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Tracheobronchial Tear in Emergency Care: Rapid Recognition, Airway Stabilization, and Definitive Management Strategies

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Abstract

Background: Tracheobronchial tear is a rare but life-threatening airway injury resulting from blunt or penetrating trauma or iatrogenic interventions. Its diagnostic challenge and high mortality underscore the need for rapid recognition and effective management.

Aim: To review the etiology, epidemiology, pathophysiology, diagnostic strategies, and management principles of tracheobronchial tears in emergency care settings.

Methods: A comprehensive literature-based analysis was conducted, synthesizing evidence from trauma registries, postmortem studies, and clinical series. Mechanisms of injury, diagnostic modalities, and treatment approaches were examined to identify best practices for emergency and perioperative care.

Results: Tracheobronchial injuries occur predominantly near the carina and main bronchi, with blunt trauma associated with higher mortality than penetrating injuries. Up to 70% of cases are initially missed, often presenting with nonspecific signs such as persistent pneumothorax and subcutaneous emphysema. Fiberoptic bronchoscopy remains the gold standard for diagnosis and airway stabilization. Definitive management typically involves surgical repair, although conservative and endoscopic strategies are viable for select iatrogenic injuries. Prognosis improves significantly with early recognition and multidisciplinary intervention.

Conclusion: Early suspicion, bronchoscopy-guided airway control, and mechanism-sensitive management are critical to reducing morbidity and mortality. Structured trauma protocols and preventive measures for iatrogenic injury can further enhance outcomes.

Keywords: Tracheobronchial tear, airway trauma, bronchoscopy, emergency management, surgical repair, iatrogenic injury

Introduction

Tracheobronchial tear—also described as tracheobronchial laceration or tracheobronchial injury—represents an uncommon but highly consequential disruption of the tracheobronchial tree. Although infrequent in clinical practice, these injuries carry a disproportionate burden of morbidity and mortality because they directly compromise airway integrity, ventilation, and the ability to maintain oxygenation under emergent conditions. The tracheobronchial tree encompasses the trachea and its major bifurcation into the right and left mainstem bronchi, and injuries may occur at any point along this conduit. Clinically, a tear may involve the trachea alone, one mainstem bronchus, or, less commonly, multiple sites with complex patterns of disruption. For the purposes of this discussion, tracheobronchial tears can be defined as partial or complete lacerations, perforations, or puncture defects occurring anywhere within the tracheobronchial tree and arising either from blunt or penetrating trauma or as a complication of iatrogenic interventions, such as airway instrumentation or invasive procedures. The pathophysiologic significance of tracheobronchial disruption stems from the fact that even small defects can precipitate major air leaks into surrounding

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tissues, impair effective ventilation, and generate life-threatening complications such as tension pneumothorax, severe subcutaneous emphysema, mediastinal emphysema, or progressive respiratory failure. Moreover, these injuries frequently coexist with other major traumatic insults, including pulmonary contusions, rib fractures, vascular injuries, and blunt cardiac trauma, which together amplify physiologic instability and complicate diagnosis. In blunt trauma, rapid deceleration forces, compressive mechanisms, and shear stress at relatively fixed airway points can produce bronchial rupture, particularly near the carina or proximal main bronchi. Penetrating trauma, by contrast, typically causes more focal lacerations but may involve nearby vascular structures, raising the risk of hemorrhage and rapid deterioration. Iatrogenic causes, while distinct in mechanism, are clinically important because they may occur in patients already vulnerable due to critical illness, emergent intubation conditions, or underlying airway fragility.

Epidemiologically, tracheobronchial injuries are strongly associated with high prehospital mortality. It has been reported that nearly 80% of tracheobronchial injuries resulting from blunt trauma are expected to cause death at the scene or during transport, often due to the severity of associated multisystem injuries and the immediacy of airway compromise.[1][2] This sobering statistic underscores why clinicians in emergency and trauma settings encounter relatively few confirmed cases: many patients do not survive long enough to reach definitive care. Nevertheless, outcomes appear to be improving, a trend attributed in part to advances in prehospital management, including earlier recognition of respiratory compromise, improved airway strategies, and more timely transport to trauma-capable centers.[1][2] Even so, survival and long-term functional outcomes remain highly dependent on early identification and appropriate management. Because tracheobronchial tears can be diagnostically elusive—sometimes presenting with nonspecific signs such as dyspnea, persistent pneumothorax despite chest tube placement, or extensive subcutaneous emphysema—maintaining a high index of suspicion is essential. Early diagnosis, rapid stabilization, and coordinated multidisciplinary management are consistently emphasized as decisive factors in achieving favorable outcomes.[1][2] In this regard, tracheobronchial injury exemplifies a time-critical emergency in which clinical vigilance and prompt intervention can alter the trajectory from rapid decompensation to survivable recovery [2].

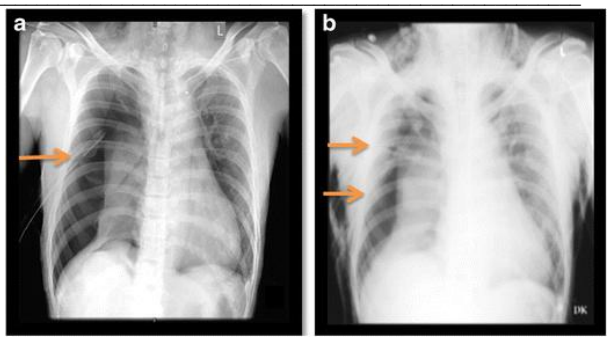

Fig. 1: Tracheobronchial tear.

Etiology

Tracheobronchial tears arise when mechanical forces exceed the tensile and elastic capacity of the tracheobronchial tree, producing partial-thickness lacerations, full-thickness ruptures, or complete transections. Although the trachea is relatively resilient—an elastic conduit composed of approximately 16 to 20 cartilaginous rings—it is not immune to disruption, particularly when high-energy forces are transmitted to the neck or thorax or when instrumentation is performed under emergent or technically challenging conditions. Anatomically, the trachea occupies a protected position, bordered anteriorly by the sternum and rib cage and posteriorly by the vertebral column. This relative protection helps explain why tracheobronchial tears are uncommon; however, the same surrounding structures can create compression and shear conditions during trauma that predispose the airway to rupture at predictable points, especially near fixed attachments and transitional zones. The etiologic spectrum is traditionally organized into penetrating trauma, blunt trauma, and iatrogenic causes, with motor vehicle collisions accounting for more than half of cases across penetrating and blunt mechanisms, reflecting the high prevalence of vehicular trauma and the magnitude of deceleration forces involved. Penetrating trauma is more likely to produce tracheobronchial laceration than blunt trauma, and within penetrating mechanisms, gunshot wounds are more common causes than stab wounds.[3] This pattern is clinically intuitive: projectiles may traverse deeper tissue planes and injure the airway even when external wounds appear modest, whereas stab wounds are often more localized but can still be devastating depending on trajectory. Penetrating injuries tend to create discrete perforations or segmental lacerations that may involve adjacent vascular structures, the esophagus, or the laryngeal framework, thereby increasing the complexity of airway management and the risk of hemorrhagic shock. While blunt trauma is a less frequent cause of tracheobronchial tear, mortality is higher in blunt injuries compared with penetrating trauma. This elevated mortality largely reflects the burden of associated multisystem trauma—such as pulmonary contusion, major vascular injury, traumatic brain injury, or blunt cardiac trauma—that

commonly accompanies high-energy blunt mechanisms and contributes to early death at the scene or during transport [3].

The anatomic distribution of injury differs by mechanism. Gunshot wounds frequently involve the cervical portion of the trachea, although injury can occur at any level depending on projectile path. Stab injuries, by contrast, almost always involve the cervical trachea, reflecting both accessibility and the typical angle of injury in assault scenarios.[3] Blunt trauma to the cervical trachea can occur indirectly through hyperextension mechanisms, as may happen during weightlifting accidents or sudden deceleration in motor vehicle collisions. Hyperextension can stretch the anterior neck structures while compressing the airway against the cervical spine, creating tensile forces that compromise the tracheal wall. Direct cervical tracheal injuries occur when external forces compress the neck and airway, such as wire strangulation during snow biking or similar high-risk activities, or when seat belts, steering wheels, and dashboards impact the anterior neck during a vehicular crash. In certain collisions, mixed mechanisms occur, combining compression with hyperextension and leading to complex injury patterns. One described scenario is "padded dashboard syndrome," in which the torso and neck experience abrupt deceleration and impact forces that together generate a high risk of airway and laryngeal disruption.[4] Within the cervical airway, the thyroid and cricoid cartilages are most commonly involved in severe injury, and a critical zone of disruption is often the region between the cricoid cartilage and the trachea.[4] This region represents an anatomical transition where rigid laryngeal structures meet the more flexible tracheal segment, predisposing it to tearing under abnormal stress. Age-related differences also influence injury patterns. In adults, calcification of laryngeal cartilages increases brittleness and therefore raises the likelihood of fractures when blunt forces are applied to the larynx. In children, by contrast, the laryngeal cartilages are more flexible and may be less prone to fracture when the force is concentrated on the larynx; however, this does not eliminate risk, as flexible structures can still deform significantly and sustain soft-tissue injury or edema. These distinctions are clinically important because they shape both the probability of structural disruption and the nature of airway compromise, with adults more prone to fracture-related instability and children potentially more prone to dynamic obstruction from swelling and soft tissue injury.

Blunt trauma more commonly involves the lower thoracic trachea and main bronchi, and the associated severe injuries contribute to the higher mortality observed in this group. Motor vehicle accidents are a dominant cause of intrathoracic airway disruption, affecting the intrathoracic trachea, carina, and mainstem bronchi. The carina is the most fixed point of the central airway system, tethered by surrounding mediastinal structures and bronchial attachments, and it is therefore highly susceptible to shear forces during sudden deceleration. When a moving body abruptly stops, different tissue compartments decelerate at different rates; the fixed carina becomes a focal point where shear stress concentrates, predisposing to rupture. As a result, blunt injuries are disproportionately common in the lower trachea and near the carina, and they may range from partial horizontal tears to complete separation. Vertical tears can extend upward from a carinal injury, reflecting propagation along the tracheal membranous wall under continuing stress. Lower tracheal injuries may also extend into one or both bronchi, causing partial or complete lacerations that compromise ventilation and can result in persistent pneumothorax despite chest tube drainage. Although intrathoracic tracheal injury is most often associated with high-energy trauma, lower-energy blows have also been reported to produce injury, underscoring that patient factors and the precise vector of force can sometimes permit airway disruption even when external trauma appears less severe. Anatomical asymmetry influences bronchial injury distribution in blunt trauma. Most blunt bronchial injuries occur on the right side, a pattern plausibly related to the bulkier right lung and the shorter right main bronchus, which may transmit forces differently and create higher local stress during compression and deceleration events. Kiser et al. reported in 2001 that bronchial injury is almost always located within 2 cm of the carina in approximately three-fourths of patients, emphasizing the predilection for pericarinal disruption in blunt mechanisms.[5] This observation has practical diagnostic implications: clinicians evaluating suspected tracheobronchial injury should pay particular attention to the pericarinal region during imaging and bronchoscopy, especially when clinical signs suggest a central airway leak.

Iatrogenic tracheobronchial injury is increasingly reported, reflecting the expanding use of airway instrumentation and advanced diagnostic and therapeutic procedures. Such injuries may occur during endotracheal intubation, rigid bronchoscopy, diagnostic transbronchial needle aspiration (TBNA) performed via endobronchial ultrasound (EBUS), and therapeutic airway interventions such as open or percutaneous dilatational tracheostomy, metallic airway stent deployment or removal, balloon bronchoplasty, and surgeries involving the head and neck, thyroid, or esophagus.[6] Most iatrogenic injuries occur in the posterior membranous portion of the trachea, an area lacking cartilaginous reinforcement and therefore more vulnerable to longitudinal splitting under mechanical stress.[6] Endotracheal tube–related injuries classically produce longitudinal tears within the membranous region, affecting both the cervical and thoracic trachea, while

cuff inflation–related injuries tend to occur in the proximal trachea where cuff pressure may be greatest relative to local tracheal wall properties. These distinctions reflect differing mechanisms: direct instrumentation trauma may generate linear shear along the posterior wall, whereas overinflated cuffs can cause pressure necrosis or rupture in a localized region. Risk factors for iatrogenic tracheobronchial tears are commonly categorized into mechanical factors and anatomical factors, a division that is clinically useful because many mechanical factors are modifiable through training, technique, and equipment selection. Mechanical procedural risks include operator inexperience and repeated intubation attempts, which increase the likelihood of inappropriate force application, malposition, or traumatic passage of instruments. Instrumentation-related risks include the use of a stylet during intubation, selection of large-sized tracheal tubes, use of double-lumen tubes, utilization of larger balloon diameters during bronchoplasty, and inadequate tube care after intubation, all of which can increase local pressure, shear stress, or focal trauma.[7] In contrast, anatomical risk factors reflect patient-specific vulnerabilities that may be less modifiable but can often be anticipated. These include tracheal diverticula, distorted airway anatomy, tracheal bronchus, congenital tracheobronchomegaly, female sex, age above 65 years, inhaled steroid use, and an inflamed trachea.[7] Structural anomalies can create points of weakness or unusual angles that predispose to tearing when standard instruments are advanced. Age-related changes may reduce tissue elasticity, while inflammation can weaken mucosa and submucosal layers, increasing susceptibility to injury. A review identified female sex, age over 65 years, and emergency intubations as prominent non-modifiable risk factors, underscoring the clinical importance of heightened caution and technique refinement in emergent airway scenarios and in older female patients.[7] In sum, the etiology of tracheobronchial tears encompasses penetrating injuries—particularly gunshot wounds—blunt trauma mechanisms dominated by motor vehicle collisions and pericarinal shear stress, and a growing category of iatrogenic injuries associated with modern airway interventions.[3][4][5][6][7] Each mechanism yields characteristic anatomic patterns and risk profiles, and recognizing these patterns is central to timely diagnosis and to selecting the safest airway and procedural strategies in emergency and perioperative care.

Epidemiology

Epidemiologic characterization of tracheobronchial tears is intrinsically challenging because the denominator of affected individuals is often unknowable. A substantial proportion of patients who sustain severe tracheobronchial disruption—particularly after high-energy blunt trauma—die at the scene or during transport before they can be systematically evaluated, documented, and included in hospital-based registries. Consequently, incidence estimates derived solely from emergency department admissions or trauma center cohorts tend to underrepresent the true burden of disease and preferentially capture survivors with less extensive injuries or those who benefited from rapid prehospital airway stabilization. This “survivor bias” is a defining limitation of tracheobronchial injury epidemiology and explains why postmortem data remain a crucial complement to clinical series in estimating overall occurrence. In postmortem series, the overall incidence of tracheobronchial injury has been reported to be approximately 2% to 3%, a figure that includes victims who died at the scene and therefore provides a broader perspective on population-level frequency than hospital-only datasets.[8][9] Across trauma mechanisms, penetrating injuries are consistently reported to cause tracheobronchial injuries more frequently than blunt trauma.[2][10] This higher proportional contribution reflects the direct pathway of penetrating objects or projectiles, which can disrupt airway structures even in the absence of substantial external deformation. Within penetrating trauma, the distribution varies by anatomical region. Penetrating neck injuries account for up to 6% of tracheobronchial injuries, whereas penetrating thoracic trauma causes tracheobronchial injury in roughly 1% to 2% of cases. These proportions highlight the vulnerability of cervical airway structures in neck trauma and the relative protection afforded by the thoracic cage, although thoracic penetration can still produce catastrophic injury when the trajectory intersects the trachea, carina, or main bronchi.

Detailed epidemiologic insights into injury patterns have been drawn from long-term studies of airway trauma. A 20-year study on airway structures published in 1992 reported that transverse tears constituted 74% of cases, with 4% located in the cervical trachea and 12% in the intrathoracic trachea, while the remaining transverse injuries involved the right main bronchus, left main bronchus, and lobar bronchi in approximately 25%, 17%, and 16% of cases, respectively.[11] Vertical tears accounted for 18% of injuries in that series, with 6.5% occurring in the cervical trachea, 10% in the intrathoracic trachea, and 1.5% in the main bronchi. The remaining 8% were classified as complex injuries involving the trachea and either the right main bronchus or both main bronchi.[11] These distributions are epidemiologically important because they illustrate that a large fraction of clinically significant injuries occur near central airway bifurcation zones and main bronchi, regions that are vulnerable to shear forces in blunt trauma and that can be technically challenging to repair. The predominance of transverse tears in this series also provides a useful conceptual frame for clinicians, since transverse patterns are consistent with tearing under compressive and deceleration-

related stress, whereas vertical tears often reflect longitudinal splitting of membranous portions under traction or instrumentation-related injury. Population-level epidemiology also suggests that the proportion of iatrogenic tracheobronchial injuries may be substantial in contemporary practice, reflecting the expanding use of airway devices, critical care ventilation, and procedural interventions. A German study conducted over five years and published in 2009 identified 1033 tracheobronchial injuries, of which 41.5% were classified as non-iatrogenic and 58.4% were iatrogenic.[12] Among the non-iatrogenic injuries in that cohort, approximately 64.3% were due to blunt trauma, with the remainder attributed to penetrating wounds, bullet wounds, and miscellaneous etiologies.[12] This distribution does not necessarily imply that iatrogenic injury is more common than traumatic injury in the general population; rather, it reflects the case mix captured within the study setting and the increasing visibility of iatrogenic complications in proceduralized healthcare environments. Nonetheless, the finding underscores that airway tears are not exclusively traumatic events and that procedural airway injury constitutes a meaningful epidemiologic category with its own prevention opportunities and risk stratification needs.

Outcomes data further clarify the epidemiologic significance of mechanism. Survival after blunt trauma is generally lower than after penetrating injury because blunt trauma is more frequently accompanied by multiple severe associated injuries, including pulmonary contusion, major vascular trauma, and traumatic brain injury. In a study of 104 patients with tracheobronchial injury followed over 15 years, overall mortality was reported as 23%, and mortality in blunt injury was approximately twice that observed in penetrating tracheobronchial injury (36% versus 16%).[2] These figures reinforce the clinical observation that blunt tracheobronchial tears often represent a marker of high-energy trauma with systemic physiologic insult rather than an isolated airway event. From an emergency medicine and trauma systems perspective, this distinction is consequential because it emphasizes that mortality risk is driven not only by airway disruption itself but also by the broader injury burden typical of blunt mechanisms. The incidence of tracheobronchial injury among patients presenting to emergency care with blunt trauma has been estimated at 0.5% to 2%.[3] Although this incidence appears low, its clinical significance is amplified by the high mortality risk, the need for rapid recognition, and the possibility of missed diagnosis if persistent pneumothorax, air leak, or subcutaneous emphysema are attributed to more common thoracic injuries. Estimating the incidence of iatrogenic tracheobronchial tears is even more difficult than estimating traumatic incidence because these events may be under-detected, under-reported, or misclassified, particularly when tears are small, clinically subtle, or attributed to underlying disease rather than instrumentation. Nevertheless, available estimates provide a useful epidemiologic anchor. The incidence of tracheobronchial injury with a single-lumen endotracheal tube has been reported at approximately 0.005%, whereas incidence associated with double-lumen tubes ranges from 0.005% to 0.19%.[7][13][14][15] The higher upper bound for double-lumen tubes likely reflects their larger diameter, stiffness, and more complex placement, often in operative settings where precise positioning is required. Posterior tracheal wall perforation has been reported to occur in about 1% of patients during percutaneous dilatational tracheostomy, reflecting the vulnerability of the membranous posterior wall during dilation and cannulation. In the same German study that characterized overall injury etiology, endotracheal intubation and mechanical ventilation accounted for 61.5% of iatrogenic injuries, nearly 30% were related to dilatational tracheostomy, and the remainder were linked to endoscopic interventions.[12] This breakdown suggests that the epidemiology of iatrogenic airway injury is concentrated in high-frequency airway management procedures, particularly those performed in emergent or critical care contexts where patient instability, difficult anatomy, and time pressure may increase risk.

Finally, airway injury is also recognized as a complication in adjacent surgical domains. In esophageal surgeries involving transthoracic and transabdominal approaches, airway injury has been reported in approximately 1% to 1.8% of cases.[16] This epidemiologic observation highlights the proximity of the esophagus to the trachea and main bronchi and the potential for operative dissection, traction, or thermal injury to compromise airway integrity. Taken together, these epidemiologic findings demonstrate that tracheobronchial tears occupy a complex landscape spanning traumatic and iatrogenic mechanisms, with incidence estimates constrained by prehospital mortality and reporting limitations, yet with consistent evidence of significant mortality and substantial procedural relevance across emergency, critical care, and surgical settings.[2][3][8][9][11][12][7][13][14][15][16]

Pathophysiology

The pathophysiology of tracheobronchial tears is determined by both the mechanism of injury and the structural characteristics of the tracheobronchial tree. Anatomically, the trachea consists of cartilaginous rings that provide rigidity and patency, while the posterior wall is membranous and relatively less reinforced. These features create predictable zones of vulnerability when external forces, penetrating objects, or sudden internal

pressure changes exceed the tissue's elastic limits. The clinical consequences of disruption are driven by loss of airway continuity, leakage of air into surrounding compartments, impaired ventilation, and the potential for rapid decompensation through pneumothorax, pneumomediastinum, or progressive respiratory failure. Penetrating trauma most commonly affects the anterior trachea, where cartilaginous rings and the ligamentous spaces between them are exposed to direct insult. When a penetrating object traverses the neck or thorax, it may lacerate cartilage, tear the intercartilaginous ligamentous tissue, or puncture the airway lumen, creating a pathway for air to escape into the soft tissues. Stab wounds—an important subset of penetrating injuries—are almost always confined to the cervical trachea because of the accessibility of the neck and typical injury trajectories. These injuries tend to be relatively localized, yet they can be physiologically profound because even small disruptions can produce extensive subcutaneous emphysema, compromise airway stability, or coexist with vascular injury. Gunshot wounds differ in pathophysiology because the projectile may traverse deeper planes and can disrupt the trachea or bronchi at any point of contact, depending on ballistic trajectory. In addition to direct laceration, projectile energy transfer can produce cavitation and tissue devitalization beyond the visible wound track, increasing the risk of delayed necrosis, expanding air leak, or progressive airway instability [13][14][15][16].

Blunt trauma produces tracheobronchial tears through three principal biomechanical processes: blow, shear stress, and burst mechanisms. In "blow" injuries, direct compressive forces deform the airway against rigid structures such as the vertebral column, generating focal tearing where tissue strain is maximal. Shear stress injuries are particularly important in high-speed deceleration events. When a large amount of energy is applied to the anterior chest, the lungs may be displaced laterally, while the carina remains relatively immobile due to its fixation within the mediastinum. This differential motion creates traction forces that can separate the bronchi from the carina or tear the airway near the bifurcation. The carina is the most fixed portion of the intrathoracic airway system, and this anatomic immobility increases its propensity to sustain injury from shear forces during sudden deceleration. As a result, injuries frequently cluster near the carina and proximal main bronchi, where the transition from fixed to mobile structures concentrates mechanical stress. The "burst" mechanism reflects rapid increases in intraluminal airway pressure, classically occurring when the glottis is closed at the moment of thoracic compression, such as during a crush injury. In this scenario, external compression abruptly raises airway pressure against a closed system, and rupture occurs at the weakest point once intraluminal pressure exceeds tissue elasticity. This mechanism explains why some patients develop central airway disruption even without a penetrating wound, and it also accounts for variability in tear patterns depending on the distribution of pressure and local tissue vulnerability. Collectively, these mechanisms demonstrate that tracheobronchial tears are not random events; they reflect predictable interactions between anatomy, tissue biomechanics, and the direction, magnitude, and timing of applied forces [13][14][15][16].

History and Physical

The clinical presentation of tracheobronchial tears is shaped by the location, extent, and mechanism of injury, yet a defining diagnostic challenge is that many patients initially manifest nonspecific respiratory complaints that overlap with more common thoracic injuries. Although the anatomic site of disruption influences both the predominant symptoms and the most informative diagnostic modality, clinicians must recognize that airway tears can present with subtle or misleading features, especially in the context of multisystem trauma or critical illness. In emergency care, the evaluation therefore depends on integrating mechanism of injury, evolving respiratory status, and a targeted physical examination that actively searches for indirect signs of airway disruption. From the history, patients may describe dyspnea ranging from mild shortness of breath to severe respiratory distress, and symptoms may evolve rapidly as air dissects through tissue planes or as ventilation becomes progressively ineffective. Breathing difficulty may be accompanied by stridor, particularly when injury involves the cervical trachea or laryngeal-adjacent structures, because edema, hematoma, or airway collapse can narrow the proximal lumen and produce high-pitched inspiratory noise. Respiratory failure may occur when airway patency is compromised or when an ongoing air leak prevents adequate ventilation despite supplemental oxygen. Additional symptoms frequently include subcutaneous emphysema, which may be experienced as swelling, "crackling" under the skin, or facial and neck fullness. Voice changes—hoarseness or even aphonia—can occur when cervical airway injury is accompanied by laryngeal trauma, recurrent laryngeal nerve involvement, or mechanical distortion of the glottic apparatus. Hemoptysis is also a key symptom, reflecting mucosal disruption and bleeding into the airway; its severity varies widely, and even small amounts can be diagnostically meaningful when correlated with trauma mechanism and imaging findings. Importantly, patients often exhibit symptoms attributable to associated injuries, such as chest wall pain, dysphagia, altered mental status, or shock, which may divert attention away from the airway as the primary source of pathology [13][14][15][16].

On physical examination, the most commonly reported signs include subcutaneous emphysema and pneumothorax. Subcutaneous emphysema has been described in a substantial proportion of cases, approximately 35% to 85%, while pneumothorax occurs in roughly 20% to 50% of patients. These findings form the basis of classic warning signs that should immediately raise suspicion of tracheobronchial injury in the appropriate clinical context: subcutaneous emphysema, pneumomediastinum, and pneumothorax. Palpation of the neck and chest may reveal crepitus, a crackling sensation caused by air within subcutaneous tissue. Visual inspection may show swelling of the neck, supraclavicular area, or chest wall, sometimes extending to the face. In the thorax, reduced breath sounds or hyperresonance may suggest pneumothorax; however, these findings can be confounded by concomitant pulmonary contusion, hemothorax, or pain-limited respiratory effort. The pattern of air distribution provides important anatomic clues. Injury to more proximal airway structures—such as the thoracic trachea or large central bronchi—often presents with pneumomediastinum accompanied by subcutaneous air, because leaked air accumulates first within the mediastinum and then tracks into cervical and subcutaneous planes. In contrast, more distal bronchial injury may preferentially result in pneumothorax with subcutaneous emphysema, reflecting direct communication of air into the pleural space. Auscultation may reveal a distinctive "mediastinal crunch" synchronized with the heartbeat, a phenomenon associated with mediastinal emphysema. This finding, while not universally present, is highly suggestive when observed and should prompt urgent evaluation for central airway injury and other causes of pneumomediastinum. A particularly important clinical scenario is persistent air leak or a non-resolving pneumothorax after placement of an intercostal chest tube. In trauma settings, pneumothorax is common and is often treated effectively with tube thoracostomy. When the pneumothorax does not improve, or when continuous bubbling indicates a large ongoing air leak, clinicians should consider tracheobronchial tear as a possible source. This is because a major airway disruption can allow a large volume of air to escape into the pleural space, overwhelming the ability of chest drainage to re-expand the lung. Recognition of this pattern is crucial because repeated tube placements or escalating ventilator pressures may worsen the air leak, while definitive management requires airway stabilization and often surgical or bronchoscopic intervention [13][14][15][16].

Less common but diagnostically relevant findings may also occur. Pneumoperitoneum has been reported after blunt chest injuries involving rupture of the trachea and right main bronchus. In these cases, mediastinal air is thought to dissect into the abdomen through the transesophageal or aortic hiatus, creating free intraperitoneal air that might otherwise suggest hollow viscus perforation. Awareness of this pathway is important because it can broaden the differential diagnosis of pneumoperitoneum in trauma and prevent misattribution of the finding to abdominal organ injury alone. In iatrogenic contexts, history may reveal post-extubation respiratory symptoms as the first sign of an unrecognized tracheal tear. A tear may initially be sealed by the inflated endotracheal tube cuff, masking symptoms and limiting air leak. Once the patient is extubated and the cuff is removed, the defect can reopen, leading to sudden onset of dyspnea, stridor, subcutaneous emphysema, or voice change. This temporal relationship—relative stability while intubated followed by deterioration after extubation—should heighten suspicion for cuff-related masking of airway injury. In penetrating neck trauma, a classic observation is air leakage from the neck wound that disappears after intubation, reflecting temporary sealing of the airway defect by the tube or cuff. This finding, when present, is strongly suggestive of tracheal injury and underscores the importance of careful airway management and post-intubation reassessment. Overall, the history and physical examination in suspected tracheobronchial tear demand both broad vigilance and focused interpretation of key signs. Because initial symptoms can be nonspecific, clinicians must actively look for patterns—subcutaneous emphysema, pneumomediastinum, pneumothorax, persistent air leak, and post-extubation deterioration—that collectively signal possible airway disruption and justify expedited imaging, bronchoscopy, and definitive management planning [13][14][15][16].

Evaluation

Evaluation of suspected tracheobronchial tear requires a deliberate balance between rapid stabilization and targeted diagnostics, because these injuries are uncommon, frequently coexist with multisystem trauma, and can deteriorate quickly if not recognized early. A definitive diagnosis is often established through a combination of clinical findings at the initial assessment, direct visualization during surgical exploration, or bronchoscopic confirmation using fiberoptic techniques in cases where suspicion is high. Nevertheless, tracheobronchial injuries may be missed in a substantial proportion of patients—reported in the range of 25% to 68%—particularly when presentations are dominated by more obvious thoracic injuries, when imaging findings are subtle, or when temporary sealing of a small defect reduces early radiographic clues. For this reason, imaging modalities play a critical supportive role by identifying indirect signs that heighten suspicion and guide timely bronchoscopy and surgical consultation, especially in chest and neck trauma. Plain chest radiography is typically the initial imaging study in

many trauma evaluations, yet it has limited sensitivity for tracheobronchial disruption. Chest radiographs may appear normal in approximately 10% of patients, particularly when the tear does not extend beyond peritracheal or peribronchial connective tissue or when a minor injury is temporarily sealed by fibrin formation.[17] This possibility is clinically important because reliance on a "normal" radiograph can create false reassurance in patients whose symptoms and mechanism suggest central airway injury. When abnormalities are present, they often reflect the distribution of air escaping from the airway and the relationship of the injury to the mediastinal pleura. The most common site of tracheobronchial tear is at the level of the carina, a region where the mediastinum is relatively fixed and thus vulnerable to shear forces during sudden deceleration. Tears that remain within the mediastinal pleura tend to manifest as pneumomediastinum, whereas tears that extend beyond the mediastinal pleura are more likely to produce pneumothorax. This distinction is clinically helpful when interpreting radiographs in context: pneumomediastinum with extensive subcutaneous emphysema may point toward a more proximal injury, while a large pneumothorax—particularly if persistent—raises concern for communication between the airway and pleural space.

Radiographic patterns may also vary by the bronchus involved. Right main bronchial injury is more often expected to produce a pneumothorax, whereas left main bronchus injury may be more likely to produce pneumomediastinum because the left main bronchus courses a longer distance within the mediastinum before entering the lung parenchyma. Such anatomical considerations help clinicians interpret whether intramediastinal air predominates over pleural air and can refine suspicion toward specific injury locations. One of the most characteristic but less frequently encountered signs is the "fallen-lung sign," seen in severely injured patients when partial or complete detachment of a main bronchus allows the lung to shift into a dependent lateral position. This differs from typical pneumothorax physiology in which the lung collapses toward the hilum; in a fallen-lung configuration, loss of bronchial tethering and disruption of normal anatomic attachments permits abnormal lung displacement, signaling a high likelihood of major bronchial disruption. Additional radiographic clues are often indirect but clinically meaningful when integrated with the patient's presentation. Classic descriptions include incorrect positioning of an endotracheal tube, overdistension of the endotracheal cuff, and a persistent pneumothorax after intercostal tube placement. A chest tube that fails to resolve pneumothorax or that demonstrates continuous large air leak should prompt reconsideration of the underlying source, including tracheobronchial rupture, rather than assuming simple parenchymal leak. In cervical injuries, abnormal elevation of the hyoid bone above the third cervical vertebra has been described and may occur due to contraction and upward pull of suprahyoid musculature when a cervical tracheal tear results in rupture of the infrahyoid muscles. While this is not a universally observed sign, it reflects how soft-tissue and muscular disruption can generate recognizable radiographic displacement patterns. Among the radiologic indicators, however, the most specific signs for tracheobronchial tear are the presence of an endotracheal tube positioned beyond expected tracheal anatomy and the fallen-lung sign, both of which suggest major structural discontinuity rather than nonspecific air leak [17].

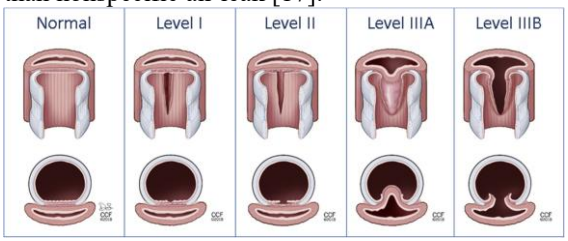

Fig. 2: Tracheobronchial injuries.

Computed tomography (CT) has become the primary noninvasive imaging modality for suspected tracheobronchial injury because it offers superior sensitivity and anatomic delineation compared with plain radiography and is widely available in trauma centers. CT is considered the noninvasive investigation of choice for evaluating suspected tracheobronchial tears, as it can identify subtle pneumothoraces, mediastinal emphysema, and even active bleeding related to airway injury. A particularly helpful CT finding is air within the wall of the trachea or bronchus, which supports the presence of a tear and indicates dissection of air into tissue planes. CT also provides the major advantage of simultaneously evaluating associated injuries—such as damage to the heart and great vessels—thereby supporting comprehensive trauma assessment and prioritization. Advanced CT techniques, including multiplanar reformats (MPR), three-dimensional reconstructions along the long axis of the airway, and virtual endoscopy, can increase diagnostic clarity by allowing clinicians to "trace" the airway and visualize suspected defects more precisely. These reconstructions can be particularly valuable when the injury is near the carina or main bronchi, where complex anatomy and overlapping mediastinal structures may obscure details on standard axial slices. Despite its advantages, CT is not infallible. Subtle injuries can yield falsely negative CT findings, especially when tears are small, partially sealed, or primarily involve the membranous wall without dramatic air escape. In such circumstances, clinical suspicion must guide next steps, and fiberoptic bronchoscopy may provide definitive visualization and clarify whether an injury exists. This diagnostic escalation reflects a key principle in airway trauma:

imaging should support, not replace, clinical reasoning. When clinical patterns strongly suggest a tracheobronchial tear—such as persistent pneumothorax, extensive subcutaneous emphysema, or unexplained pneumomediastinum—bronchoscopy should not be delayed solely because CT findings are equivocal [17].

Magnetic resonance imaging (MRI) offers theoretical benefits, including multiplanar display capabilities, superior soft-tissue contrast, avoidance of ionizing radiation, and reduced risk of contrast-induced nephropathy when contrast is not required. However, in the acute trauma context, these advantages are typically outweighed by logistical and safety constraints. MRI requires preparation and longer scanning times, and monitoring unstable trauma patients in the MRI environment is more complex, potentially increasing risk. Furthermore, MRI availability is variable across institutions, and time-sensitive airway evaluation often demands faster modalities. For these reasons, MRI is not routinely used as a primary diagnostic study for suspected tracheobronchial tear in emergency settings. Nuclear imaging does not meaningfully contribute to the diagnosis of tracheobronchial injury, particularly in minor tears, because it lacks the anatomic resolution required to identify airway wall defects. However, physiologic studies may demonstrate indirect consequences of airway obstruction. If partial or complete airway obstruction occurs with intact vascular supply, ventilation-perfusion (V/Q) mismatch may be present, accompanied by a physiological reduction in perfusion to poorly ventilated lung regions. While such findings can reflect functional impairment, they are nonspecific and do not localize or define the airway injury with sufficient precision to guide acute management. Angiography similarly has a limited direct role in diagnosing airway tears and becomes useful primarily when there is suspicion of active bleeding or when assessment of associated vascular injuries is required. In that context, angiography may help delineate vascular damage that accompanies penetrating trauma or high-energy blunt mechanisms, informing operative planning and hemostatic strategies. Ultimately, fiberoptic bronchoscopy is regarded as the gold standard for detection of tracheal tear or tracheobronchial injury.[17] Its value lies in direct visualization of the airway mucosa and lumen, allowing clinicians to locate the injury, assess its extent, and determine whether there is partial thickness disruption, full thickness rupture, or complete transection. Beyond diagnosis, bronchoscopy can contribute to initial airway management by enabling guided placement of endotracheal tubes distal to the injury, helping isolate ventilation to an intact bronchial segment, and reducing the risk of worsening the tear through blind instrumentation. In suspected or confirmed cases, bronchoscopy therefore serves both diagnostic and therapeutic purposes, functioning as a pivotal tool in the emergency and operative pathway for patients with tracheobronchial tears.[17]

Treatment / Management

The management of tracheobronchial tear is anchored in one overriding priority: establishing and maintaining a safe airway while minimizing further disruption of injured tracheobronchial structures. Because these injuries can rapidly progress to catastrophic ventilatory failure, the earliest decisions—often made under time pressure in the emergency department or trauma bay—frequently determine outcomes. A major clinical challenge is that tracheobronchial tears are not always suspected at the time airway control is first considered. Patients may arrive with respiratory distress, hypoxemia, altered mental status, or hemodynamic instability that prompts urgent intubation, and the default reflex in many resuscitations is rapid sequence intubation. However, when an airway tear is present, conventional rapid sequence techniques can be hazardous. Blind or forceful advancement of an endotracheal tube through a disrupted airway may extend the laceration, create a false passage, worsen air leak into the mediastinum or pleural space, and precipitate complete loss of airway continuity. For this reason, when tracheobronchial injury is suspected, maintaining spontaneous breathing is generally preferred until a controlled airway strategy is established, because spontaneous ventilation reduces peak airway pressures and limits the risk of enlarging the defect. This management principle aligns with guidance such as the disrupted-airway algorithm highlighted in an American Society of Anesthesiologists newsletter, which emphasizes preserving spontaneous respiration until a safe airway is definitively secured. In stable or partially stable patients, fiberoptic bronchoscopic intubation is widely regarded as the ideal initial technique because it permits direct visualization of airway anatomy and injury, facilitates suctioning of blood clots and secretions, and enables deliberate placement of the endotracheal tube with the cuff positioned distal to the injured segment. This cuff positioning is crucial: if the cuff sits within or proximal to the tear, it can worsen disruption, fail to seal the airway, and allow ongoing air leak. Fiberoptic bronchoscopy also offers the practical advantage of avoiding neck extension, making it particularly appropriate when cervical spine injury is suspected or cannot be excluded. Moreover, when the tear involves the carina or a main bronchus, bronchoscopy can guide selective intubation into the uninjured bronchus, enabling single-lung ventilation as a temporizing strategy. This approach can stabilize oxygenation and ventilation while definitive repair is planned. Nonetheless, fiberoptic bronchoscopy has limitations. Visualization may be poor in the setting of active

bleeding, and distal airway collapse can prevent adequate endoscopic navigation. Additionally, the technique may be difficult to perform in restless, uncooperative, or profoundly unstable trauma patients, particularly when sedation must be minimized to preserve spontaneous breathing and cardiovascular stability [18].

When fiberoptic techniques are not feasible or are compromised by blood and secretions, rigid bronchoscopy performed in the operating theater under inhalational anesthesia can be preferable. Rigid bronchoscopy provides superior airway control, allows more effective removal of clots and secretions, and can serve as a conduit for ventilation during airway interventions. In select circumstances, rigid endoscopic measures may also provide temporary stabilization of the airway defect, including endoscopic "patching" strategies, although these typically function as bridges rather than definitive solutions. The major drawback is that rigid bronchoscopy generally requires neck extension and is therefore contraindicated when cervical spine injury is suspected, underscoring the need to tailor airway strategy to the full trauma context rather than to the airway injury alone. In the most severe cervical injuries—particularly complete transection involving the larynx and proximal trachea—transoral intubation may be impossible or may exacerbate tissue disruption. In such scenarios, immediate surgical airway access may be lifesaving. If there is an open cervical wound that exposes the disrupted airway, the tracheal segment may be grasped directly and a tracheostomy tube introduced through the injured lumen to re-establish ventilation. When no usable pathway exists, an emergency bedside open tracheostomy may be required. Surgeons may aim to pass the tracheostomy tube through the injured trachea in a way that preserves tracheal length and facilitates later reconstruction. Once ventilation is stabilized, clinicians must rapidly assess for concomitant injuries given the cervicothoracic region's proximity to critical structures. The overall survival of these patients is often driven by the severity of associated trauma. Therefore, evaluation must specifically consider injuries to the vocal cords, esophagus, vertebral bodies and spinal cord, lungs and pleura, ribs, sternum and clavicle, carotid arteries, jugular veins, and the great vessels [18].

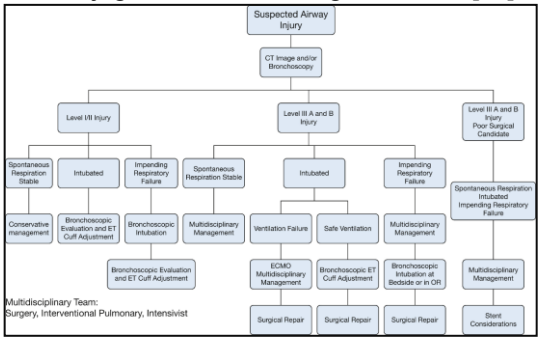

Fig. 3: Treatment of Tracheobronchial injuries.

Ventilator management in confirmed airway injury requires restraint. High airway pressures and excessive positive end-expiratory pressure (PEEP) can worsen air leakage, promote mediastinal dissection, and compromise the integrity of an evolving repair. If the tear extends to the carina or involves a main bronchus, single-lung ventilation with endoscopic tube placement into the uninjured bronchus can reduce the air leak and stabilize gas exchange. In cases where adequate oxygenation and ventilation cannot be achieved despite optimal positioning and conservative ventilator settings, extracorporeal membrane oxygenation (ECMO) should be considered as a bridge to recovery or to definitive repair, whether surgical or bronchoscopic. ECMO can reduce the need for injurious airway pressures and provide time for multidisciplinary planning in complex injuries. Definitive management most often involves surgical repair, and historically the vast majority of tracheobronchial injuries have been managed operatively. Conservative management, intended to allow spontaneous healing, is associated with the risk of late airway stenosis due to scar formation and remodeling. When late stenosis is identified bronchoscopically, dilation may provide temporary relief by reopening the lumen and facilitating secretion clearance, but definitive surgical correction is often delayed for four to six months to allow scar maturation. In such cases, complex reconstructive procedures, such as bronchial sleeve resection with a pedicled tissue flap, may be required. In cervical injuries, laryngeal and vocal cord damage must be carefully assessed and managed prior to or in conjunction with tracheal repair, because functional outcomes depend on preserving phonation and airway protection mechanisms. Despite the primacy of surgery, there has been a notable shift toward minimally invasive options—particularly endoscopic stent placement—in select patients, especially those with iatrogenic injuries or those at high operative risk.[18][15]

Decisions regarding acute surgical repair are commonly guided by the risk of airway obstruction, the magnitude of air leak, and the risk of mediastinal infection.[19] Many experts favor surgical repair for tears exceeding 4 cm, reflecting concern that larger defects are unlikely to seal reliably and carry higher risk of complications. Emergency surgery is generally indicated when there is esophageal prolapse into the tracheobronchial lumen, inability to ventilate, or discovery of a tear intraoperatively. Conversely, a conservative approach is often considered when the tear is less than 2 cm. For intermediate tears measuring approximately 2 to 4 cm, management is individualized based on clinical stability, comorbidities, tear location and geometry, associated injuries, and the availability of specialized expertise. Some clinicians advocate conservative management for patients who are self-ventilating or require only minimal ventilatory support and who have no

evidence of mediastinal or esophageal injury. This selective conservatism is particularly applicable to iatrogenic tears, which are often single, linear lacerations in contrast to the more complex and destructive tears observed in blunt or penetrating trauma. Nonetheless, even in iatrogenic cases, surgical intervention is warranted when conservative management fails, evidenced by worsening pneumomediastinum, progressive subcutaneous emphysema, persistent pneumothorax with ongoing air leak, or failure of lung re-expansion despite adequate tube thoracostomy. Certain posterior membranous wall lacerations after iatrogenic events—such as endotracheal intubation or percutaneous tracheostomy—have been successfully managed conservatively under stringent conditions.[20][21][20][19] These conditions include minimal and nonprogressive pneumomediastinum, absence of mediastinitis, feasibility of evacuating pneumothorax without a persistent large air leak, absence of esophageal injury, ability to maintain ventilation, and the ability to position the endotracheal cuff away from the injury while ventilating with minimal PEEP. This carefully defined physiologic and anatomic profile reflects the underlying goal of conservative therapy: prevent ongoing contamination and mechanical stress at the tear site while allowing tissue edges to approximate and heal.

When surgery is undertaken, several principles guide operative success. Optimal exposure is required to accurately identify tear margins and associated injuries. Debridement should be conservative, preserving maximal airway length for future reconstruction. Preservation of the laterally oriented tracheal blood supply is emphasized to reduce ischemia and anastomotic failure. Repairs are typically buttressed, and tracheostomy is considered, especially in polytrauma patients, to secure airway access and reduce mechanical stress on the repair. Cervical tracheal repair often begins with a collar incision and anterior mobilization of the trachea toward the carina, performed carefully to avoid damaging lateral blood supply. Suture technique is also relevant: placing knots outside the tracheal lumen reduces the likelihood of intraluminal granuloma formation that could later narrow the airway. Postoperative tracheostomy is frequently required due to the high incidence of unilateral or bilateral recurrent laryngeal nerve injury, especially in incomplete transections below the cricoid cartilage. Despite this, low-tension anastomosis is often achievable with meticulous mobilization. When there is associated esophageal injury, surgical sequencing generally prioritizes esophageal repair first with flap interposition, followed by tracheal repair at the end, to reduce contamination and to provide tissue separation between suture lines. Intrathoracic tracheal and bronchial repairs often require thoracotomy, with incision side determined by injury location. A right fourth intercostal thoracotomy provides access to the lower trachea, carina, and right main bronchus, whereas a left fourth intercostal thoracotomy is used for the left main bronchus. In patients who cannot tolerate thoracotomy positioning due to spinal injuries, a transsternal approach via median sternotomy may be used for lower tracheal and carinal injuries. Regardless of approach, bronchoscopy should be performed before leaving the operating suite to confirm repair integrity, airway patency, and appropriate endotracheal tube positioning. In intubated patients, it is imperative that the endotracheal cuff is not positioned adjacent to the repair site, as cuff pressure can compromise healing and precipitate dehiscence.

To guide non-surgical management in iatrogenic injuries, Cardillo and colleagues proposed a morphologic classification of tracheal wall injuries during endotracheal intubation, which some experts have extrapolated to other iatrogenic scenarios.[22][15] In this framework, level 1 represents mucosal or submucosal injury, level 2 involves deeper extension into the muscular wall with associated subcutaneous or mediastinal emphysema, level 3A denotes complete laceration with herniation of esophageal or mediastinal soft tissue, and level 3B includes these findings plus esophageal injury or mediastinitis. Levels 3A and 3B are regarded as clear indications for surgery, whereas level 1 can be managed conservatively and level 2 is individualized. Although broad-spectrum antibiotics are often administered for approximately one week in conservatively managed patients, robust clinical data supporting this practice remain limited, while standard perioperative antibiotic protocols apply in surgical cases. Additional classification systems, such as Schaefer-Fuhrman for laryngotracheal trauma, have also been applied to identify candidates for nonoperative management. Debate persists, with most experts favoring primary surgical repair as the definitive treatment, while others report no meaningful difference between surgical and conservative approaches in carefully selected iatrogenic injuries.[23][18] For patients who are poor surgical candidates due to multiple comorbidities—including moderate to severe cardiopulmonary disability—endoscopic placement of self-expandable metallic stents (SEMS) can serve as a valuable alternative or bridge. These stents mechanically cover the defect and promote granulation tissue formation that helps seal the injured area. Endoscopic reevaluation is typically performed after several weeks, and stents may be removed after approximately 4 to 6 weeks if healing is adequate. In selected cases, stenting stabilizes the airway sufficiently to permit delayed surgery under improved physiologic conditions. Adjunctive

techniques have also been reported, such as endobronchial glue injection combined with stenting for small lacerations less than 5 mm.[24] Endoluminal repair strategies may be considered in patients who can tolerate specialized ventilation approaches, such as jet insufflation, and who have preserved posterior wall attachments that allow stable endoscopic manipulation.[6] In summary, management of tracheobronchial tears requires a staged and mechanism-sensitive approach: early controlled airway stabilization with preservation of spontaneous breathing when feasible; prompt use of bronchoscopy as both diagnostic and airway-guiding tool; careful ventilator strategies that minimize airway pressures; comprehensive assessment for associated cervicothoracic injuries; and definitive repair through surgery in most traumatic cases, with selective conservative or endoscopic strategies reserved for carefully chosen iatrogenic injuries and high-risk surgical candidates.[18][15][19][20][21][22][23][24][6]

Differential Diagnosis

The differential diagnosis of suspected tracheobronchial tear is broad because the hallmark clinical and radiologic findings—pneumomediastinum, subcutaneous emphysema, and pneumothorax—are not specific to tracheal or bronchial disruption. These findings represent downstream manifestations of air escaping from an anatomic compartment where it is normally contained, and multiple structures within the aerodigestive tract and thorax can serve as the source. Consequently, clinicians must interpret these signs in the context of mechanism of injury, clinical trajectory, airway symptoms, and associated imaging patterns, while maintaining an organized approach to ruling out alternative causes that require different urgent interventions. First, injuries to the larynx can mimic tracheobronchial tear because laryngeal fractures, mucosal lacerations, or glottic disruption may produce subcutaneous emphysema and airway compromise, including stridor and hoarseness. In such cases, air dissects into cervical fascial planes and can extend into the mediastinum, generating pneumomediastinum without necessarily involving the intrathoracic trachea or bronchi. Second, pulmonary parenchymal injury is a highly common alternative explanation in trauma. Alveolar rupture from pulmonary contusion, barotrauma, or direct lung laceration can produce pneumothorax and subcutaneous emphysema. Air from ruptured alveoli can also track along bronchovascular sheaths into the mediastinum (a mechanism often invoked to explain pneumomediastinum in blunt trauma), making pneumomediastinum an overlap finding rather than a definitive indicator of central airway tear. Third, esophageal injury is a critical competing diagnosis because esophageal perforation can produce pneumomediastinum, mediastinal inflammation, and systemic toxicity. Differentiating esophageal perforation from tracheobronchial disruption is essential because delays in diagnosing esophageal injury can rapidly lead to mediastinitis, sepsis, and high mortality, and management priorities may differ [20].

Air may also enter thoracic compartments from external sources. Chest wall trauma, including open pneumothorax or penetrating wounds, can introduce air directly from outside into the pleural space and soft tissues, producing subcutaneous emphysema and pneumothorax without primary airway disruption. In addition, air can spread transdiaphragmatically from the abdomen into the thorax and mediastinum, particularly in the presence of pneumoperitoneum or abdominal hollow viscus perforation. This pathway can create pneumomediastinum and subcutaneous emphysema patterns that resemble thoracic airway injury, especially in polytrauma patients where multiple cavities may be affected simultaneously. Given that blunt and penetrating mechanisms contribute significantly to tracheobronchial tears, it is also crucial to consider the broader constellation of concomitant cervicothoracic injuries that can coexist and dominate the clinical picture. In neck and chest trauma, clinicians must carefully evaluate for vascular injuries involving the carotid arteries, jugular veins, aorta, and pulmonary trunk, as well as injuries to neurologic structures such as the recurrent laryngeal and vagus nerves. These associated injuries are not merely coexisting findings; they can be the principal drivers of shock and rapid decompensation and may require immediate intervention independent of airway repair. Moreover, complications arising from these injuries—including hemorrhagic shock, mediastinal infection, aspiration, pneumonia, atelectasis, bronchiectasis, stroke, and progressive multiorgan dysfunction—can significantly increase mortality in patients with suspected or confirmed tracheobronchial injury. Therefore, differential diagnosis in this setting is not limited to identifying an alternate source of air leak; it also entails comprehensive assessment for life-threatening associated injuries that may masquerade as, exacerbate, or complicate a tracheobronchial tear [20].

Prognosis

Prognosis after a tracheobronchial tear is determined by an interplay of patient factors and injury-specific variables, with outcomes ranging from complete recovery to fatal decompensation. Clinically, the most important determinants include the patient's physiologic condition at presentation, the severity and anatomic extent of airway disruption, the time elapsed before recognition, the burden of concomitant injuries, and the definitive management strategy used. Blunt trauma carries an especially grave prognosis at the population level because many patients do not survive long enough to reach hospital care. It has been estimated that nearly 80% of patients

with blunt trauma–associated tracheobronchial injuries die before arriving at a medical facility, a statistic that reflects not only airway compromise but also the lethality of associated injuries such as major vascular disruption, traumatic brain injury, or severe pulmonary contusion. In survivors, however, prognosis has improved markedly over time, reflecting advances in prehospital care, trauma systems, imaging, airway management, and surgical techniques. Mortality in traumatic tracheobronchial injury has decreased substantially from approximately 36% prior to 1950 to around 9% by 2001.[25] This trend highlights how early recognition, safer airway strategies, and prompt access to specialized surgical repair can shift outcomes, even for injuries once considered uniformly fatal. Despite these improvements, specific conditions remain strongly associated with worse outcomes. Superimposed mediastinitis represents a major prognostic inflection point, as infection within the mediastinum can progress rapidly to sepsis and multiorgan dysfunction. Delayed diagnosis, especially when surgical correction is postponed beyond the optimal window, also increases mortality, partly because persistent air leak and contamination foster inflammation and infection and partly because prolonged ventilatory instability increases physiologic stress. Surgical repair performed in critically ill patients carries very high risk; mortality as high as 80% has been observed in critically ill patients undergoing repair, underscoring that physiologic reserve at the time of intervention is often as important as the technical success of reconstruction. Demographic factors have also been examined. A systematic review by Minambres et al. (2009) identified male sex as being associated with increased mortality risk in tracheobronchial injuries, although this association may reflect differences in mechanism, injury severity, and the etiologies leading to respiratory failure rather than sex being a direct biologic determinant of outcome.[7] Long-term prognosis also depends on surveillance and management of late sequelae. Even after successful acute stabilization and repair, patients require follow-up to detect complications such as airway stenosis, malacia, or recurrent infection. Because stenosis may develop during healing, periodic bronchoscopic assessment can be necessary until complete recovery is confirmed. This follow-up carries substantial health-system costs, particularly for patients requiring repeated therapeutic dilations, endoscopic toileting of secretions, or staged reconstructive procedures. Ultimately, favorable prognosis is most likely when diagnosis is early, contamination and air leak are controlled, ventilation can be maintained with low airway pressures, and definitive repair is performed before mediastinal infection and systemic decompensation develop [7][21].

Complications

Complications of tracheobronchial tear are diverse and reflect both the local consequences of airway disruption and the systemic consequences of trauma and critical illness. The type and severity of the injury strongly influence the complication profile. In blunt trauma, the epidemiologic reality is that most patients die at the scene or during transport; this high early mortality is compounded by severe concomitant injuries, which may independently be fatal. Among those who survive, presentations may range from small, initially asymptomatic lesions to profound respiratory distress and respiratory failure. A major immediate risk is sudden airway collapse, which can occur when structural integrity is lost and the airway lumen becomes unstable under negative inspiratory pressure or positive-pressure ventilation. Air leak syndromes constitute both common presenting features and ongoing complications. Subcutaneous emphysema, pneumomediastinum, and pneumothorax are frequent manifestations of tracheobronchial disruption and can progress to hemodynamic compromise, especially when tension physiology develops. Persistent pneumothorax despite chest tube placement, massive air leak, and failure of lung re-expansion are particularly concerning and may signal major bronchial disruption. Infectious complications further amplify risk. Contamination of the mediastinum or persistent atelectasis can lead to pneumonia, mediastinitis, sepsis, and multiorgan dysfunction, all of which markedly worsen prognosis. Chronic or prolonged lobar collapse may result in irreversible bronchiectasis and significant loss of pulmonary reserve, limiting long-term functional status even after airway continuity is restored [21].

Iatrogenic injuries have their own complication spectrum. These tears may extend into adjacent structures, including the esophagus, and can precipitate life-threatening mediastinitis if not recognized and managed promptly. Untreated tears often heal with fibrosis and late stenosis. Cuff-related injuries can begin as superficial mucosal lacerations but may progress to clinically significant stenosis or, in severe cases, tracheoesophageal fistula formation. Surgical repair also carries risks, including anastomotic separation (dehiscence), strictures, and fistula formation. Hemorrhagic catastrophes, such as vascular fistulas leading to sudden life-threatening bleeding, are rare but feared complications in the postoperative period. Recurrent laryngeal nerve palsy can result in persistent hoarseness, aspiration risk, and impaired airway protection. Endoscopic stenting, while increasingly used in selected patients, can introduce complications such as infection, stent migration, granulation tissue formation causing secondary stenosis, metal fatigue, and mucus plugging with acute obstruction. A crucial systems-level complication is missed diagnosis. Approximately 50% to 70% of tracheobronchial tears

may be missed initially, and delayed recognition can lead to late presentations with dyspnea, stridor, and recurrent infections. Importantly, mortality is higher in patients whose injuries are missed early and who later require surgery, likely reflecting progression to infection, scarring, and physiologic deterioration before definitive management [21].

Postoperative and Rehabilitation Care

Postoperative care after tracheal repair is directed at protecting the anastomosis, preventing infection, and restoring functional breathing, phonation, and swallowing. After surgical repair of tracheal injuries, the neck is often maintained in the Pearson position—flexion fixation—for approximately 7 to 14 days to reduce tension across the repair site and minimize the risk of anastomotic stretching or dehiscence. Ventilatory strategy remains critical: the cuff of the endotracheal tube should not overlie the area of repair, because cuff pressure can compromise mucosal perfusion and disrupt healing. When intubation must continue, careful tube positioning and frequent reassessment are required to ensure the cuff remains distal or otherwise safely positioned relative to the anastomosis. Prophylactic antibiotics are commonly administered for at least a week, reflecting concern for mediastinal contamination and postoperative infection. Beyond infection control, aggressive pulmonary hygiene is essential. Pain control, chest physiotherapy, suctioning, and early mobilization help reduce atelectasis and pneumonia risk. In patients with laryngeal involvement or recurrent laryngeal nerve injury, multidisciplinary rehabilitation is often required. Speech and swallow therapy may be necessary to reduce aspiration risk, restore safe oral intake, and address dysphonia. If a tracheostomy is present, coordinated tracheostomy care, humidification, secretion management, and a structured decannulation plan are needed. Longitudinal follow-up typically includes surveillance bronchoscopy or imaging when clinically indicated, particularly to detect stenosis or granulation tissue formation. Early recognition of narrowing can permit less invasive management (e.g., dilation) before advanced scarring mandates complex reconstruction. Thus, postoperative and rehabilitation care is not merely supportive; it is a preventive strategy aimed at avoiding secondary morbidity and preserving long-term airway function [21].

Consultations

Management of tracheobronchial injury requires early, coordinated multidisciplinary involvement, because the clinical priorities span airway control, trauma resuscitation, definitive repair, and long-term functional recovery. Emergency clinicians often provide initial evaluation and stabilization; maintaining a high index of suspicion is essential to prevent missed injury. Airway management frequently requires specialized expertise from anesthesia, pulmonology, or otorhinolaryngology to perform fiberoptic bronchoscopic intubation and to position the tube distal to the injury. In a subset of unstable, uncooperative patients with profuse airway bleeding, rigid bronchoscopy under inhalational anesthesia may be required to clear blood and secretions and to perform bridging measures, while maintaining spontaneous ventilation when possible. Intensive care consultation is integral, as many patients require ventilatory optimization, hemodynamic support, and continuous monitoring for evolving pneumothorax, mediastinal complications, and sepsis. Cardiothoracic surgery and otorhinolaryngology consultations are commonly necessary to evaluate injury extent and determine the need and approach for surgical repair—cervical exploration versus thoracotomy or sternotomy—while simultaneously addressing associated neck or thoracic injuries. Post-discharge, follow-up by thoracic surgery or ENT is often needed to monitor for stenosis, voice changes, swallowing dysfunction, and other late sequelae. Interventional pulmonology involvement is particularly important in patients managed conservatively or with endoscopic stenting, as these patients require bronchoscopic reassessment, stent surveillance and removal planning, and management of granulation tissue or mucus impaction. Effective consultation is therefore not episodic; it is continuous coordination across acute, perioperative, and longitudinal phases of care [21].

Patient Education

Prevention and education operate at two levels: reducing traumatic incidence and minimizing iatrogenic risk. In trauma systems, improving provider awareness—including paramedics trained in advanced trauma life support principles—can reduce mortality through earlier recognition of airway compromise, careful ventilation strategies, and timely transport. Engineering interventions such as consistent seatbelt use and airbag deployment reduce the severity of blunt motor vehicle injuries, forming a cornerstone of population-level prevention. For high-risk activities such as motorcycling and mountain biking, head and neck protection measures are critical to reducing cervical trauma patterns that can compromise the airway. Mental health interventions also play a role in mitigating self-harm–related airway injuries, highlighting that prevention extends beyond mechanical safety to psychosocial care. For iatrogenic deterrence, emphasis is placed on procedural competency and equipment choices. Use of high-volume, low-pressure cuffs can reduce localized tracheal trauma in intubated patients. Proper tube sizing, careful use of stylets and bougies, avoidance of repeated traumatic attempts, and strict prevention of cuff overinflation can reduce the likelihood and severity of tracheal injury during intubation. For difficult airways or suspected airway injury, early use of fiberoptic bronchoscopy is protective because it reduces blind manipulation,

limits further mucosal trauma, and prevents creation of life-threatening false passages. Surgical teams also benefit from structured training and adherence to precautions during head and neck operations, including cautious use of cautery in peritracheal planes. In esophageal malignancy resections, routine preoperative or intraoperative bronchoscopy can help identify airway infiltration risk and reduce inadvertent injury. Patient and family education should address both immediate and delayed complications. Individuals undergoing high-risk iatrogenic interventions should be counseled on warning symptoms such as progressive neck swelling, dyspnea, voice changes, fever, and persistent cough, which may indicate occult airway injury or infection. Similarly, survivors of blunt or penetrating airway trauma should understand that late stenosis or recurrent infections can occur and that follow-up evaluations are essential. Clear education improves early re-presentation for care when complications develop and supports shared decision-making regarding surveillance and rehabilitation [22][23][24].

Other Issues

Several clinical "pearls" consistently emerge in tracheobronchial injury care. A major one is that up to 50% to 70% of injuries may be missed initially, and delayed diagnosis with later surgical repair is associated with higher mortality. This reality mandates a high index of suspicion in all trauma patients—particularly those with chest and neck injuries, persistent air leak, non-resolving pneumothorax, extensive subcutaneous emphysema, or unexplained pneumomediastinum. Another key principle is that most traumatic tracheobronchial injuries ultimately require surgical management, yet there is a growing trend toward conservative or semi-conservative strategies for select iatrogenic posterior wall tears, provided strict criteria are met and bronchoscopy-guided airway positioning is feasible. Endoscopic stenting and other interventional pulmonary approaches can serve as definitive therapy in some high-risk patients or as bridging therapy when surgery is temporarily unsafe. Importantly, even when minimally invasive approaches succeed acutely, definitive surgical repair may still be required later, and some patients may improve without surgery if injury is small, contamination is absent, and ventilation can be maintained with minimal pressure [23][24].

Enhancing Healthcare Team Outcomes

Tracheobronchial tear management exemplifies interprofessional care: outcomes depend on continuous communication and coordination among emergency clinicians, paramedics, anesthesiologists, pulmonologists, otorhinolaryngologists, cardiothoracic surgeons, intensivists, nurses, respiratory therapists, and rehabilitation specialists. Optimal prehospital trauma management improves survival by stabilizing oxygenation without exacerbating airway disruption. In the emergency department, clinician vigilance reduces missed diagnoses, directly impacting mortality. Timely involvement of airway specialists facilitates bronchoscopy-guided intubation and safe tube positioning, which reduces iatrogenic extension of the tear and mitigates false passage formation. Surgical specialists must coordinate not only airway repair but also interventions for associated vascular, esophageal, and thoracic injuries, often requiring staged procedures and careful sequencing. Intensive care teams ensure ventilatory strategies that avoid injurious pressures and maintain physiologic stability while monitoring for evolving pneumothorax, mediastinitis, and sepsis. Interventional pulmonology is essential for patients managed conservatively or with stents, providing surveillance bronchoscopy, secretion management, and device-related complication mitigation. Rehabilitation services—including physiotherapy and speech/swallow therapy—are frequently required after surgical or endoscopic treatment to restore function and prevent aspiration. Across all phases, structured handoffs, shared imaging review, and clear documentation of injury location, repair details, and airway tube positioning are practical teamwork behaviors that reduce complications and promote consistent, high-quality care for patients with tracheobronchial tears [24][25].

Conclusion:

Tracheobronchial tear represents a time-critical emergency where delayed recognition can lead to catastrophic outcomes. Despite its rarity, clinicians must maintain vigilance in trauma and critical care settings, particularly when faced with persistent pneumothorax, extensive subcutaneous emphysema, or unexplained pneumomediastinum. The cornerstone of successful management lies in controlled airway stabilization—preferably via fiberoptic bronchoscopy—to prevent exacerbation of the injury during intubation. Definitive surgical repair remains the standard for most traumatic cases, while conservative or endoscopic approaches may be appropriate for select iatrogenic injuries under strict criteria. Prognosis is strongly influenced by early diagnosis, prevention of mediastinal infection, and avoidance of high airway pressures during ventilation. Multidisciplinary coordination among emergency physicians, anesthesiologists, surgeons, and intensivists is essential to optimize outcomes. Preventive strategies, including improved trauma systems and procedural competency, can reduce incidence and complications. Ultimately, timely intervention transforms a potentially fatal injury into a survivable event, emphasizing the importance of structured protocols and continuous education in airway trauma management.

References:

1. Ecker RR, Libertini RV, Rea WJ, Sugg WL, Webb WR. Injuries of the trachea and bronchi. The Annals of thoracic surgery. 1971 Apr:11(4):289-98
2. Kummer C, Netto FS, Rizoli S, Yee D. A review of traumatic airway injuries: potential implications for airway assessment and management. Injury. 2007 Jan:38(1):27-33
3. Karmy-Jones R, Wood DE. Traumatic injury to the trachea and bronchus. Thoracic surgery clinics. 2007 Feb:17(1):35-46
4. Nelson LA. Airway trauma. International anesthesiology clinics. 2007 Summer:45(3):99-118
5. Kiser AC, O'Brien SM, Detterbeck FC. Blunt tracheobronchial injuries: treatment and outcomes. The Annals of thoracic surgery. 2001 Jun:71(6):2059-65
6. Welter S. Repair of tracheobronchial injuries. Thoracic surgery clinics. 2014 Feb:24(1):41-50. doi: 10.1016/j.thorsurg.2013.10.006.
7. Miñambres E, Burón J, Ballesteros MA, Llorca J, Muñoz P, González-Castro A. Tracheal rupture after endotracheal intubation: a literature systematic review. European journal of cardio-thoracic surgery : official journal of the European Association for Cardio-thoracic Surgery. 2009 Jun:35(6):1056-62. doi: 10.1016/j.ejcts.2009.01.053.
8. Johnson SB. Tracheobronchial injury. Seminars in thoracic and cardiovascular surgery. 2008 Spring:20(1):52-7. doi: 10.1053/j.semtcvs.2007.09.001.
9. Bertelsen S, Howitz P. Injuries of the trachea and bronchi. Thorax. 1972 Mar:27(2):188-94
10. Rossbach MM, Johnson SB, Gomez MA, Sako EY, Miller OL, Calhoon JH. Management of major tracheobronchial injuries: a 28-year experience. The Annals of thoracic surgery. 1998 Jan:65(1):182-6
11. Symbas PN, Justicz AG, Ricketts RR. Rupture of the airways from blunt trauma: treatment of complex injuries. The Annals of thoracic surgery. 1992 Jul:54(1):177-83
12. Schneider T, Volz K, Dienemann H, Hoffmann H. Incidence and treatment modalities of tracheobronchial injuries in Germany. Interactive cardiovascular and thoracic surgery. 2009 May:8(5):571-6. doi: 10.1510/icvts.2008.196790.
13. Massard G, Rougé C, Dabbagh A, Kessler R, Hentz JG, Roeslin N, Wihlm JM, Morand G. Tracheobronchial lacerations after intubation and tracheostomy. The Annals of thoracic surgery. 1996 May:61(5):1483-7
14. Borasio P, Ardissone F, Chiampo G. Post-intubation tracheal rupture. A report on ten cases. European journal of cardio-thoracic surgery : official journal of the European Association for Cardio-thoracic Surgery. 1997 Jul:12(1):98-100
15. Grewal HS, Dangayach NS, Ahmad U, Ghosh S, Gildea T, Mehta AC. Treatment of Tracheobronchial Injuries: A Contemporary Review. Chest. 2019 Mar:155(3):595-604. doi: 10.1016/j.chest.2018.07.018.
16. Parekh K, Iannettoni MD. Complications of esophageal resection and reconstruction. Seminars in thoracic and cardiovascular surgery. 2007 Spring:19(1):79-88
17. Juvekar NM, Deshpande SS, Nadkarni A, Kanitkar S. Perioperative management of tracheobronchial injury following blunt trauma. Annals of cardiac anaesthesia. 2013 Apr-Jun:16(2):140-3. doi: 10.4103/0971-9784.109772.
18. Herrmann D, Volmerig J, Al-Turki A, Braun M, Herrmann A, Ewig S, Hecker E. Does less surgical trauma result in better outcome in management of iatrogenic tracheobronchial laceration? Journal of thoracic disease. 2019 Nov:11(11):4772-4781. doi: 10.21037/jtd.2019.10.22.
19. de Lesquen H, Avaro JP, Gust L, Ford RM, Beranger F, Natale C, Bonnet PM, D'Journo XB. Surgical management for the first 48 h following blunt chest trauma: state of the art (excluding vascular injuries). Interactive cardiovascular and thoracic surgery. 2015 Mar:20(3):399-408. doi: 10.1093/icvts/ivu397.
20. Jougon J, Ballester M, Choukroun E, Dubrez J, Reboul G, Velly JF. Conservative treatment for postintubation tracheobronchial rupture. The Annals of thoracic surgery. 2000 Jan:69(1):216-20
21. Gómez-Caro A, Ausín P, Moradiellos FJ, Díaz-Hellín V, Larrú E, Pérez JA, de Nicolás JL. Role of conservative medical management of tracheobronchial injuries. The Journal of trauma. 2006 Dec:61(6):1426-34; discussion 1434-5
22. Cardillo G, Carbone L, Carleo F, Batzella S, Jacono RD, Lucantoni G, Galluccio G. Tracheal lacerations after endotracheal intubation: a proposed morphological classification to guide non-surgical treatment. European journal of cardio-thoracic surgery : official journal of the European Association for Cardio-thoracic Surgery. 2010 Mar:37(3):581-7. doi: 10.1016/j.ejcts.2009.07.034.
23. Lee SK, Kim DH, Lee SK, Kim YD, Cho JS, I H. Does Surgical Repair Still have a Role for Iatrogenic Tracheobronchial Rupture? Clinical Analysis of a Thoracic Surgeon's Opinion. Annals of thoracic and cardiovascular surgery : official journal of the Association of Thoracic and Cardiovascular Surgeons of Asia. 2016 Dec 20:22(6):348-353. doi: 10.5761/atcs.oa.16-00189.

24. Madden BP. Evolutional trends in the management of tracheal and bronchial injuries. Journal of thoracic disease. 2017 Jan:9(1):E67-E70. doi: 10.21037/jtd.2017.01.43.
25. Prokakis C, Koletsis EN, Dedeilias P, Fligou F, Filos K, Dougenis D. Airway trauma: a review on epidemiology, mechanisms of injury, diagnosis and treatment. Journal of cardiothoracic surgery. 2014 Jun 30:9():117. doi: 10.1186/1749-8090-9-117..
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