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# Rapid Identification and Emergency Management of Tension Pneumothorax: Evidence-Based Practice for Emergency, Nursing and Allied Health Professionals

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## Abstract

**Background:** Tension pneumothorax is a life-threatening condition characterized by progressive intrapleural pressure that compromises both respiratory and cardiovascular function. It is considered a "do-not-miss" diagnosis in emergency and critical care settings due to its rapid progression and reversibility with timely intervention.

**Aim:** To review evidence-based practices for rapid identification and emergency management of tension pneumothorax across prehospital, emergency, and critical care environments.

**Methods:** This narrative review synthesizes current literature on pathophysiology, clinical presentation, diagnostic strategies, and management protocols. Sources include trauma registries, critical care guidelines, and recent studies on procedural success rates and complications.

**Results:** Tension pneumothorax often arises from trauma, invasive procedures, or barotrauma during positive-pressure ventilation. Clinical recognition relies on signs of severe respiratory distress, hypotension, unilateral absent breath sounds, and mediastinal shift. Point-of-care ultrasound demonstrates high sensitivity (94%) and specificity (100%) for rapid diagnosis. Immediate needle decompression followed by chest tube thoracostomy remains the cornerstone of treatment, with success rates exceeding 90% when performed correctly. Delays in intervention significantly increase mortality, while early decompression markedly improves survival.

**Conclusion:** Rapid identification and immediate decompression are critical to preventing obstructive shock and cardiac arrest. Interprofessional coordination and adherence to evidence-based protocols enhance outcomes and reduce complications. Preventive strategies, including trauma safety, ventilator management, and patient education, further mitigate risk.

**Keywords:** Tension pneumothorax, emergency management, needle decompression, chest tube thoracostomy, critical care, trauma.

## Introduction

Pneumothorax refers to the presence of air within the pleural space, a pathologic state that disrupts the normal pressure relationship between the lung and the chest wall and results in partial or complete lung collapse. Under physiologic conditions, the pleural cavity functions as a sealed potential space, allowing the lungs to remain expanded through the maintenance of a slightly negative intrapleural pressure relative to atmospheric pressure. When air gains access to this space, the negative pressure gradient is lost and pleural pressure becomes abnormally positive, promoting elastic recoil of the lung and mechanical collapse. The clinical consequences of pneumothorax are therefore fundamentally mechanical: impaired lung expansion


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leads to reduced ventilation, compromised gas exchange, and increased breathing work. The severity of these effects varies by pneumothorax size, rate of accumulation, patient reserve, and the presence of underlying lung disease. Tension pneumothorax represents the most life-threatening manifestation of this process. It develops when progressive intrapleural pressure is not merely sufficient to collapse the lung, but is transmitted to the mediastinum, producing dynamic compression of central thoracic structures (see Image. Left-Sided Tension Pneumothorax Radiograph). Although tension pneumothorax is relatively uncommon compared with other causes of acute respiratory distress, it follows a malignant clinical course and can rapidly lead to death if not promptly treated.[1][2] This condition is most clinically relevant because it is both time-critical and, importantly, reversible when recognized early and treated with immediate decompression. As such, tension pneumothorax is considered a “do-not-miss” diagnosis in acute care settings. It may present across the continuum of care, including prehospital environments, emergency departments, operating rooms, and intensive care units, particularly in patients receiving positive-pressure ventilation or those exposed to thoracic trauma.[3][4][5][6]

A clear understanding of thoracic anatomy and pleural physiology helps explain why tension pneumothorax is uniquely dangerous. The thorax is commonly conceptualized as three compartments: two lateral pulmonary cavities—right and left—each containing a lung, and a centrally located mediastinum that houses the heart, great vessels, trachea, esophagus, and major neural structures. Each lung is covered by visceral pleura, while the inner surface of the chest wall and diaphragm is lined by parietal pleura. The space between these layers is the pleural cavity, which ordinarily contains only a thin film of serous fluid. This fluid lubricates pleural surfaces, allowing near-frictionless movement as the lungs expand and recoil during the respiratory cycle. The integrity of this pleural system is essential: it permits coupling between the chest wall and the lungs such that thoracic expansion translates into lung expansion. During inspiration, diaphragmatic contraction causes downward displacement of the diaphragm, and the external intercostal muscles elevate and expand the rib cage outward. These actions increase thoracic volume and decrease pleural pressure, generating a slightly more negative intrapleural pressure that promotes lung expansion and airflow into the alveoli. During expiration, the diaphragm relaxes and moves upward while the rib cage recoils inward, decreasing thoracic volume. Pleural pressure becomes less negative and may become transiently positive during forced expiration, facilitating airflow out of the lungs. Importantly, under normal circumstances these pressure changes remain within a physiologic range that supports ventilation without destabilizing thoracic structures. When the pleura is disrupted—whether by penetrating injury, blunt trauma, ruptured blebs, invasive procedures, or barotrauma from mechanical ventilation—air may enter the pleural cavity. As air accumulates, the pleural pressure rises, producing lung collapse and reducing effective ventilation of the affected side. This collapse decreases oxygenation and ventilation efficiency and can manifest clinically as dyspnea, tachypnea, pleuritic chest pain, and hypoxemia. In a simple pneumothorax, air entry may be limited or may equilibrate, allowing the patient to remain relatively stable while definitive management is arranged. In tension pneumothorax, however, a functional “one-way valve” mechanism typically develops, whereby air enters the pleural space during inspiration but cannot escape effectively during expiration. This leads to progressively rising intrapleural pressure with each breath, particularly in the presence of positive-pressure ventilation, which can drive air into the pleural space more forcefully [4][5][6].

As intrapleural pressure rises beyond a critical threshold, the pathophysiology extends beyond the collapsed lung. The increasing pressure compresses the mediastinum and displaces it toward the contralateral hemithorax, mechanically compressing the unaffected lung and further impairing ventilation. At the same time, compression of the great veins—particularly the superior and inferior vena cava—reduces venous return to the heart. Reduced preload leads to diminished cardiac output, hypotension, and ultimately obstructive shock. In severe cases, myocardial ischemia may develop as coronary perfusion becomes compromised. Tracheal deviation, classically described as a clinical sign, reflects the mechanical displacement of the mediastinum, though it may be absent early or difficult to detect in certain patients. The overall trajectory of tension pneumothorax is therefore a rapid convergence of respiratory failure and cardiovascular collapse, driven by mechanical forces rather than primary cardiac pathology. Because the condition can progress quickly and because imaging may be unavailable or too slow in unstable patients, early recognition and immediate management are essential to prevent mortality.[1][2] Clinicians must maintain a high index of suspicion in appropriate settings—particularly following trauma, in patients with sudden respiratory compromise on mechanical ventilation, or after procedures that may breach pleural integrity. Prompt decompression relieves intrapleural pressure, allows re-expansion of the lung, and reverses mediastinal compression, thereby restoring venous return and cardiac output. In practical terms, this means that proficiency in emergency thoracic decompression is a critical competency for healthcare professionals involved in

acute care.[7] For interprofessional teams, including nurses, paramedics, respiratory therapists, and physicians, effective outcomes depend on rapid assessment, immediate escalation, coordinated action, and continuous monitoring during and after decompression to ensure physiologic stabilization and prevent recurrence [7].

In summary, pneumothorax is defined by the presence of air in the pleural space, producing lung collapse through abnormal positive pleural pressure. Tension pneumothorax represents a severe, progressive variant in which rising intrapleural pressure compresses the mediastinum and central vascular structures, leading to obstructive shock and potentially death if untreated.[1][2] Its ability to present across prehospital, emergency, and critical care settings requires that clinicians recognize it quickly and intervene decisively.[3][4][5][6][7]

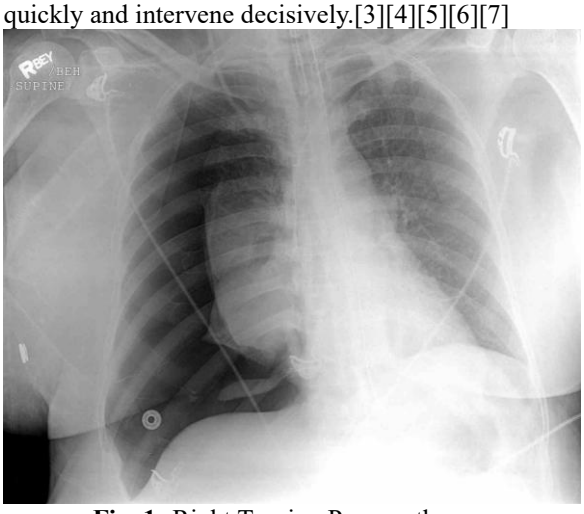

**Fig. 1:** Right Tension Pneumothorax.

**Etiology**

Pneumothorax is broadly categorized according to whether pleural air accumulation occurs as a consequence of trauma or in the absence of an identifiable traumatic event. This classification is clinically useful because it guides risk stratification, informs diagnostic reasoning, and supports targeted prevention strategies. Importantly, while tension pneumothorax is often associated with traumatic mechanisms, it is not limited to trauma alone. Any pathway that allows air to enter the pleural space and become trapped under pressure can progress to tension physiology. Therefore, recognition of both traumatic and atraumatic etiologies is essential for clinicians working across prehospital, emergency, operative, and critical care environments. Traumatic pneumothorax most commonly results from direct physical disruption of the pleura and lung parenchyma. In out-of-hospital settings, penetrating injuries such as stab wounds or gunshot wounds can introduce air through the chest wall and directly violate the pleural cavity. Blunt trauma is also a major contributor and may cause pneumothorax through multiple mechanisms, including alveolar rupture from sudden compressive forces, pleural tears, or lung laceration from fractured ribs. Rib fractures are particularly relevant because sharp bony fragments can puncture the visceral pleura, creating an internal air leak that may enlarge with each respiratory cycle.[8][9] Additionally, pulmonary decompression sickness—most often associated with diving-related barotrauma—can produce alveolar rupture and pleural air leakage when rapid pressure changes cause gas expansion within the lungs.[8][9] These prehospital etiologies are frequently complicated by hypoxemia, pain-related splinting, and concurrent hemorrhage or contusion, which can obscure early clinical signs and increase the likelihood of deterioration into a tension state if not recognized and treated promptly [8][9].

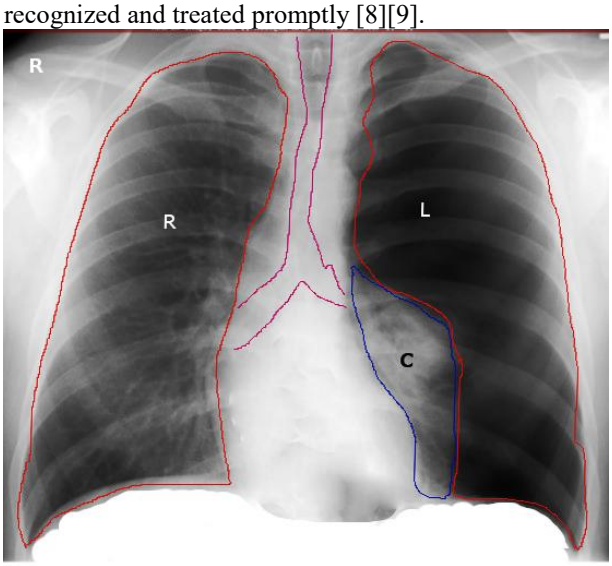


**Fig. 2**: Left Tension pneumothorax.

Within hospital settings, traumatic pneumothorax is often iatrogenic, arising as an unintended complication of diagnostic or therapeutic procedures that traverse or threaten pleural integrity. Central venous catheterization, particularly via the subclavian vein and internal jugular vein approaches, is a well-recognized cause. The anatomic proximity of these vessels to the lung apices increases the risk of pleural puncture, especially in emergent insertions, in patients with challenging anatomy, or when procedural landmarks are distorted. Lung biopsy procedures may also precipitate pneumothorax because tissue sampling can create a parenchymal defect that allows air to escape into the pleural space. Similarly, barotrauma from positive pressure ventilation is a clinically important mechanism, particularly in patients with stiff, noncompliant lungs or obstructive airway disease. Elevated airway pressures and overdistension can rupture alveoli, allowing air to dissect along tissue planes and enter the pleural cavity, with the potential to progress rapidly into tension physiology if a one-way valve mechanism develops. Other iatrogenic causes include percutaneous tracheostomy, in which airway and mediastinal tissue manipulation may create pathways for pleural air entry, and thoracentesis, where

inadvertent pleural injury can occur despite being performed specifically to remove pleural fluid. Pacemaker insertion is another recognized risk, as venous access and lead placement may involve the subclavian region near the pleural apex. Bronchoscopy, while typically low risk for pneumothorax in routine cases, can contribute particularly when combined with transbronchial biopsies or when airway pressures are elevated. Cardiopulmonary resuscitation can cause pneumothorax through rib fractures or barotrauma during aggressive ventilation, and intercostal nerve blocks may create pleural puncture if needle placement extends beyond the intended plane.[10] Collectively, these hospital-associated etiologies highlight that pneumothorax is not limited to trauma patients; it can arise as an adverse procedural outcome in diverse clinical populations. Atraumatic pneumothorax is typically classified as either primary or secondary. Primary spontaneous pneumothorax occurs without an obvious precipitating cause and often reflects rupture of small subpleural blebs, frequently in patients without known lung disease. Secondary spontaneous pneumothorax arises in the context of underlying pulmonary pathology, where structural abnormalities or chronic inflammation increase the likelihood of air leak into the pleural space. Although the clinical presentation and risk of recurrence may differ between primary and secondary forms, both can deteriorate into tension pneumothorax under the right conditions, especially when positive pressure ventilation is applied or when the pleural defect acts as a one-way valve. In summary, pneumothorax etiologies span a continuum from traumatic mechanisms—penetrating and blunt trauma, rib fractures, and decompression-related barotrauma—to iatrogenic causes within healthcare settings, including central venous catheterization, lung biopsy, positive pressure ventilation, tracheostomy, thoracentesis, pacemaker insertion, bronchoscopy, cardiopulmonary resuscitation, and intercostal nerve blocks.[8][9][10] Atraumatic pneumothorax may be primary or secondary to underlying lung disease. Across all categories, the central safety implication is that tension pneumothorax can arise from any of these pathways, requiring vigilance, early recognition, and rapid escalation to definitive management when clinical deterioration suggests evolving tension physiology.

**Epidemiology**

Estimating the true epidemiology of tension pneumothorax is inherently challenging because the condition is frequently treated before definitive diagnostic confirmation, particularly in prehospital and austere environments. Many patients suspected of having tension physiology receive immediate decompressive interventions—most commonly needle thoracostomy—before arrival at trauma centers, which can both save lives and obscure the ability to capture confirmed cases in hospital-based registries. As a result, population-level incidence and prevalence data are often underestimates, and epidemiologic figures must be interpreted with awareness of treatment-related “ascertainment bias.” Despite these limitations, available evidence consistently reinforces that tension pneumothorax, while less common than simple pneumothorax, is a clinically important contributor to preventable mortality in trauma and critical illness due to its rapid progression and reversibility when promptly managed. Trauma remains one of the most significant contexts in which tension pneumothorax occurs. Approximately 20% of patients with traumatic injury present with an associated pneumothorax or tension pneumothorax, and this proportion can rise to nearly 50% in the setting of severe chest trauma.[11][12] These figures highlight that pleural air complications are common in serious trauma and should remain high on the differential diagnosis when patients exhibit acute respiratory compromise, hemodynamic instability, or sudden deterioration after injury. The likelihood of traumatic pneumothorax is influenced by both the mechanism and magnitude of injury. Penetrating injuries such as gunshot wounds and stab wounds can directly disrupt pleural integrity and create ongoing air leaks, while blunt mechanisms can cause pneumothorax through rib fractures, lung lacerations, or alveolar rupture from rapid compressive forces. In severe trauma, concurrent hemorrhage, pulmonary contusion, or airway obstruction can coexist and complicate clinical recognition, increasing the importance of structured trauma assessment and rapid escalation when tension pneumothorax is suspected [11][12].

Military and combat-related data further underscore the lethality of tension pneumothorax when treatment is delayed or inaccessible. A review of military deaths associated with thoracic trauma indicates that up to 5% of combat casualties have tension pneumothorax at the time of death.[11][12] This statistic is particularly significant because it suggests that tension pneumothorax contributes to potentially preventable deaths in high-risk operational settings. In environments where evacuation is delayed, resources are limited, or injuries are complex, the time window for intervention may be short. Consequently, combat trauma systems have emphasized early recognition and rapid decompression capability, and these lessons have informed civilian trauma protocols, especially in prehospital emergency medical services. From a broader perspective, traumatic and tension pneumothorax are generally more common than spontaneous pneumothorax, largely because trauma and invasive interventions create direct pathways for pleural air entry and because mechanically ventilated patients are at higher risk for rapid progression to tension physiology. Nevertheless, spontaneous

pneumothorax remains clinically relevant, and tension physiology can develop even in initially stable presentations. Tension pneumothorax has been reported to develop in approximately 1% to 2% of cases that initially present as idiopathic spontaneous pneumothorax, emphasizing that "spontaneous" does not equate to "benign." Even when a patient initially appears stable, worsening pleural air accumulation or the application of positive pressure ventilation can precipitate rapid decompensation, particularly in patients with limited cardiopulmonary reserve. A major contemporary epidemiologic trend is the increasing burden of iatrogenic pneumothorax in hospital settings. This increase is closely linked to greater reliance on positive pressure ventilation (PPV) and central venous catheterization (CVC), both of which are common in modern critical care. PPV increases alveolar pressure and can contribute to barotrauma, especially when lung compliance is poor or airway pressures are elevated. Similarly, CVC placement—particularly via the internal jugular or subclavian veins—carries risk because of anatomic proximity to pleural structures and the potential for needle or guidewire misdirection. Multiple factors correlate with increased pneumothorax risk during venous access, including failed initial cannulation attempts, subclavian approaches, and the presence of PPV, which can increase lung expansion into the procedural field and reduce margin for error.[13] These risk factors are clinically important because many are modifiable through technique, supervision, and ultrasound guidance [13].

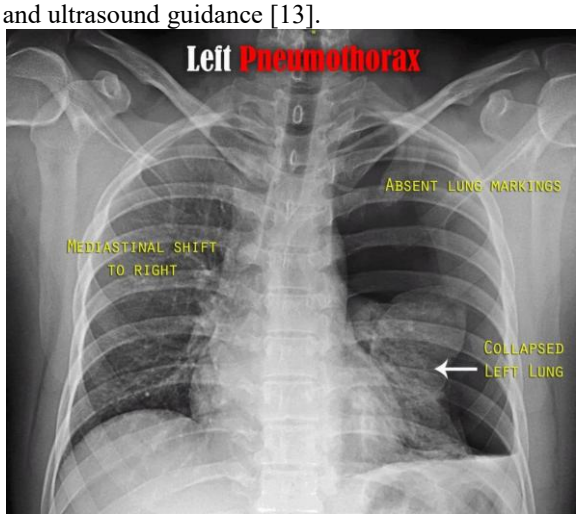


**Fig. 3:** Left sided tension pneumothorax.

The risk of CVC-related pneumothorax is reported to rise when catheters are placed in the internal jugular or subclavian veins, and published incidence estimates vary widely—from approximately 1% to 13%—with some circumstances reporting rates as high as 30%.[14] Such variability reflects differences in operator experience, patient anatomy, urgency of placement, use of ultrasound, and the specific definition of pneumothorax used in studies. Importantly, ultrasound guidance has been shown to reduce pneumothorax risk, supporting its incorporation as a standard safety practice when feasible.[14] Although iatrogenic pneumothorax typically produces significant morbidity—such as longer hospital stays, increased oxygen requirements, and potential need for chest tube placement—it rarely results in death when recognized and treated promptly. However, because critically ill patients often have minimal physiologic reserve, even "rarely fatal" complications can carry substantial clinical consequences and contribute to downstream deterioration. Hospital admission–based estimates further contextualize the burden. The incidence of iatrogenic pneumothorax has been reported at approximately 5 to 7 per 10,000 hospital admissions, indicating that it remains relatively uncommon at the population level but important due to its preventability and impact. In one recent study, 95% of pneumothorax episodes were iatrogenic, illustrating how modern hospital pneumothorax patterns can differ from community-based spontaneous presentations. Barotrauma related to mechanical ventilation accounted for 69.6% of cases in this study, and notably, 41.1% of these episodes progressed to tension pneumothorax, underscoring how quickly ventilator-associated air leaks can evolve into life-threatening physiology. Central venous catheter insertion was responsible for 13.2% of cases.[15] These findings highlight that, within acute care hospitals, the epidemiology of pneumothorax is increasingly driven by invasive supportive therapies rather than by spontaneous etiologies alone. In summary, the epidemiology of tension pneumothorax is difficult to measure precisely because many cases are treated before arrival to definitive care, but available evidence indicates it remains a critical threat across trauma and critical care.[11][12] Pneumothorax or tension pneumothorax occurs in roughly 20% of trauma presentations and up to 50% of severe chest trauma, and tension pneumothorax may contribute to as many as 5% of combat deaths from thoracic trauma.[11][12] While tension physiology develops in a minority of spontaneous pneumothorax cases, iatrogenic pneumothorax is increasingly prominent due to PPV and CVC use, with risk elevated by failed cannulation, subclavian approaches, and mechanical ventilation.[13] CVC-related pneumothorax incidence ranges broadly and can be reduced with ultrasound guidance.[14] Contemporary hospital data suggest most pneumothoraces may be iatrogenic, with ventilator barotrauma a leading cause and a substantial proportion progressing to tension pneumothorax.[15]

**Pathophysiology**

Tension pneumothorax represents a progressive, life-threatening disruption of pleural mechanics in which accumulating intrapleural air converts a primarily respiratory problem into combined respiratory and circulatory failure. Under normal physiologic conditions, the pleural cavity

functions as a sealed potential space in which intrapleural pressure remains negative relative to both alveolar and atmospheric pressure. This negative pressure is crucial because it couples the lungs to the chest wall. Although the lung tissue has an inherent tendency to recoil inward due to elastic forces, it remains expanded because the thoracic cage tends to recoil outward and because the negative pleural pressure gradient maintains apposition between visceral and parietal pleura. The result is stable lung inflation at functional residual capacity and efficient ventilation during the respiratory cycle. A pneumothorax begins when this sealed system is breached and a pathologic communication forms between the pleural cavity and either the lung parenchyma or the external environment. Once air enters the pleural space, the negative pressure gradient is lost, and intrapleural pressure becomes less negative and may become positive. As pleural air accumulates, pleural pressure rises further, mechanically compressing the affected lung. This compression decreases alveolar ventilation by reducing lung expansion, which leads to ventilation–perfusion mismatch, impaired oxygenation, and reduced carbon dioxide clearance. Clinically, these physiologic changes manifest as dyspnea, tachypnea, hypoxemia, and increased work of breathing. The degree of impairment depends on the size and rate of pneumothorax development, the patient's baseline lung function, and the presence of concurrent injuries or disease. Tension pneumothorax develops when the pleural air accumulation is not self-limited and instead becomes progressively pressurized. This typically occurs through a functional one-way valve mechanism: air is drawn or forced into the pleural cavity during inspiration but cannot escape effectively during expiration. The process may be accelerated by positive-pressure ventilation, which increases intrathoracic and alveolar pressures and can drive air into the pleural space more forcefully. As pressure rises beyond the capacity of the hemithorax to accommodate it, the pathophysiology extends beyond the ipsilateral lung. Intrapleural pressure becomes high enough to compress the mediastinum and displace it toward the contralateral side (see Image. Left Tension Pneumothorax Radiograph). This mediastinal shift has two major consequences: progressive compromise of ventilation by compressing the contralateral lung, and progressive compromise of circulation by compressing mediastinal vascular structures.[16]

The hemodynamic collapse associated with tension pneumothorax is primarily driven by impaired venous return. Compression of the great veins—particularly the superior vena cava—limits blood flow back to the right atrium, reducing preload.[17] With diminished preload, right ventricular filling falls, leading to reduced stroke volume and a decline in cardiac output. Because systemic perfusion depends on cardiac output, hypotension and signs of obstructive shock may develop rapidly. In addition, elevated intrathoracic pressure can increase pulmonary vascular resistance by compressing pulmonary vasculature, further straining right ventricular function and worsening cardiopulmonary coupling. This combination—reduced venous return and increased afterload for the right heart—creates a physiologic spiral in which both oxygen delivery and circulatory stability deteriorate concurrently. Tracheal deviation is often described as a consequence of mediastinal displacement, reflecting the mechanical movement of central thoracic structures away from the side of rising pressure.[17] While it may be a helpful clinical clue in some cases, it is not uniformly present, especially early in the process or in patients with short necks, obesity, cervical collars, or limited examination conditions. Nevertheless, the underlying concept is consistent: as the pressurized pleural space expands, it pushes mediastinal contents across the midline, distorting normal airway and vascular anatomy and worsening both ventilation and perfusion. As cardiac output declines, tissue oxygen delivery decreases, compounding the hypoxemia that is already present due to lung collapse and ventilation–perfusion mismatch. The resulting global hypoperfusion promotes anaerobic metabolism and lactic acidosis. Simultaneously, impaired ventilation can contribute to hypercapnia, which further worsens acidosis. Acidemia has deleterious physiologic consequences: it reduces myocardial contractility, predisposes to arrhythmias, and impairs responsiveness to endogenous catecholamines. If the pressure is not relieved, circulatory collapse may progress rapidly to pulseless electrical activity and cardiac arrest.[18][19] This trajectory explains why tension pneumothorax is treated as an immediate, clinical diagnosis rather than one that should be delayed for confirmatory imaging in unstable patients. In summary, tension pneumothorax begins with a breach in pleural integrity that allows air to enter the pleural cavity, raising pleural pressure and collapsing the lung, thereby impairing ventilation and oxygenation.[16] When pleural pressure continues to rise through a one-way valve mechanism, mediastinal compression and contralateral shift occur, compromising both respiratory mechanics and cardiovascular function. Compression of the superior vena cava reduces venous return and cardiac output, while increased pulmonary vascular resistance and hypoxemia further destabilize cardiopulmonary physiology.[17] Without urgent decompression, the resulting shock leads to acidosis and can culminate in cardiac arrest.[18][19]

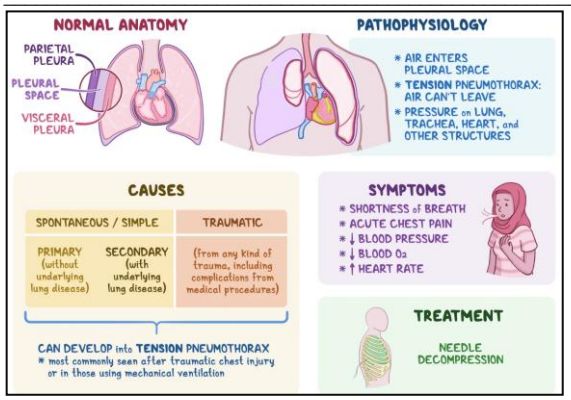


**Fig. 4:** Tension Pneumothorax.

**History and Physical**

Tension pneumothorax is a true medical emergency in which delays in recognition and decompression can rapidly lead to irreversible hypoxemia, obstructive shock, and cardiac arrest. For this reason, the history and physical examination are not performed as a comprehensive diagnostic exercise but as a rapid, focused assessment aimed at identifying a time-critical, reversible cause of decompensation. In unstable patients, tension pneumothorax is treated as a clinical diagnosis rather than a radiographic one, and emergent intervention is prioritized over confirmatory imaging. The bedside evaluation therefore emphasizes recognition of a characteristic pattern: acute respiratory compromise coupled with hemodynamic instability, often in a patient with a plausible precipitating mechanism such as trauma, positive pressure ventilation (PPV), or recent invasive thoracic or vascular procedures. On presentation, patients commonly exhibit severe respiratory distress, manifested by marked dyspnea, tachypnea, accessory muscle use, and visible chest retractions. Hypoxemia may be apparent clinically through cyanosis, agitation, or altered mental status, particularly when oxygenation is profoundly impaired. As mediastinal compression worsens venous return, signs of shock emerge, including hypotension, cool clammy skin, delayed capillary refill, and decreasing level of consciousness. The cardiovascular response may include tachycardia initially, which can progress to bradycardia in pre-arrest states. In the classic description, the affected hemithorax becomes enlarged and hyperresonant due to trapped intrapleural air, and breath sounds are diminished or absent on auscultation over the involved side. These findings reflect both lung collapse and the insulating effect of pleural air, which reduces effective sound transmission. However, clinicians must remember that in noisy environments or in patients with bilateral lung disease, auscultation may be less reliable; therefore, interpretation should be integrated with the overall clinical picture. Tracheal deviation and mediastinal shift toward the contralateral side are frequently described as hallmark signs, reflecting the mechanical displacement caused by rising intrathoracic pressure. When present, tracheal deviation is highly concerning because it suggests advanced tension physiology, yet it may be absent early in the course or difficult to appreciate in patients with short necks, obesity, cervical immobilization, or limited access to the neck. As such, the absence of visible tracheal deviation should not delay decompression if other findings support tension pneumothorax. Jugular venous distension (JVD) can occur due to impaired venous return; however, this sign may be blunted in hypovolemic trauma patients or may not be visible in patients with poor venous compliance, making it a supportive rather than definitive finding [20].

History-taking, when feasible, should rapidly identify antecedent events that elevate pretest probability. Recent blunt or penetrating trauma to the chest, rib fractures, or blast-related injuries are common triggers in the prehospital and emergency department context. In hospitalized patients, PPV is a major risk factor because barotrauma can cause alveolar rupture and pleural air leakage, with tension physiology developing quickly as ventilator pressures continue to drive air into the pleural space. Central venous catheterization (CVC), especially via subclavian or internal jugular approaches, is another important antecedent because accidental pleural puncture may occur and may deteriorate rapidly in ventilated patients. A focused history should also consider underlying pulmonary conditions that predispose to air leaks or rapid deterioration, including severe asthma, pneumonia, chronic obstructive pulmonary disease, and other conditions associated with hyperinflation or fragile lung architecture. Symptomatically, patients frequently report sharp pleuritic chest pain, often radiating to the ipsilateral back or shoulder, along with abrupt dyspnea. In some cases—particularly in ventilated or sedated patients—subjective symptoms cannot be reported, and clinicians must rely on physiologic changes such as sudden desaturation, increasing airway pressures, or unexplained hypotension. Additional early physical findings can further support the diagnosis. The affected hemithorax may demonstrate reduced tactile fremitus because pleural air dampens transmission of vibratory sound through the chest wall. Percussion may reveal hyperresonance, consistent with air in the pleural space. Subcutaneous emphysema—palpable crepitus in the soft tissues of the chest wall or neck—may be present and suggests air dissection into subcutaneous planes, often accompanying traumatic or barotrauma-related pneumothoraces. While helpful, subcutaneous emphysema is not required for diagnosis and may be absent. Importantly, the clinical trajectory is often one of rapid deterioration: ventilation failure and obstructive shock can evolve over minutes, particularly in patients receiving PPV. Because tension pneumothorax is a clinical diagnosis, the definitive “confirmation” in unstable patients is often therapeutic. The release of air after inserting a large-

bore needle into the traditionally described location—second intercostal space at the midclavicular line—supports the diagnosis by demonstrating pressurized pleural air escape. Nevertheless, clinical practice recognizes that emergent decompression should be guided by institutional protocols and operator training, and the key concept remains unchanged: decompression should not be delayed when the diagnosis is strongly suspected, because waiting for imaging can be fatal.[20] In the most extreme presentation, an unconscious patient without respirations or a palpable pulse must receive immediate resuscitation and stabilization measures regardless of the presumed cause, while reversible etiologies such as tension pneumothorax are addressed rapidly within standard resuscitation algorithms.[20]

**Evaluation**

Evaluation of suspected tension pneumothorax must be organized around a single overriding principle: the patient's physiologic stability determines how much diagnostic confirmation is appropriate before intervention. Because tension pneumothorax can progress rapidly to obstructive shock and cardiac arrest, diagnostic processes must never delay life-saving decompression in unstable patients. Accordingly, assessment begins with immediate appraisal of airway, breathing, and circulation, with stabilization proceeding in parallel with targeted evaluation. When the diagnosis is uncertain, clinicians must decide whether to pursue bedside confirmation tools—most notably point-of-care ultrasound—or to proceed directly to treatment based on clinical probability and the consequences of delay. In practical terms, the diagnostic threshold for intervention is lower in hemodynamically unstable patients, whereas hemodynamically stable patients can generally undergo imaging to confirm the diagnosis and evaluate alternative causes of symptoms. In an unstable patient, bedside ultrasound has become a central tool for rapid confirmation when available and when an appropriately trained operator is present. Ultrasound is particularly valuable because it is portable, can be performed within seconds at the bedside, and does not require patient transport. In the context of tension physiology, this speed is clinically meaningful because transporting an unstable patient to radiography or computed tomography may worsen hemodynamics or delay decompression. When performed by a skilled operator, ultrasound has demonstrated high diagnostic performance, with reported sensitivity of 94% and specificity of 100% for pneumothorax detection.[21][22][23][24][25] These metrics support point-of-care ultrasound as an effective front-line diagnostic modality, especially in critical care and emergency medicine environments where rapid decisions are required.

Key ultrasound findings reflect the loss of normal pleural apposition and movement. The absence of lung sliding is a principal sign: under normal circumstances, the visceral and parietal pleura move relative to one another with respiration, producing a visible shimmering motion on ultrasound. When pleural air separates these layers, this sliding motion is no longer seen. While absence of lung sliding raises suspicion for pneumothorax, it should be interpreted in clinical context because other conditions—such as apnea, mainstem bronchus intubation, pleurodesis, or severe lung fibrosis—can also reduce sliding. Therefore, the lung point is particularly useful because it represents the boundary between aerated lung that remains apposed to the chest wall and the region where pleural air has separated pleural layers. Identification of a lung point is considered highly specific for pneumothorax and strengthens diagnostic confidence.[21][22][23][24][25] In patients who are profoundly unstable, ultrasound can be integrated into resuscitation workflows, allowing clinicians to confirm or refute pneumothorax while simultaneously managing oxygenation, circulation, and potential reversible causes of shock. Despite the strengths of ultrasound, clinical judgment remains essential. If diagnostic doubt persists after ultrasound examination—whether due to suboptimal windows, operator uncertainty, or conflicting findings—needle decompression should still be considered when tension pneumothorax remains plausible and the patient is deteriorating. This reflects the risk–benefit balance: the consequences of missing tension pneumothorax in an unstable patient are often fatal, whereas prompt decompression can be life-saving. Consequently, in unstable cases, ultrasound supports decision-making but does not replace the need for immediate therapeutic action when the clinical picture indicates tension physiology [21][22][23][24][25].

For hemodynamically stable patients, radiographic evaluation is recommended because imaging can confirm pneumothorax, characterize its size, assess mediastinal shift, and identify associated findings such as subcutaneous emphysema or alternative diagnoses (see Image. Right Tension Pneumothorax Radiograph). Initial imaging typically involves a chest x-ray, which remains the standard first-line radiographic test in many settings.[26][27] Classic radiographic signs of pneumothorax include visualization of a thin visceral pleural line with absence of lung markings beyond that edge, reflecting air in the pleural space rather than aerated lung tissue. Larger pneumothoraces may show partial or complete collapse of the ipsilateral lung. In tension pneumothorax, additional radiographic features indicate pressure effect, including mediastinal shift away from the affected side, tracheal deviation toward the contralateral side, and flattening or

depression of the ipsilateral hemidiaphragm due to increased intrathoracic pressure. Subcutaneous emphysema may also be visible, presenting as streaks of lucency within the soft tissues of the chest wall or neck. These findings collectively reinforce the diagnosis and help clinicians differentiate a simple pneumothorax from one producing tension physiology. Computed tomography of the chest may be employed when chest x-ray findings are equivocal or when detailed characterization is required, such as in complex trauma or when concurrent pulmonary pathology obscures plain radiography interpretation. CT is regarded as the most definitive imaging modality for pneumothorax because it can detect small volumes of pleural air and define anatomy with high resolution. However, routine CT use in suspected tension pneumothorax is not recommended because it requires patient transport, takes longer to obtain, and may delay urgent intervention. In suspected tension pneumothorax, CT is therefore best reserved for stable patients in whom diagnosis remains uncertain after initial evaluation or when additional injuries or complications must be assessed. In summary, evaluation of suspected tension pneumothorax is guided by stability and urgency. In unstable patients, concurrent resuscitation and focused bedside assessment are prioritized, and point-of-care ultrasound can rapidly support diagnosis with high reported sensitivity and specificity when performed by skilled operators, using findings such as absent lung sliding and the lung point.[21][22][23][24][25] If uncertainty persists and tension pneumothorax remains plausible, decompression should not be delayed. In stable patients, chest x-ray provides confirmatory evidence and identifies classic radiographic signs of pneumothorax and tension physiology.[26][27] CT may clarify uncertain cases but should not be used routinely when tension pneumothorax is suspected due to the imperative for timely intervention.

**Treatment / Management**

Tension pneumothorax is a time-critical emergency that can arise in prehospital environments, emergency departments, operating rooms, and intensive care units, and management must be adapted to the clinical context and the patient's hemodynamic status. The defining priority is immediate stabilization, because tension physiology can progress rapidly to obstructive shock and cardiac arrest. Accordingly, initial management begins with a structured assessment of airway, breathing, and circulation in all patients, particularly those with suspected chest trauma. This approach ensures that reversible threats to life are identified promptly while resuscitative measures are initiated in parallel. In trauma-related presentations, clinicians must also address open chest injuries. Penetrating chest wounds should be sealed promptly with an airtight occlusive dressing, commonly supported by sterile plastic sheeting, to limit further air entry through the chest wall and reduce the risk of worsening pneumothorax or development of a sucking chest wound physiology. Oxygen therapy is a core supportive intervention and should be instituted early. Administration of 100% supplemental oxygen reduces pneumothorax size by lowering the alveolar partial pressure of nitrogen. By reducing nitrogen concentration in alveoli, a diffusion gradient is created that accelerates nitrogen reabsorption from the pleural space into the bloodstream, thereby promoting resolution of trapped pleural air. Without oxygen supplementation, only a small proportion of trapped pleural air—approximately 1.25%—is absorbed over 24 hours, underscoring why high-concentration oxygen can meaningfully enhance the physiologic rate of resolution when definitive interventions are being prepared.[28] While oxygen alone is not an adequate treatment for tension pneumothorax, it is a critical bridge therapy that improves oxygenation and supports pleural air resorption in stable patients and after decompression.

Positive-pressure ventilation (PPV) requires particular caution in suspected tension pneumothorax. PPV can exacerbate tension physiology by increasing alveolar and intrathoracic pressures, forcing additional air through pleural defects and accelerating pleural air accumulation. Therefore, PPV should be avoided initially when tension pneumothorax is suspected, especially before pleural decompression. When respiratory failure mandates ventilatory support, clinicians should prioritize rapid decompression first. PPV can be instituted more safely after the pleural space has been decompressed and a chest tube is placed, because continuous drainage prevents reaccumulation and reduces the risk of recurrent tension physiology.[28][29] The cornerstone of immediate treatment in an unstable patient is emergent pleural decompression. If the patient is hemodynamically unstable and clinical suspicion for tension pneumothorax is high, needle decompression must be performed without delay. In this context, the diagnosis is treated clinically: waiting for imaging can be fatal. Traditional needle placement is described at the second anterior intercostal space along the midclavicular line, with insertion performed above the rib to reduce the risk of neurovascular bundle injury, typically using an angiocatheter.[30] However, clinical practice has increasingly recognized that success rates vary depending on chest wall thickness and anatomic considerations. When time permits and trained personnel are available, insertion at the fifth intercostal space along the anterior axillary line is often preferred because it tends to offer higher success rates and fewer complications, particularly in patients with increased chest wall thickness.[30] Because inadequate catheter length is a common cause of failure, long angiocatheters—often greater than 8 cm—are recommended to ensure pleural

penetration and effective decompression across diverse body habitus.[30]

Needle decompression functions as an emergency temporizing measure. By releasing pressurized pleural air, it reduces intrapleural pressure, allows partial reexpansion of the collapsed lung, and relieves mediastinal compression, thereby improving venous return and cardiac output. Nevertheless, needle decompression is not definitive management because the catheter can kink, become dislodged, or fail to provide ongoing drainage if the air leak persists. Additionally, clinicians should recognize that rapid reexpansion of a collapsed lung may increase the risk of reexpansion pulmonary edema, a complication that can worsen oxygenation and require intensified respiratory support.[30] For this reason, stabilization after decompression must include continuous monitoring of respiratory status, oxygen saturation, hemodynamics, and evolving ventilatory requirements. Following emergent decompression, definitive management typically involves chest tube thoracostomy (CTT), which provides continuous evacuation of pleural air and prevents recurrence of tension physiology. In stable patients, chest radiography is generally obtained to confirm pneumothorax and guide treatment, and chest x-ray is also used after tube placement to confirm position and assess lung reexpansion.[30] Serial chest x-rays assist in tracking resolution and ensuring that the lung remains expanded over time. Removal of the chest tube becomes appropriate once the lung has fully reexpanded, no ongoing air leak is detected, and the patient shows sustained clinical improvement, including stable respiratory mechanics and oxygenation. Chest tube management is inherently interprofessional. Experienced nurses, respiratory therapists, surgeons, and intensive care clinicians collaborate to ensure safe drainage system function, appropriate suction settings when indicated, secure tube fixation, dressing integrity, pain control, and continuous monitoring for complications such as tube occlusion, dislodgement, infection, or subcutaneous emphysema. When performed appropriately, CTT resolves approximately 90% of pneumothorax cases, making it the mainstay of definitive management.[31][32][33] Escalation to operative approaches—such as video-assisted thoracoscopic surgery (VATS) or thoracotomy—is generally reserved for cases that fail to resolve with CTT, recurrent events, or complex situations involving ongoing air leaks or structural lung pathology.[31][32][33][34]

Surgical intervention is often indicated in specific high-risk scenarios, including bilateral pneumothoraces, recurrent ipsilateral pneumothoraces, pulmonary decompression sickness, or persistent air leaks lasting more than seven days. During VATS, pleurodesis is commonly performed to reduce recurrence risk by eliminating the pleural space through adhesion formation between pleural layers. Mechanical pleurodesis may involve abrasion using scratchpads or dry gauze or may include parietal pleura stripping. Chemical pleurodesis provides an alternative for patients who cannot tolerate mechanical techniques and may use agents such as talc, minocycline, doxycycline, or tetracycline. Autologous blood patch pleurodesis has also demonstrated efficacy in selected cases, particularly when persistent air leak is present and other methods are not feasible. In certain recurrent or unresolved pneumothoraces, endobronchial valve placement may be considered to reduce airflow to the leaking segment and facilitate healing. Contemporary evidence indicates that pleurodesis significantly reduces the risk of recurrence, with mechanical pleurodesis reported to lower recurrence risk to less than 5%.[35][36] In summary, management of tension pneumothorax requires immediate, physiologically driven decision-making: stabilize airway, breathing, and circulation; seal open chest wounds; administer high-concentration oxygen to improve oxygenation and accelerate pleural air resorption; avoid PPV until decompression is achieved; and perform immediate needle decompression when instability and clinical suspicion are high.[28][29][30]. Definitive control is achieved with chest tube thoracostomy, supported by serial imaging and coordinated interprofessional care, with operative interventions reserved for refractory, recurrent, or complicated cases and with pleurodesis strategies deployed to reduce recurrence and improve long-term outcomes.[31][32][33][34][35][36].

**Differential Diagnosis**

Tension pneumothorax is a time-sensitive diagnosis because delayed recognition can lead rapidly to obstructive shock and cardiac arrest. Clinically, it often presents with abrupt respiratory compromise and hemodynamic instability, features that overlap with several other high-acuity cardiopulmonary and traumatic conditions. For this reason, clinicians must maintain a broad differential diagnosis when evaluating patients with acute dyspnea, chest pain, hypoxemia, tachycardia, or hypotension, particularly in emergency, prehospital, and critical care settings. At the same time, when tension pneumothorax is strongly suspected, definitive decompression should not be delayed for extensive diagnostic testing, as this disorder is both rapidly fatal and rapidly reversible with appropriate intervention. The differential diagnosis is therefore most useful when the presentation is atypical, when the patient is hemodynamically stable enough to permit further evaluation, or when competing explanations for shock and respiratory distress remain plausible. Pulmonary embolism is a major consideration because it can cause sudden dyspnea, pleuritic chest pain, tachycardia, hypoxemia, and hypotension in massive cases. Like tension

pneumothorax, massive pulmonary embolism can lead to obstructive physiology, but the mechanisms differ: embolic occlusion increases pulmonary vascular resistance and impairs right ventricular output rather than compressing mediastinal structures. Bedside clues that may favor pulmonary embolism include risk factors for venous thromboembolism, syncope, and signs of right heart strain, while lung auscultation is often relatively preserved. In contrast, tension pneumothorax is more likely to produce unilateral absent breath sounds and unilateral hyperresonance with thoracic asymmetry. Acute coronary syndrome and myocardial infarction are also common diagnostic considerations, as they may present with chest pain, diaphoresis, dyspnea, and hemodynamic instability. However, these conditions primarily reflect myocardial ischemia and pump failure rather than mechanical impairment of ventilation. Respiratory distress in myocardial infarction is often secondary to pulmonary edema, which typically produces bilateral crackles rather than unilateral absence of breath sounds. Similarly, acute pericarditis may produce pleuritic chest pain and dyspnea, but it does not usually cause unilateral thoracic findings. When hypotension is prominent in a pericardial process, clinicians should also consider cardiac tamponade, which can mimic obstructive shock; however, tamponade is more likely to present with muffled heart sounds, jugular venous distension, and relatively clear lungs on auscultation [36].

Acute aortic dissection is another critical differential diagnosis, especially in patients with severe, sudden chest or back pain and signs of shock. Dissection can produce ischemia, neurologic symptoms, or pulse deficits, depending on the vessels involved. While dyspnea may occur, it is not typically associated with unilateral absent breath sounds or a hyperexpanded hemithorax. If pleural effusion or hemothorax develops secondary to dissection, auscultatory findings may change, but the pattern differs from tension pneumothorax because fluid tends to produce dullness rather than hyperresonance. Pneumonia can present with dyspnea, hypoxemia, fever, and chest pain, and severe cases may lead to sepsis and hypotension. However, pneumonia generally evolves over hours to days rather than minutes and is characterized by focal crackles, bronchial breath sounds, or decreased breath sounds with consolidation—findings that do not typically include an enlarged hyperresonant hemithorax or contralateral tracheal deviation. Moreover, infectious symptoms and systemic inflammatory signs often provide additional context. In trauma settings, rib fracture and diaphragmatic injuries must be considered because they can cause chest pain, splinting, respiratory distress, and abnormal breath sounds. Rib fractures can be associated with pneumothorax or hemothorax, and diaphragmatic rupture may cause respiratory compromise through herniation of abdominal contents into the thorax. These entities may coexist with pneumothorax rather than simply mimic it, which is why a trauma evaluation must remain comprehensive. Diaphragmatic injuries may produce bowel sounds in the thorax or abnormal gastric bubble position on imaging, whereas isolated rib fractures may show localized tenderness and crepitus without the full hemodynamic profile of tension physiology. Ultimately, tension pneumothorax is distinguished from many alternative diagnoses by a characteristic constellation of bedside findings that reflect unilateral pleural pressure elevation and mediastinal compression. The combined physical findings of severe respiratory distress, hypotension, an enlarged hemithorax, ipsilaterally absent breath sounds, and contralateral tracheal deviation strongly support tension pneumothorax over pulmonary embolism, acute coronary syndromes, aortic dissection, pneumonia, pericarditis, and isolated musculoskeletal injury. Because rapid deterioration can occur, clinicians must integrate this pattern recognition with the patient's context—such as recent trauma, positive-pressure ventilation, or invasive procedures—and act decisively when tension pneumothorax remains the most immediately reversible threat [36].

**Prognosis**

The prognosis of tension pneumothorax is primarily determined by the speed of recognition and the timeliness of definitive decompression. Regardless of the initiating cause, tension pneumothorax is characterized by rapid physiologic deterioration because escalating intrapleural pressure simultaneously compromises ventilation and venous return. Without prompt intervention, the condition can progress quickly to refractory hypoxemia, obstructive shock, cardiac arrest, and death. For this reason, delays in diagnosis and treatment are strongly associated with poor outcomes, and prognosis worsens in proportion to the duration and severity of circulatory compromise before decompression is achieved. In unstable patients, the clinical imperative is therefore immediate action based on bedside assessment rather than prolonged diagnostic confirmation, as early decompression can abruptly reverse the pathophysiologic spiral and markedly improve survival. Although tension pneumothorax is distinct from uncomplicated pneumothorax, recurrence patterns of pneumothorax remain clinically relevant when counseling patients and planning follow-up. Uncomplicated pneumothorax may recur within a window of approximately 6 months to 3 years, and recurrence risk is higher in individuals who smoke or who have underlying chronic lung disease such as chronic obstructive pulmonary disease. Immunocompromised populations, including those with acquired immunodeficiency syndrome, also demonstrate higher recurrence rates, reflecting altered lung integrity and vulnerability to secondary pulmonary

pathology.[37][38] These recurrence trends emphasize that long-term prognosis is not only about surviving the acute episode but also about addressing modifiable risk factors such as smoking and optimizing management of underlying lung disease. For many patients, preventive strategies—ranging from smoking cessation to consideration of definitive procedures such as pleurodesis in recurrent cases—can significantly reduce future risk.

Prognosis also varies according to etiology and clinical context. Ventilator-associated tension pneumothorax is associated with particularly high mortality and may frequently prove fatal.[39] This poor prognosis reflects several compounding factors: mechanically ventilated patients are often critically ill with limited cardiopulmonary reserve, positive-pressure ventilation can accelerate pleural air accumulation, and clinical deterioration may occur abruptly with rapid progression to shock. Moreover, the presence of underlying severe lung pathology—such as acute respiratory distress syndrome or severe obstructive disease—can complicate both recognition and stabilization, making even prompt intervention less consistently successful. In contrast, procedure-related tension pneumothorax generally has a more favorable prognosis.[40] When tension physiology arises as an iatrogenic complication in a monitored environment, it is often detected quickly, and personnel and equipment for immediate decompression and chest tube placement are typically available. Early recognition, controlled settings, and rapid definitive management collectively improve outcomes, reducing the likelihood of prolonged hypoxia or shock. Overall, tension pneumothorax carries a prognosis that ranges from excellent to fatal depending on response time, physiologic reserve, and precipitating cause. Rapid identification and decompression are the strongest determinants of survival, while recurrence risk after uncomplicated pneumothorax is influenced by smoking status and comorbid pulmonary or immunologic conditions.[37][38] Ventilator-associated tension pneumothorax remains among the highest-risk forms with substantial mortality,[39] whereas procedure-related cases tend to have better outcomes when promptly recognized and managed.[40]

**Complications**

Tension pneumothorax is intrinsically life-threatening because it can rapidly progress to severe hypoxemia and obstructive shock. Even when patients survive the acute event, clinically meaningful complications may arise, either as direct consequences of the underlying lung and pleural injury or as iatrogenic sequelae related to emergency decompression and chest tube thoracostomy (CTT). Complications linked to the primary pathophysiology reflect the capacity of pressurized air to dissect along tissue planes and enter adjacent anatomical compartments. Pneumopericardium may occur when air tracks into the pericardial sac, potentially worsening hemodynamics and creating diagnostic confusion with tamponade physiology. Pneumoperitoneum may develop when air migrates through fascial planes into the abdominal cavity, sometimes raising concern for hollow viscus perforation and prompting further evaluation. Hemothorax can coexist with traumatic pneumothorax or occur secondary to vascular injury during thoracic instrumentation, contributing to hypovolemia, respiratory compromise, and the need for transfusion or surgical intervention. A bronchopulmonary fistula represents a persistent communication between the bronchial tree and pleural space, leading to ongoing air leak, impaired lung reexpansion, and prolonged hospitalization; in severe cases, it may necessitate surgical repair or advanced bronchoscopic interventions. CTT-related complications include damage to the intercostal neurovascular bundle, particularly when the tube is placed improperly relative to the rib margin, which can lead to bleeding, hematoma formation, and neuropathic pain. Local pain is common even with optimal placement and can limit ventilation through splinting, thereby worsening atelectasis and delaying recovery. Skin and soft tissue infection at the CTT insertion site can occur, especially with prolonged tube dwell time, inadequate dressing integrity, or immunocompromised status. More severe infectious complications include empyema, characterized by purulent pleural infection, and pyopneumothorax, in which both pus and air are present within the pleural cavity, often requiring prolonged drainage, targeted antimicrobial therapy, and sometimes operative management. These complications illustrate why post-decompression care must extend beyond immediate physiologic stabilization to include meticulous tube management, pain control, infection prevention strategies, and ongoing reassessment for persistent air leak or incomplete lung expansion. Ultimately, timely diagnosis and appropriately executed management improve outcomes by limiting both the duration of tension physiology and the downstream complications associated with delayed intervention and prolonged pleural pathology [37][38][39][40].

**Consultations**

The consultation pathway in tension pneumothorax is shaped by urgency and the need to transition rapidly from temporizing measures to definitive management and etiologic evaluation. In the acute phase, needle decompression may be performed by the clinician who first identifies the condition in a patient with respiratory distress and hypotension, because immediate relief of pleural pressure can be life-saving and cannot be deferred while awaiting specialty input. Once the patient is stabilized and definitive drainage has been established—typically through CTT—specialty

consultation becomes essential to guide further evaluation, address underlying causes, and prevent recurrence or persistent complications such as prolonged air leak. Early involvement of the appropriate specialist also supports safe decision-making about imaging, tube management strategy, and escalation to operative interventions when indicated. A thoracic surgeon or trauma surgeon is commonly consulted when the pneumothorax is large, recurrent, bilateral, associated with significant trauma, or fails to resolve with standard chest tube drainage. Surgical expertise is also critical when complications are suspected, including hemothorax requiring operative control, bronchopulmonary fistula, or persistent air leak beyond the expected timeframe. A pulmonologist is particularly valuable in patients with underlying lung disease, ventilator dependence, or complex respiratory physiology, and may assist with bronchoscopy, management of obstructive or infectious lung conditions, and planning for recurrence prevention strategies. An intensivist is frequently central to care when tension pneumothorax occurs in critically ill patients, especially those on mechanical ventilation, because ventilator settings, sedation, hemodynamics, and multi-organ support must be optimized concurrently with pleural drainage. An interventional radiologist may be consulted for image-guided drainage in anatomically challenging cases, loculated collections, or when alternative access routes are required. Overall, early referral facilitates coordinated care, refines management strategies, and improves outcomes by ensuring that definitive treatment is tailored to both the acute event and the patient's underlying risk profile [40].

**Patient Education**

Deterrence and patient education focus on reducing the likelihood of pneumothorax development and, in higher-risk individuals, preventing progression to tension physiology or recurrence. Because tension pneumothorax can arise from traumatic injury, underlying pulmonary disease, or barotrauma-related mechanisms, prevention strategies must be individualized and should emphasize modifiable risk factors. Patients should be counseled that while not all cases are preventable, proactive measures significantly reduce risk and can improve outcomes by promoting earlier presentation and faster intervention. Trauma prevention is a foundational component. Patients should be advised to take precautions during high-risk activities by wearing seatbelts, adhering to speed limits, following occupational safety protocols, and using appropriate protective equipment during contact sports or recreational activities. These measures reduce the likelihood of blunt and penetrating thoracic injury, rib fractures, and subsequent pleural disruption. For individuals with chronic pulmonary conditions such as asthma or chronic obstructive pulmonary disease, education should emphasize the importance of treatment adherence, symptom monitoring, and timely escalation during exacerbations. Effective control of airway inflammation and bronchospasm can reduce episodes of high intrathoracic pressure and air trapping that predispose to alveolar rupture and pneumothorax. For divers and aviators, prevention is closely tied to safe pressure-change practices. Patients must be educated on gradual ascent principles to prevent decompression sickness and barotrauma-associated pulmonary injury.[41] Additional precautions include avoiding alcohol prior to diving or flying, spacing out dives or flights to reduce cumulative exposure risk, refraining from air travel shortly after deep-sea diving, and maintaining good physical conditioning, which supports respiratory reserve and safe physiologic adaptation. Smoking cessation is strongly recommended because smoking increases the risk of lung pathology and spontaneous pneumothorax, and it also contributes to recurrence risk in those with prior episodes. Finally, patients should be instructed to seek prompt medical attention for new or worsening dyspnea or pleuritic chest pain, especially following trauma or recent procedures, because early recognition is central to preventing progression to life-threatening tension physiology [41].

**Other Issues**

Several practical principles—often termed "clinical pearls"—support safe and effective management of tension pneumothorax and help clinicians avoid common pitfalls. First, tension pneumothorax remains fundamentally a clinical diagnosis. Although imaging can be helpful in stable patients, treatment should not be delayed in unstable patients when the clinical pattern strongly suggests tension physiology. The condition can arise from traumatic and atraumatic causes and may occur in prehospital, emergency department, perioperative, and intensive care settings; therefore, clinicians across multiple disciplines must maintain vigilance, especially in contexts that elevate risk, such as mechanical ventilation or recent thoracic procedures. Second, hemodynamic instability combined with high clinical suspicion mandates immediate needle decompression, followed by definitive chest tube thoracostomy. This sequence reflects the core distinction between temporizing and definitive care: needle decompression rapidly relieves pressure, while CTT provides continuous drainage and reduces recurrence risk. In contrast, if the patient is stable, diagnostic imaging—most commonly a chest x-ray—may be obtained before treatment to confirm the diagnosis, assess size, and evaluate alternative explanations for symptoms. Third, patients with pulmonary conditions that predispose to high peak inspiratory pressures are at increased risk, and ventilator-associated tension pneumothorax may evolve abruptly; clinicians should therefore monitor for sudden deterioration, rising airway pressures, and unexplained hypoxemia in mechanically ventilated

patients. Fourth, CTT is sufficient for the majority of pneumothorax cases and achieves resolution in most patients when appropriately placed and managed. However, clinicians should recognize indications for escalation, including persistent air leak, failure of lung reexpansion, recurrent ipsilateral pneumothorax, bilateral pneumothoraces, or specific etiologies such as decompression sickness. In such cases, surgical approaches, including video-assisted thoracoscopic surgery (VATS) with pleurodesis, may be necessary to prevent recurrence and achieve durable resolution. Across all scenarios, the most consistent determinant of favorable outcome is the speed of diagnosis and intervention: quick recognition and prompt decompression prevent significant morbidity and reduce mortality by interrupting the progression to obstructive shock [41].

**Enhancing Healthcare Team Outcomes**

High-quality outcomes in tension pneumothorax depend on coordinated interprofessional action because the condition demands simultaneous diagnosis, resuscitation, procedural intervention, and post-procedural monitoring. The first responders play a crucial role in early identification and immediate decompression, particularly in prehospital environments where time to definitive care may be prolonged. Depending on the system, this may involve emergency medical technicians, paramedics, or designated clinicians trained to perform needle decompression. In the emergency department, emergency medicine clinicians commonly lead initial stabilization and procedural management, while intensivists typically assume primary responsibility in the intensive care unit, where tension pneumothorax frequently arises in ventilated patients and may present as sudden deterioration. Nursing staff are essential throughout the care continuum. During acute stabilization, nurses may be assigned to establish intravenous access, initiate cardiac monitoring, administer oxygen, prepare emergency equipment and medications, and provide continuous observation for rapid changes in vital signs. After initial stabilization, nurses coordinate ongoing care, manage analgesia and sedation when needed, monitor chest tube function and drainage systems, and reinforce patient education regarding warning symptoms and preventive strategies. Their surveillance role is particularly important because recurrence or tube malfunction can lead to rapid reaccumulation of pleural air. Radiologists contribute by interpreting imaging studies to determine the extent of lung collapse, identify mediastinal shift, and detect associated injuries such as hemothorax, rib fractures, or subcutaneous emphysema. This information supports decisions regarding tube position, need for additional drainage, and escalation to surgical management. Respiratory therapists provide expertise in oxygen delivery strategies, ventilator management after decompression and CTT placement, monitoring of respiratory mechanics, and titration of support to reduce barotrauma risk while maintaining adequate gas exchange. Surgeons—trauma or thoracic—often perform CTT in certain systems and are critical when pneumothorax does not resolve, when persistent air leak suggests fistula, or when operative interventions such as VATS are indicated. Pulmonologists add value in patients with underlying lung disease, those requiring prolonged ventilatory support, or those who need advanced airway and bronchoscopy-based evaluation, and they also support long-term risk reduction planning. Across these roles, collaboration and structured communication are the mechanisms by which outcomes improve. Closed-loop communication, clear role assignment during resuscitation, timely escalation to specialty care, and shared monitoring plans reduce delays and prevent preventable complications. When interprofessional teams function cohesively—from first recognition to definitive management and follow-up education—patients with tension pneumothorax are more likely to receive rapid decompression, safe chest tube management, and appropriate escalation of care, thereby improving survival and reducing long-term morbidity [41].

**Conclusion:**

Tension pneumothorax represents a time-critical emergency where survival hinges on rapid recognition and prompt decompression. The condition transforms a localized respiratory problem into a systemic crisis by simultaneously impairing ventilation and venous return, leading to obstructive shock and cardiac arrest if untreated. Clinical diagnosis should be prioritized over imaging in unstable patients, as delays can be fatal. Immediate needle decompression provides temporary relief, but definitive management requires chest tube thoracostomy to ensure continuous evacuation and prevent recurrence. Interprofessional collaboration—spanning emergency clinicians, nurses, respiratory therapists, and surgeons—is essential for safe execution of procedures, monitoring, and escalation when necessary. Preventive measures, including adherence to ventilator safety protocols, ultrasound-guided central venous access, and patient education on trauma and pulmonary health, reduce incidence and recurrence. Prognosis varies widely: ventilator-associated cases carry high mortality, while procedure-related events fare better with early intervention. Ultimately, the strongest determinant of outcome is speed—rapid diagnosis and intervention interrupt the physiologic spiral toward death, transforming a potentially fatal event into a reversible condition. Continuous training, protocol standardization, and vigilance across care settings remain the foundation for improving survival and minimizing complications.

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