryngol.* **2014**, *271*, 2481–2487.
 80. Ulsan, A.; Sanli, M.; Isik, A.F.; et al. Surgical treatment of postintubation tracheal stenosis: A retrospective 22-patient series from a single center. *Asian J. Surg.* **2018**, *41*, 356–362.
 81. Ezemba, N.; Echieh, C.P.; Chime, E.N.; et al. Postintubation tracheal stenosis: Surgical management. *Niger. J. Clin. Pract.* **2019**, *22*, 134–137.
 82. Elsayed, H.; Mostafa, A.M.; Soliman, S.; et al. First-line tracheal resection and primary anastomosis for postintubation tracheal stenosis. *Ann. R. Coll. Surg. Engl.* **2016**, *98*, 425–430.
 83. Álvarez-Maldonado, P.; Hernández-Ríos, G.; Hernández-Solís, A.; et al. Tracheal resection and anastomosis in postintubation tracheal stenosis: A systematic review. *Eur. J. Cardiothorac. Surg.* **2024**, *66*, ezae330.
 84. Valencia, D.; Overman, D.; Tibesar, R.; et al. Surgical management of distal tracheal stenosis in children. *Laryngoscope* **2011**, *121*, 2665–2671.
 85. Beeman, A.; Ramaswamy, M.; Thiruchelvam, T.; et al. Slide tracheoplasty in long segment tracheobronchial stenosis. *Ann. Thorac. Surg.* **2025**, *120*, 355–364.
 86. Marston, A.P.; White, D.R. Subglottic stenosis. *Clin. Perinatol.* **2018**, *45*, 787–804.
 87. Ferreirinha, J.; Caviezel, C.; Weder, W.; et al. Postoperative outcome of tracheal resection in benign and malignant tracheal stenosis. *Swiss Med. Wkly.* **2020**, *150*, w20383.
 88. Ebada, H.A.; Abd El-Fattah, A.M.; Salem, E.H.; et al. Challenging tracheal resection anastomosis: Case series. *Auris Nasus Larynx* **2020**, *47*, 616–623.
 89. Vinh, V.H.; Khoi, N.V.; Quang, N.V.D.; et al. Surgical repair for post-tuberculosis tracheobronchial stenosis. *Asian Cardiovasc. Thorac. Ann.* **2021**, *29*, 26–32.
 90. Kanlikama, M.; Celenk, F.; Gonuldas, B.; et al. Cervical tracheal resection and anastomosis for postintubation tracheal stenosis. *J. Craniofac. Surg.* **2018**, *29*, e578–e582.
 91. Saetti, R.; Ronzani, G.; Meneghesso, S.; et al. Operative technique: tracheal resection and anastomosis in a revision surgery. *Head Neck* **2023**, *45*, 2730–2734.
 92. Piazza, C.; Lancini, D.; Filauro, M.; et al. Post-COVID-19 airway stenosis treated by tracheal resection and anastomosis: A bicentric experience. *Acta Otorhinolaryngol. Ital.* **2022**, *42*, 99–105.
 93. Zhan, Y.; Zhang, S.; Chen, M.; et al. Management of postintubation tracheal stenosis in a neurosurgical patient with tracheomalacia and scarring tendency: A case report. *Medicine (Baltimore)* **2026**, *105*, e48392.
 94. Parshin, V.D.; Porkhanov, V.A.; Polyakov, I.S.; et al. Improving surgical technique for tracheal resection with anastomosis. *Pirogov Russ. J. Surg.* **2024**, *1*, 6–20.
 95. Son, J.; Son, B.S.; Park, J.M.; et al. Innovative approaches in tracheal resection and anastomosis surgery: Integrating extracorporeal membrane oxygenation for enhanced safety. *Yonsei Med. J.* **2025**, *66*, 289–294.
 96. ElSobki, A.A.F.; El-Kholy, N.A.; Abdou, E.H.E.; et al. Swallowing outcomes after tracheal resection and anastomosis: Full versus mini infrahyoid laryngeal drop. *Eur. Arch. Otorhinolaryngol.* **2024**, *281*, 5899–5905.
 97. Karapolat, S.; Turkyilmaz, A.; Seyis, K.N.; et al. A comfortable solution to tracheal anastomosis protection: Tracheal retention sutures. *Heart Lung Circ.* **2018**, *27*, e39–e41.

98. Aydogmus, U.; Ozturk, G.; Kis, A.; et al. An experimental study on timing in tracheal stenosis surgery. *Thorac. Cardiovasc. Surg.* **2022**, *70*, 513–519.
99. Huang, C.Y.; Chang, T.S.; Hwang, L.A.; et al. Novel airway-cartilage combined model for medialization laryngoplasty and laryngotracheal reconstruction surgery planning. *J. Chin. Med. Assoc.* **2022**, *85*, 1076–1082.
100. Den Hondt, M.; Vranckx, J.J. Reconstruction of defects of the trachea. *J. Mater. Sci. Mater. Med.* **2017**, *28*, 24.
101. Genden, E.M.; Harkin, T.; Laitman, B.M.; et al. Vascularized tracheal transplantation: A twenty month follow up. *Laryngoscope* **2023**, *133*, 1839–1845.
102. Genden, E.M.; Chen, Y.W. Tracheal transplantation: Lessons learned that may apply to lung transplantation. *Curr. Opin. Organ Transplant.* **2024**, *29*, 407–411.
103. Cui, P.; Zhao, D.; Liang, L.; et al. De-epithelialized viable tracheal allotransplantation without immunosuppressants: 5-year follow-up. *Ann. Otol. Rhinol. Laryngol.* **2024**, *133*, 384–389.
104. Yeou, S.H.; Shin, Y.S. Tissue-engineered tracheal reconstruction. *Biomimetics* **2025**, *10*, 457.
105. Wei, S.; Zhang, Y.; Luo, F.; et al. Tissue-engineered tracheal implants: Advancements, challenges, and clinical considerations. *Bioeng. Transl. Med.* **2024**, *9*, e10671.
106. Parshin, V.D.; Lyundup, A.V.; Tarabrin, E.A.; et al. Long-term outcomes of tracheal transplantation: Success and unsolved problems. *Khirurgiia (Sofia)* **2018**, *11*, 1–9.
107. Weber, J.; Martins, R.S.; Muslim, Z.; et al. Anastomotic stenosis of bioengineered trachea grafts is driven by transforming growth factor β 1-induced signaling, proinflammatory macrophages, and delayed epithelialization. *JTCVS Open* **2023**, *15*, 489–496.
108. McMillan, A.; Hoffman, M.R.; Xu, Y.; et al. 3D bioprinted ferret mesenchymal stem cell-laden cartilage grafts for laryngotracheal reconstruction in a ferret surgical model. *Biomater. Sci.* **2025**, *13*, 1304–1322.
109. Jakobsen, K.K.; Grønhøj, C.; Jensen, D.H.; et al. Mesenchymal stem cell therapy for laryngotracheal stenosis: A systematic review of preclinical studies. *PLoS One* **2017**, *12*, e0185283.
110. Ganji, F.; Le Billan, F.; Haykal, S.; et al. Tracheal regeneration: Recent progress in the application of stem cells in tracheal bioengineering. *Int. J. Mol. Sci.* **2026**, *27*, 2891.
111. Smith, M.M.; Buck, L.S. Update on the diagnosis and management of pediatric laryngotracheal stenosis. *Expert Rev. Respir. Med.* **2022**, *16*, 1035–1041.
112. An, J.; Song, J.W. Life-threatening subglottic stenosis of granulomatosis with polyangiitis: A case report. *Med. Kaunas* **2021**, *57*, 423–430.
113. Queenan, N.; Trivedi, J.; Bertoni, D.; et al. Characterizing radiation-related laryngotracheal stenosis. *Am. J. Otolaryngol.* **2025**, *46*, 104643.
114. Di Felice, C.; Machuzak, M.S.; Shepherd, R.W. Use of mitomycin-C in laryngotracheal stenosis: A focused clinical review. *J. Bronchol. Interv. Pulmonol.* **2023**, *30*, 223–231.
115. Maurizi, G.; Vanni, C.; Rendina, E.A.; et al. Surgery for laryngotracheal stenosis: Improved results. *J. Thorac. Cardiovasc. Surg.* **2021**, *161*, 845–852.
116. Kokje, V.B.C.; Ishii, A.; Sandu, K. Moderate grade subglottic stenosis in children: Laryngotracheal reconstruction versus cricotracheal resection and anastomosis. *Front. Pediatr.* **2022**, *10*, 914892.
117. Obiyo, L.T.; Tobes, D.; Cole, N.M. Anesthetic recommendations for maternal and fetal safety in nonobstetric surgery: A balancing act. *Curr. Opin. Anaesthesiol.* **2024**, *37*, 285–291.
118. Lopez, C.E.; Salloum, J.; Varon, A.J.; et al. The management of pregnant trauma patients: A narrative review. *Anesth. Analg.* **2023**, *136*, 830–830.
119. Okune, Y.; Tateiwa, H.; Tsuruno, T.; et al. Comprehensive anesthetic management for posterior mediastinal tumor resection in the prone position: A case report. *Cureus* **2025**, *17*, e85210.
120. Brascia, D.; De Palma, A.; Cantatore, M.G.; et al. Not only acute respiratory failure: COVID-19 and post-intubation/tracheostomy upper airway lesions. *Front. Surg.* **2023**, *10*, 1150254.
121. Motz, K.M.; Lina, I.A.; Samad, I.; et al. Sirolimus-eluting airway stent reduces profibrotic Th17 cells and inhibits laryngotracheal stenosis. *JCI Insight* **2023**, *8*, e158456.
122. Del Puppo, M.; Meister, L.; Médale, M.; et al. Heliox simulations for initial management of congenital laryngotracheal stenosis. *Pediatr. Pulmonol.* **2023**, *58*, 230–238.
123. Allena, N.; Penikilapate, S.; Allu, S.; et al. Optimizing recovery: Heliox therapy for post-extubation stridor management. *Cureus* **2025**, *17*, e78740.
124. Nguyen, H.C.B.; Chao, T.N.; Cohen, N.A.; et al. Persistent inflammation and nitric oxide dysregulation are transcriptomic blueprints of subglottic stenosis. *Front. Immunol.* **2021**, *12*, 748533.
125. Cusumano, G.; Paternò, D.S.; Terminella, A.; et al. We were already there where the narrow path: Benign tracheal stenosis treatment—narrative review on the state of the art and future direction. *Updates Surg.*

2025, 78, 429–441.

126. Meister, K.D.; Pandian, V.; Hillel, A.T.; et al. Multidisciplinary safety recommendations after tracheostomy during COVID-19 pandemic: State-of-the-art review. *Otolaryngol. Head Neck Surg.* **2021**, *164*, 984–1000.
127. Gao, G.; Zhao, K.; Liu, M. Research progress in intelligent diagnosis of vocal fold lesions based on multimodal deep learning: A narrative review. *ENT Updates* **2026**, *16*, 26–41.



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