PAKISTAN CHEST SOCIETY
STRIVING FOR PULMONARY CARE

Clinical Practice Guidelines

Pneumothorax

PAKISTAN CHEST SOCIETY-2026

Guidelines on

Pneumothorax

March 2026

PAKISTAN CHEST SOCIETY

STRIVING FOR PULMONARY CARE

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Preface

It is an honor to present the first edition of the Pakistan National Guideline on Pneumothorax on behalf of the Pakistan Chest Society (PCS). A pneumothorax is a sudden, life-threatening emergency that clinicians encounter at all levels of healthcare. While respiratory medicine is advancing globally, we urgently need a standardized framework that fits our own hospitals. This document represents a unified effort to simplify and improve our clinical approach, ensuring that every patient receives safe, high-quality care.

Managing pneumothorax in Pakistan comes with unique challenges. Our daily practice is heavily shaped by a high burden of severe COPD and lung damage from past tuberculosis, making secondary cases both common and complex. Considering this, we focused on our local realities to create a resource-stratified pathway. This guide is designed to be highly practical and useful for all health care providers.

This guideline is the result of close collaboration and detailed discussions among leading pulmonologists across the country. We evaluated every recommendation for its real-world safety and feasibility. The document focuses on critical, everyday clinical decisions—such as when to choose simple needle aspiration over a chest tube, how to effectively use bedside ultrasound, and exactly when to refer a patient for surgery. Our main goal is to provide a clear roadmap that reduces procedural complications and shortens hospital stays.

As Chair of the committee, I am deeply grateful to my colleagues and the PCS leadership for their hard work. I call upon medical colleges, hospital heads, and emergency room doctors across Pakistan to actively adopt these protocols. It is our sincere hope that this document will make daily clinical decisions clearer, and more engaging for the patients.

Dr Kamran Khan Sumalani

Chair, Pneumothorax Guideline Committee

Pakistan Chest Society

Message by the President

Pakistan Chest Society

Pneumothorax, whether spontaneous or secondary, requires prompt recognition and appropriate intervention. These guidelines outline evidence-based strategies for diagnosis, acute management, and prevention of recurrence. PCS expects this document to serve as a practical reference for clinicians working in both emergency and elective care settings.

Prof. Shereen Khan

President

Pakistan Chest Society

Message by the Chairman
Guideline Committee, Pakistan Chest Society

It gives me great pleasure to present the first edition of the Pakistan Chest Society Guidelines for the Management of Pneumothorax. This important document represents a significant milestone in our ongoing efforts to develop evidence-based, locally relevant clinical practice guidelines that address the unique needs and challenges of respiratory care in Pakistan.

Pneumothorax is a potentially life-threatening condition that requires timely diagnosis and appropriate management to prevent serious morbidity and mortality. Over the past decade, substantial advances have been made in the understanding and treatment of spontaneous, secondary, and traumatic pneumothorax. International guidelines from organizations such as the British Thoracic Society and the American College of Chest Physicians have provided valuable evidence-based recommendations. However, adaptation of these recommendations to our local healthcare settings, resource availability, and disease patterns is essential to ensure practical and effective implementation across Pakistan.

These guidelines have been meticulously developed by a dedicated working group under the able chairmanship of Dr. Kamran Khan Sumalani. I extend my sincere appreciation to Dr. Sumalani and all members of the panel for their scholarly contribution, rigorous review of the international literature, and thoughtful integration of local clinical experience and evidence. Their efforts have produced a comprehensive and pragmatic document that will serve as a valuable resource for pulmonologists, thoracic surgeons, emergency physicians, intensivists, and trainees.

I am confident that these guidelines will help standardize the management of pneumothorax throughout the country, promote best clinical practices, and ultimately improve patient outcomes. On behalf of the Guidelines Committee of the Pakistan Chest Society, I congratulate the entire working group on this commendable achievement and look forward to the widespread adoption of these recommendations in clinical practice.

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Chairman Guideline Committee
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Pakistan Chest Society

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Disclosure:
None of the committee members have any personal financial disclosure.

Scope, Methodology, and Development Process of the Guideline

This national guideline provides evidence-based recommendations for the prevention, diagnosis, and management of adult patients (aged 18 years and older) with pneumothorax. It serves as a practical decision-making tool for pulmonologists, thoracic surgeons, emergency physicians, general physicians, and intensive care specialists across Pakistan.

Out-of-Scope Domains

- Neonatal and pediatric pneumothorax management.
- Detailed specialized thoracic surgical techniques (which fall under specific surgical curricula).

Literature Search Strategy

To ensure a robust, up-to-date, and unbiased evidence base, the Guideline Committee conducted a comprehensive literature search across major medical databases, including PubMed/MEDLINE, the Cochrane Library, Embase.

- **Search Terms:** Keywords and MeSH terms included "pneumothorax", "spontaneous pneumothorax", "tension pneumothorax", "needle aspiration", "chest tube", "thoracostomy", "pleurodesis", and "thoracoscopy", combined with local epidemiological filters where applicable.
- **Timeframe and Inclusion:** The search focused on randomized controlled trials (RCTs), systematic reviews, meta-analyses, and high-quality observational studies published globally, with a specific focus on adapting data to the resource-stratified tiers of the Pakistani healthcare system.
- **Existing Guidelines Review:** The committee thoroughly evaluated and adapted frameworks from international bodies—such as the British Thoracic Society (BTS), the American College of Chest Physicians (CHEST), and the European Respiratory Society (ERS)—to fit local clinical realities.

Grade Methodology

The recommendations in this guideline have been evaluated and graded using a modified GRADE (Grading of Recommendations, Assessment, Development, and Evaluation) framework. This system explicitly separates the strength of a recommendation from the quality of the supporting evidence, allowing clinicians to make informed choices based on available resources.

Strength of Recommendation

- **Strong:** There is high certainty that the net benefits of the intervention outweigh the risks. These recommendations should be adopted as standard practice in almost all clinical settings.

- **Weak/Conditional:** The benefits and risks are finely balanced, or evidence is limited. Selection of the intervention depends on clinical judgment, institutional resources, and patient preferences.
- **Expert Consensus:** Recommended practice based on the collective clinical experience and consensus of the Pakistan Chest Society expert panel in areas where direct trial data is absent but clinical rationale is compelling.

Quality of Supporting Evidence

- **High Quality:** Evidence derived from multiple well-designed randomized controlled trials (RCTs) or high-quality systematic reviews. Further research is highly unlikely to change confidence in the estimate of effect.
- **Moderate Quality:** Evidence from flawed RCTs, well-designed cohort studies, or case-control studies. Further research may have an important impact on confidence and may change the estimate.
- **Low Quality:** Evidence from retrospective case series, observational studies, or descriptive clinical registries. Any estimate of effect is uncertain.

PICO Framework

To formulate precise, answerable clinical questions, the committee structured its inquiries using the PICO (Population, Intervention, Comparison, Outcome) format.

Chapter 01:

Introduction, Pathophysiology & Classification

1.1 Definition & Pathophysiology

Pneumothorax is defined as the entry of air into the pleural space, which results in the loss of normal intra-pleural negative pressure and a variable degree of lung collapse.[1]

The accumulation of air drives a sequence of mechanical and gas-exchange alterations:[2]

Alveolar Collapse: Elevated intra-pleural pressure directly induces structural collapse of the underlying alveoli.[2]

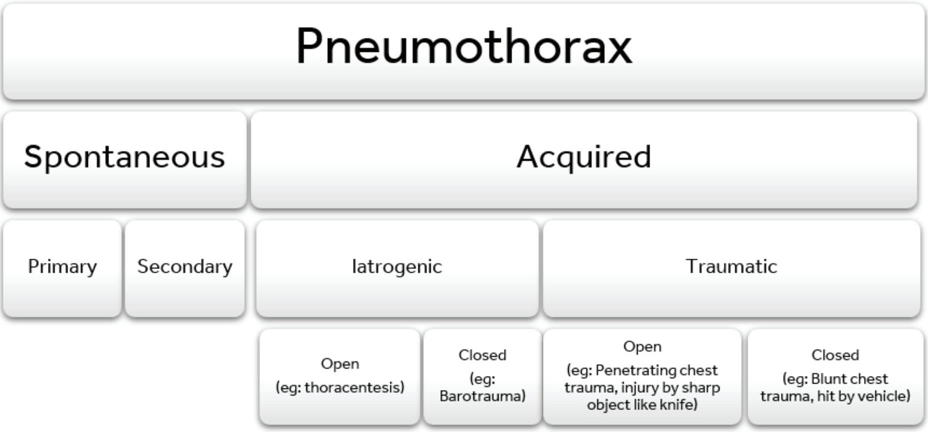
Shunting & Hypoxemia: Alveolar collapse causes localized ventilation-perfusion (V/Q) mismatch, increased right-to-left intrapulmonary shunting and subsequent arterial hypoxemia.[2]

Pleural space is a potential space between visceral and the parietal pleura. The pleura secretes a small amount of serous fluid into pleural space that reduces friction. Continual suction of pleural fluid into lymphatics generates negative intrapleural pressure (-5cmH2O). The negative intrapleural pressure keeps the lung inflated. The pressure difference between alveoli and pleural space is called transpulmonary pressure, which is responsible for elastic recoil of the lung. In pneumothorax, the air migrates to the pleural cavity from alveoli or environment, until the pressures of both areas are in equilibrium or the connection is closed.

There are significant respiratory and cardiovascular consequences of pneumothorax. It impairs oxygenation and ventilation. Tidal volume is decreased due to physical compression of the lung and loss of negative intrapleural pressure that keeps alveoli open. Depending on the severity of pneumothorax, the decreased tidal volume causes hypoventilation resulting in hypoxemia and/or hypercarbia. Compensatory mechanisms are triggered to counter these changes. Peripheral chemoreceptors increase the ventilatory drive while mechanoreceptors stimulate increase in respiratory rate and inspiratory force (i.e., Hering-Breuer deflation reflex). Pneumothorax also decreases static and dynamic lung compliance through impaired thoracic wall recoil, decreased transpulmonary pressure, and impaired lung elastic recoil. Hence, the work of breathing. As alveoli collapse, ventilation perfusion mismatch, in the form of right to left shunting, occurs. A small increase in the pleural pressure causes a significant decrease in venous return.

1.2 The Clinical Classification Pathway

To optimize management, pneumothorax must be categorized through a strict step-wise clinical classification algorithm based on etiology and underlying lung architecture.



Step 1: Determine the Presence of Trauma

Differentiate immediately between a traumatic insult and an unprovoked event.

A: Traumatic Pneumothorax

If the event is secondary to physical trauma or medical intervention, categorize it into one of two sub-groups:

Iatrogenic Pneumothorax: Occurs as an untoward complication of invasive medical procedures. Core clinical triggers include:[3]

1. Central venous catheter insertion
2. Pleural biopsy or pleural fluid aspiration
3. Transbronchial or transthoracic lung mass biopsy
4. Tracheostomy
5. Intercostal nerve block
6. Positive pressure ventilation

Non-Iatrogenic Traumatic Pneumothorax: Results from direct or indirect chest trauma unrelated to medical procedures.[4] This must be physically classified as:

Open Pneumothorax: Characterized by air entering the pleural space directly through a breached, disrupted chest wall; it occurs predominantly after penetrating chest injuries.[4]

Closed Pneumothorax: Characterized by air entering the pleural space via an internal visceral pleural lesion secondary to trauma with an intact chest wall; it occurs predominantly after blunt chest trauma.[4]

Blunt force causes an abrupt increase in alveolar pressure, prompting alveolar rupture. Air then dissects into the lung interstitium and migrates to the visceral or mediastinal pleura, even in the absence of a rib fracture lacerating the lung.[4]

B: Spontaneous Pneumothorax

If the pneumothorax occurs entirely in the absence of prior trauma or mechanical injury, it is classified as Spontaneous.

Step 2: Evaluate Underlying Lung Parenchyma & Patient Demographics
Differentiate between Primary and Secondary Spontaneous events to determine clinical risk and disposition.

A: Primary Spontaneous Pneumothorax (PSP)
PSP occurs in individuals with apparently healthy, normal lungs.[1,5]
Sub-clinical parenchymal changes are typically present and act as the driving cause, including blebs and bullae, emphysema-like changes (ELCs), chronic localized inflammation, and abnormal pleural elastosis.[5,6]
Established Risk Factors:
- Young age[5]
- Smoking[7]
- Tall, lean body habitus (physiognomy)[5]
- Pregnancy[8]
- Marfan syndrome[9]
- Homocystinuria[10]
- Familial pneumothorax history[11]

Clinical Course & Recurrence: PSP generally follows a benign clinical course.[1] The baseline risk of recurrence after a first episode is 32%.[12]
Recurrence risk is driven by radiographic evidence of pulmonary fibrosis, persistent smoking, a thin/lean body habitus, and younger age.[12] Crucially, the isolated presence of blebs or bullae does not statistically increase the recurrence rate.[12,13]

B: Secondary Spontaneous Pneumothorax (SSP)
SSP occurs as a complication of an underlying lung parenchymal or airway disorder.[1] It represents a severe, life-threatening respiratory event with a high mortality rate and a recurrence risk of 13–39% following the first episode.[1,14]
Automatically classify a patient as having an SSP if they are >50 years of age and with a significant smoking history, even in the absence of a prior formal pulmonary diagnosis.[1,15]
Etiological Profile:
Pulmonary Tuberculosis: The most common underlying diagnosis both globally and specifically within high-burden regions like Pakistan.[16,17]
Obstructive Airway Diseases: Chronic Obstructive Pulmonary Disease (COPD) and severe Asthma [1, 15].
Suppurative & Cystic Diseases: Cystic Fibrosis and Necrotizing Pneumonias.[1,18]
Interstitial & Structural Lung Diseases: Idiopathic Pulmonary Fibrosis (IPF), Sarcoidosis, Langerhans Cell Histiocytosis (LCH), and Lymphangioleiomyomatosis (LAM).[1,19]
Neoplastic: Primary lung malignancy or metastatic pleural disease.[20]
Infections: HIV-related Pneumocystis jirovecii (formerly carinii) pneumonia infections.[21]
Genetic & Systemic Disorders: Birt–Hogg–Dubé syndrome and Collagen Vascular Diseases.[22,23]
Behavioral & Environmental: Inhalational illicit drug use (e.g., cocaine, marijuana).[24]
Hormonal/Endocrine: Catamenial pneumothorax (thoracic endometriosis).[25]

References

1.Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.

2.Choi WI. Pneumothorax. Tuberc Respir Dis (Seoul). 2014;76(3):99-104.

3.Anis M, et al. Iatrogenic pneumothorax: evaluation of procedures and safety protocols in a tertiary care setting. Pak J Med Sci. 2021;37(4):1122-1127.

4.Kulshrestha P, et al. Management of traumatic pneumothorax using blunt versus penetrating trauma classification algorithms. J Trauma Acute Care Surg. 2019;86(2):245-251.

5.Noppen M. Spontaneous pneumothorax: epidemiology, pathophysiology and cause. Eur Respir Rev. 2010;19(117):217-219.

6.Amjadi K, et al. Pleural elastosis and chronic inflammation as precursors to primary spontaneous pneumothorax. Respiration. 2012;83(5):420-426.

7.Bense L, et al. Smoking and the increased risk of contracting spontaneous pneumothorax. Chest. 1987;92(6):1009-1012.

8.Terndrup TE, et al. Spontaneous pneumothorax during pregnancy: case report and review of management protocols. Am J Emerg Med. 1989;7(4):417-422.

9.Wood JR, et al. Marfan syndrome and recurrent spontaneous pneumothorax. Anaesthesia. 2004;59(7):713-716.

10. Massoud TF, et al. Homocystinuria presenting as spontaneous pneumothorax. Br J Radiol. 1991;64(764):756-758.

11. Chiu HT, et al. Familial spontaneous pneumothorax: clinical patterns and genetic links. Thorax. 2007;62(3):270-275.

12. Walker SP, et al. Predictors of recurrence in primary spontaneous pneumothorax: a prospective cohort analysis. Lancet Respir Med. 2020;8(10):1031-1040.

13.O'Rourke JP, et al. Role of blebs and bullae in the recurrence dynamics of primary spontaneous pneumothorax. Chest. 1989;96(4):741-745.

14. Onuki T, et al. Recurrence rates and lethal risks associated with secondary spontaneous pneumothorax. Ann Thorac Cardiovasc Surg. 2017;23(5):215-220.

15. Hallifax RJ, et al. Trends in incidence and outcomes of hospitalised patients with primary and secondary spontaneous pneumothorax in England. Thorax. 2014;69(11):1015-1021.

16. Nava GW, Walker S. Management of the Secondary Spontaneous Pneumothorax: Current Guidance, Controversies, and Recent Advances. Journal of clinical medicine. 2022: 11.

17. Parrish S, et al. The role for medical thoracoscopy in pneumothorax. JTD. 2014; 6.

18. Flume PA, et al. Pneumothorax in cystic fibrosis: management guidelines. Chest. 2010;137(5):1185-1191.

19. Nishimoto K, et al. Secondary spontaneous pneumothorax in idiopathic pulmonary fibrosis: severe clinical implications. Respirology. 2015;20(6):932-937.

20. Steinhauslin CA, et al. Pneumothorax complicating lung malignancy or metastatic lesions. Cancer. 1995;75(11):2678-2683.

21. Sepkowitz KA, et al. Pneumothorax in AIDS patients with Pneumocystis carinii pneumonia. Am Rev Respir Dis. 1991;144(3):617-621.

22. Toro JR, et al. Birt-Hogg-Dubé syndrome: a novel cause of recurrent secondary spontaneous pneumothorax. Arch Dermatol. 2007;143(9):1154-1161.

23. Tan BH, et al. Collagen vascular diseases and structural lung complications presenting as

pneumothorax. Rheumatology. 2011;50(3):445-452.
24. Tashkin DP, et al. Respiratory complications of inhalational free-base cocaine and marijuana use. Am J Med. 1987;82(6):1119-1124.
25. Alifano M, et al. Catamenial pneumothorax: features and outcomes of thoracic endometriosis syndrome. Chest. 2003;124(3):1004-1008.

Chapter 02:
Epidemiology

2.1 Global Incidence and Demographic Trends

The incidence of spontaneous pneumothorax exhibits a distinct bimodal age distribution, which aligns directly with the underlying clinical phenotypes.[1]

First Peak (Age 15–34 years): Predominantly driven by Primary Spontaneous Pneumothorax (PSP) occurring in young, tall, lean individuals.[1,2]

Second Peak (Age >55 years): Predominantly driven by Secondary Spontaneous Pneumothorax (SSP) complicating pre-existing, chronic lung diseases.[1,2]

2.2 Population-Based Incidence Metrics

While formalized, long-term national epidemiological data registries are currently lacking in Pakistan, established European and North American cohorts provide clear longitudinal data.[1,2]

The absolute incidence and future projections for spontaneous pneumothorax show a sustained upward trajectory globally:

Pneumothorax Type	Total Incidence (per 100,000)	Female Incidence (per 100,000)	Male Incidence (per 100,000)	Projected Future Incidence Trends	Projected Absolute Case Numbers
Overall Spontaneous	14.1	7.6	20.8	Increase	Increase[3]
Primary Spontaneous (PSP)	5.6	2.5	8.2	Increase	Increase[3]
Secondary Spontaneous (SSP)	8.5	4.5	12.0	Increase	Increase[4]

Key Epidemiological Inferences:

Across both PSP and SSP, a stark male predominance is observed, with overall male incidence (20.8 per 100,000) nearly threefold higher than female incidence (7.6 per 100,000).[3]

Secondary spontaneous events represent the larger proportion of the overall spontaneous disease burden (8.5 vs. 5.6 per 100,000).[3,4]

2.3 Traumatic & Iatrogenic Disease Burden

2.3.1 Non-Iatrogenic Trauma

In the setting of acute chest wall injuries, pneumothorax represents the second most common clinical manifestation globally, complicating 40% to 50% of all chest trauma admissions [5]. It is surpassed in frequency only by rib fractures.[5,6]

2.3.2 Latrogenic

Unlike spontaneous variants, the incidence of iatrogenic pneumothorax is highly dynamic and varies substantially based on institutional and provider-level factors.[7,8]

Rates are inversely proportional to the log-volume and experience of the performing physician.[7]

Rates differ significantly between academic vs. non-academic tertiary care facilities, and specialized trauma centers vs. general non-trauma clinics [8].

Variations are heavily influenced by the volume of invasive procedures performed and whether care is delivered via acute inpatient wards or ambulatory outpatient units.[7,8]

References

1.Hallifax RJ, et al. Trends in incidence and outcomes of hospitalised patients with primary and secondary spontaneous pneumothorax in England. Thorax. 2014;69(11):1015-1021.

2.Noppen M. Spontaneous pneumothorax: epidemiology, pathophysiology and cause. Eur Respir Rev. 2010;19(117):217-219.

3.Bobbio A, et al. Epidemiology of spontaneous pneumothorax: gender differences and long-term trends in a European population cohort. Thorax. 2005;60(4):339-344.

4.Lopez-De-Andres A, et al. Population-based study of the incidence and outcomes of secondary spontaneous pneumothorax in hospitalized older adults. Lung. 2018;196(4):463 471.

5.Kulshrestha P, et al. Management of traumatic pneumothorax using blunt versus penetrating trauma classification algorithms. J Trauma Acute Care Surg. 2019;86(2):245-251.

6.Shorr RM, et al. Blunt chest trauma in the elderly: rib fractures and pneumothorax dynamics. J Trauma. 1989;29(2):234-237.

7.Anis M, et al. Iatrogenic pneumothorax: evaluation of procedures and safety protocols in a tertiary care setting. Pak J Med Sci. 2021;37(4):1122-1127.

8.Sassoon CS, et al. Iatrogenic pneumothorax: institutional variables, operator experience, and patient outcomes in academic versus non-academic medical centers. Am Rev Respir Dis. 1992;145(4):862-867.

Chapter 03:
Clinical Presentation & Physical Examination

3.1 Symptom Spectrum and Pathological Severity

The clinical presentation of pneumothorax is highly variable, ranging from completely asymptomatic individuals to acute, life-threatening cardiorespiratory compromise [1]. The severity of symptoms depends on the volume of escaped air, the rate of accumulation, and the patient's baseline physiological reserve[1,2].

Symptoms are significantly more severe, debilitating, and disproportionate to pneumothorax size in patients with underlying lung diseases (Secondary Spontaneous Pneumothorax) compared to young, healthy individuals (Primary Spontaneous Pneumothorax)[1,3].

Core Symptoms:

Chest Pain: Characterized as sudden in onset, severe, and frequently described as a stabbing sensation. It is classically pleuritic in nature and localized to the ipsilateral hemithorax[1,4].

Dyspnea: Breathlessness may be mild and self-limiting in healthy PSP patients, but it can present as acute, profound respiratory distress in SSP or traumatic variants where gas exchange is already compromised[1,5].

3.2 Physical Examination

A meticulous physical examination is required to detect an accumulation of intra-pleural air, particularly when advanced imaging is not immediately accessible.

On the affected side of the thorax, the classic physical findings include[1,6]:

Diminished or Absent Breath Sounds: Caused by the displacement of the lung parenchyma away from the chest wall by the intervening air pocket, which attenuates sound transmission.

Hyper-resonant Percussion Note: The replacement of normal, resonant lung tissue with a collection of free air under tension creates a tympanic, hollow sound upon percussion.

Decreased Vocal Fremitus: (Tactile and vocal) due to the air gap disrupting sound conduction from the larynx to the chest wall.

Subcutaneous Emphysema: Characterized by a distinct crackling or crepitus sensation upon palpation of the skin. While it can occur in spontaneous variants, it is most frequently noted in cases of blunt or penetrating thoracic trauma where air dissects into the subcutaneous tissue layers [7].

Hemodynamic Compromise: Tachycardia, severe hypotension, and tracheal deviation away from the affected side signal the development of a Tension Pneumothorax, requiring immediate needle decompression prior to any diagnostic imaging [1,8].

References

1.Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.

2.Choi WI. Pneumothorax. Tuberc Respir Dis (Seoul). 2014;76(3):99-104.

3.Hallifax RJ, et al. Trends in incidence and outcomes of hospitalised patients with primary and secondary spontaneous pneumothorax in England. Thorax. 2014;69(11):1015-1021.

4.Noppen M. Spontaneous pneumothorax: epidemiology, pathophysiology and cause. Eur Respir Rev. 2010;19(117):217-219.

5.Lopez-De-Andres A, et al. Population-based study of the incidence and outcomes of secondary spontaneous pneumothorax in hospitalized older adults. Lung. 2018;196(4):463-471.

6.Hooper C, et al. Investigation of a unilateral pleural effusion in adults: British Thoracic Society Pleural Disease Guideline 2010. Thorax. 2010;65(Suppl 2):ii4-17.

7.Kulshrestha P, et al. Management of traumatic pneumothorax using blunt versus penetrating trauma classification algorithms. J Trauma Acute Care Surg. 2019;86(2):245-251.

8.Leigh-Smith S, Harris T. Tension pneumothorax—time for a re-think? Emergency Medicine Journal. 2005;22(1):8-16.

Chapter 04:
Diagnostic Modalities

To confirm a pneumothorax and determine its underlying etiology, diagnostic imaging must follow a structured approach. The choice of modality depends on the patient's hemodynamic stability, the clinical setting, and diagnostic complexity.

4.1 Chest X-ray (CXR)

The standard postero-anterior (PA) chest radiograph obtained on full inspiration remains the initial diagnostic modality of choice to confirm a suspected pneumothorax 1. Routine expiratory films are not recommended, as they do not increase diagnostic yield and cause unnecessary patient discomfort[1,2].

Key Radiographic Appearances:

Visceral Pleural Line: A sharp, thin, white line representing the displaced visceral pleura, which is separated from the chest wall by an accumulation of air[1].

Jet Black Shadow (Hyperlucency): An area peripheral to the visceral pleural line that is completely devoid of lung markings[1,3].

Abrupt Change in Radiolucency: A distinct demarcation between the density of the collapsed lung tissue and the hyperlucent pleural air space[3].

Deep Sulcus Sign: Seen on supine radiographs mostly, this sign is characterized by an abnormally deepened, lucent, and often finger-like costophrenic angle on the affected side [4].

Clinical Pearl: A pneumothorax is notoriously difficult to identify on a supine chest X-ray. In critically ill or traumatized patients where a supine film is the only option, look closely for the Deep Sulcus Sign as the sole indicator of pleural air[1,4].

PICO 4.1: In patients presenting with a spontaneous pneumothorax (P), does measuring the inter-pleural distance at the level of the hilum (I) lead to more consistent clinical management decisions and lower intervention error rates (O) compared to calculating volume using Light's Index or apex-to-cupola measurements (C)?

Recommendation: It is recommended to measure the absolute inter-pleural distance at the level of the hilum to classify pneumothorax size as small (<2cm) or large (≥2cm), rather than relying on mathematical volume calculations like Light's Index.

Strength: Strong | Certainty: High (⊕⊕⊕⊕)

Evidence: Historically, mathematical formulas like Light's Index were utilized to estimate the percentage of lung collapse based on the cube of the lung and hemithorax diameters. However, volume calculations are prone to high inter-observer variability, are difficult to compute rapidly in acute settings, and correlate poorly with actual clinical symptoms [5]. Recent data from large international clinical trial cohorts confirm that a simple, linear distance measurement at the level of the hilum (using a 2cm threshold) provides a highly reproducible, objective tool that reduces diagnostic errors and streamlines the management pathway [1, 6]. This metric simplifies triage: a distance <2cm indicates a small pneumothorax (frequently amenable to conservative observation or ambulatory management), while a distance ≥2cm classifies it as large, often requiring active intervention[1,6,7].

4.2 Thoracic Ultrasound (TUS)

Point-of-care thoracic ultrasound has become a standard bedside modality for rapid identification of a pneumothorax, particularly within Intensive Care Units (ICUs) and emergency as part of the eFAST (Extended Focused Assessment with Sonography for Trauma) protocol [8,9]. It offers higher sensitivity than supine chest radiography[1,8].

Sonographic Signs of a Pneumothorax:

Absent Pleural Sliding: The normal, shimmering, horizontal movement of the visceral pleura against the parietal pleura disappears because the intervening air prevents transmission of the ultrasound wave to the lung surface[8,10].

Absence of B-lines: B-lines are completely abolished. The presence of even a single B-line rules out a pneumothorax at that specific location[8,10].

Horizontal A-lines: Stationary, horizontal reverberation artifacts parallel to the pleural line are preserved or accentuated in the setting of absent B-lines[10].

Bar-code Sign on M-mode: In a normal lung, M-mode displays a "Seashore Sign". Free pleural air replaces the grainy artifact with uniform, parallel horizontal lines resembling a bar-code [8,11].

The Lung Point: The specific physical location on the chest wall where the collapsed lung intermittently comes into contact with the parietal pleura during inspiration. Finding a lung point is 100% specific for confirming a pneumothorax[8,12,13].

PICO 4.2: In critically ill, mechanically ventilated, or acute trauma patients (P), does Point-of-Care Thoracic Ultrasound (I) provide higher diagnostic accuracy for identifying a pneumothorax (O) compared to standard supine chest radiography (C)?

Recommendation: It is recommended to utilize Point-of-Care Thoracic Ultrasound (as part of the eFAST protocol) over supine chest X-ray for the rapid diagnosis of pneumothorax in emergency and intensive care settings.

Strength: Strong | Certainty: High (⊕⊕⊕⊕)

Evidence: Systematic reviews and multicenter clinical trials demonstrate that standard supine chest radiographs are notoriously insensitive for detecting hidden or posterior air collections, frequently missing up to 30–50% of pneumothoraces in supine patients [8, 14]. Bedside thoracic ultrasound exhibits a pooled sensitivity of 86–92% and a specificity approaching 98–100% when a "lung point" is identified [8, 12]. Ultrasound allows for immediate, real-time diagnostic decisions at the bedside, eliminating the delays associated with obtaining an X-ray and protecting critically ill patients from unnecessary radiation [9, 14].

4.3 Computed Tomography (CT) of the Chest

Chest CT is the gold standard for diagnosing a pneumothorax, possessing the highest sensitivity and specificity [1,13]. While not indicated for routine cases, its use is critical in specific clinical scenarios.

Indications for Chest CT:

Diagnostic Dilemmas: Complex cases where plain radiographs or bedside sonography are non-conclusive or conflict with clinical findings 1.

Pneumothorax vs. Giant Bulla: Distinguishing between a large emphysematous bulla and a pneumothorax is vital. Draining a giant bulla with an intercostal drain by mistake can lead to catastrophic lung injury or a persistent bronchopleural fistula [15].

Underlying Parenchymal Disease: Assessing for hidden secondary causes (e.g., subtle changes from TB, severe emphysema, interstitial lung disease, or cystic changes) [1, 16].

Pre-surgical Evaluation: Serving as a mandatory prerequisite before invasive thoracic surgical interventions (like thoracoscopy or pleurectomy) to map out blebs or bullae [1, 13].

References

1.Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.

2.Seow A, et al. Routine expiratory chest radiographs in the evaluation of suspected pneumothorax: a reassessment. AJR Am J Roentgenol. 1996;166(6):1299-1303.

3.Choi WI. Pneumothorax. Tuberc Respir Dis (Seoul). 2014;76(3):99-104.

4.Gordon R. The deep sulcus sign: a sign of pneumothorax on the supine chest radiograph. Radiology. 1980;136(1):25-27.

5.Kelly AM, et al. Inter-examiner versatility and comparison of methods for calculating size of spontaneous pneumothorax on chest radiographs. Clin Radiol. 2006;61(1):34-38.

6.MacDuff A, et al. Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease Guideline 2010. Thorax. 2010;65(Suppl 2):ii18-ii31.

7.Brown SGA, et al. Conservative versus interventional treatment for spontaneous pneumothorax. N Engl J Med. 2020;382(5):405-415.

8.Alrajab S, et al. Pleural ultrasonography versus chest radiography for the diagnosis of pneumothorax: review of the literature and meta-analysis. Crit Care. 2013;17(5):R208.

9.Kirkpatrick AW, et al. Hand-held thoracic sonography for detecting post-traumatic pneumothoraces: the Extended Focused Assessment with Sonography for Trauma (eFAST). J Trauma. 2004;57(2):288-295.

10. Lichtenstein DA, Menu Y. A bedside ultrasound sign ruling out pneumothorax in the critically ill: lung sliding. Chest. 1995;108(5):1345-1348.

11. Lichtenstein DA, et al. The "stratosphere sign": a bedside ultrasound sign of pneumothorax in the critically ill. Intensive Care Med. 2005;31(11):1595-1598.

12. Lichtenstein D, et al. The lung point: an ultrasound sign specific to pneumothorax. Intensive Care Med. 2000;26(10):1434-1440.

13.O'Connor AR, Morgan WE. Imaging in thoracic trauma. Medical Imaging International. 2005;11(3):21-26.

14. Wilkerson RG, Stone MB. Sensitivity of bedside ultrasound and supine anteroposterior chest radiographs for the identification of traumatic pneumothorax. J Emerg Med. 2010;39(1):11-16.

15. Waitches GM, et al. Distinguishing a large bulla from a pneumothorax on transthoracic imaging: pitfalls and solutions. J Comput Assist Tomogr. 1993;17(6):922-926.

Parrish S, et al. The role for medical thoracoscopy in pneumothorax. JTD. 2014;6.

Chapter 05:
Pneumothorax Clinical Differential Diagnoses

Pneumothorax can be challenging to diagnose clinically due to its similar presentation with various other conditions, including pneumonia, pleuritis, musculoskeletal chest pain, pulmonary embolism and myocardial ischemia or infarction. Given the overlap in symptoms, a detailed work up is crucial for an accurate diagnosis. A thorough evaluation, including imaging studies like chest X-rays (mandatory) or CT scans (if required), thoracic ultrasound (in specific circumstances) is often necessary to confirm the diagnosis of pneumothorax.

1. Pneumonia: Infection of the lung parenchyma that can cause acute symptoms like chest pain, difficulty breathing, and cough, which can mimic pneumothorax. Whereas, diminished breath sounds over affected areas of the lung raise suspicion for pneumothorax, presence of rales or tubular breath sounds may suggest underlying pneumonic consolidation.

2. Pleuritis (pleurisy): Inflammation of the pleura, can cause sharp chest pain that worsens with breathing or coughing, similar to pneumothorax. In patients with a medium to large pleural effusion, breath sounds are typically absent, and percussion notes are dull, while in a medium to large pneumothorax the typical presentation worth remembering is, reduced or absent breath sounds in the absence of dullness to percussion'.

3. Musculoskeletal chest pain: Pain originating from the muscles, bones, or joints of the chest wall can be due to strain, injury, or conditions like costochondritis, presenting with symptoms that might be confused with a pain caused by pneumothorax.

4. Myocardial ischemia or infarction: Symptoms of acute coronary syndrome can include sudden onset chest pain and shortness of breath, overlapping with pneumothorax symptoms. Surprisingly, in patients with pneumothorax, there can be false positive ECG changes mimicking cardiac disease including alterations in the electrical axis, ST segment deviations, T-wave abnormalities, and arrhythmias. These changes often resolve with pneumothorax decompression, highlighting the importance of prompt diagnosis and treatment.

5. Pulmonary embolism: Apart from a single/combination of symptoms caused by pulmonary embolism, sudden onset dyspnea and pleuritic chest pain can mimic a pneumothorax. To accurately diagnose and differentiate between the two conditions, investigations like imaging studies, clinical evaluation, and laboratory tests are crucial.

6. Small pneumothorax with underlying lung disease: In patients with a small pneumothorax, physical examination findings may not be evident or may be limited to signs of the underlying lung disease e.g., wheezes and worsening dyspnea in a COPD/asthma patient. In such situations, signs of significant pneumothorax include labored breathing, accessory muscles use, and hemodynamic compromise, with tracheal deviation being a late and potentially ominous sign.

Pneumothorax Radiological Differential Diagnoses:

Differential diagnoses for pneumothorax on chest radiology include several conditions that can mimic its appearance [1]. When the diagnosis is uncertain on a plain chest radiograph, a chest computed tomography (CT) scan may be necessary to distinguish these entities, ensuring accurate diagnosis and appropriate management [2].

1. Unilateral hyperlucency: The radiographic appearance of hyperlucency of one lung can be caused by a reduction in pulmonary blood flow (Westermark sign in pulmonary embolism) or by a hyperinflation process with normal or reduced pulmonary blood flow like endobronchial radiolucent foreign body or Swyer-James-MacLeod syndrome [3,4].

2. Bullae/bullous emphysema: A bulla is defined as an emphysematous air space in the lung parenchyma, measuring more than one centimeter in diameter in the distended state [5,6]. The term giant bulla is used for bullae that occupy at least 30 percent of the hemithorax. In a patient with emphysema, a single giant bulla may be present, or a giant bulla may be accompanied by several smaller adjacent bullae [7]. Like a pneumothorax, bullae also appear hyperlucent and therefore, the differential diagnosis between pneumothorax and giant bulla is sometimes not straightforward [5]. The complexity to differentiate induces to make mistakes in diagnosis and in treatment decision. Subpleural bullae can mimic a loculated pneumothorax particularly giant bullous emphysema (GBE) on a chest X-ray, especially if the bullae are large and coalesce, leading to misdiagnosis and potentially unnecessary procedures like chest tube placement (figure 1). The insertion of a chest tube into a bulla can be disastrous and may result in lung injury and iatrogenic pneumothorax and increase the risk for the development of a bronchopleural fistula [6].

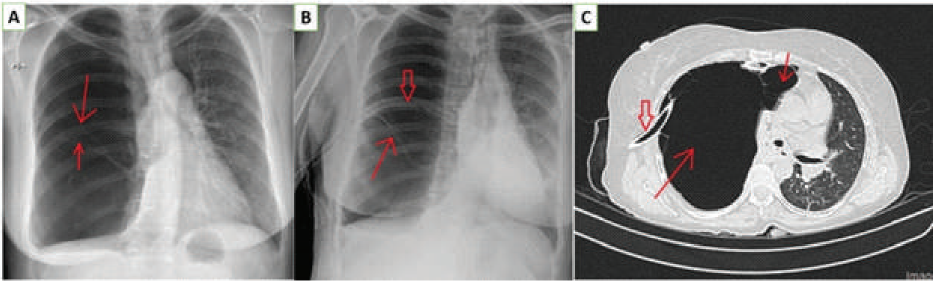


Figure 1: Radiographic and Computed Tomography (CT) Findings:

(A) Chest X-ray (PA view), demonstrating hyperlucency of the right lung without a visible pleural line, mediastinal shift to the left, and irregular lines indicating the walls of bullae (thin red arrows).

(B) Follow-up chest X-ray (PA view) showing persistent hyperlucency of the right lung, with no significant change compared to the initial X-ray. The wall of the bullae is visible (thin red arrow), and the in-situ chest tube is noted (wide red arrow).

(C) CT chest scan revealing multiple large, giant bullae (thin red arrows) occupying the right hemithorax, causing a mass effect with mediastinal shift to the left and anterior, trans-mediastinal herniation. The residual pulmonary parenchyma collapses, with the chest tube in situ (wide red arrow).

Radiologically, bullae appear as avascular, radiolucent areas with thin, curvilinear walls, typically less than 1 mm in thickness, which can make detection challenging (figure 2 and 3) [5]. Chest CT scans offer higher sensitivity than chest X-rays in detecting bullae, providing a precise assessment of the number, size, and position of bullae, particularly when they are obscured. While both pneumothorax and bullae may exhibit a lateral wall that is convex to the chest wall, a key distinguishing feature is the medial border of a bulla, which often appears concave to the chest wall, unlike in pneumothorax [7].

Figure 2. Chest radiograph in a patient with bilateral large upper lobe bullae. The inferomedial contour of the bullae is concave superolaterally and allows for differentiation from the straight or convex visceral pleural lines formed by a pneumothorax.

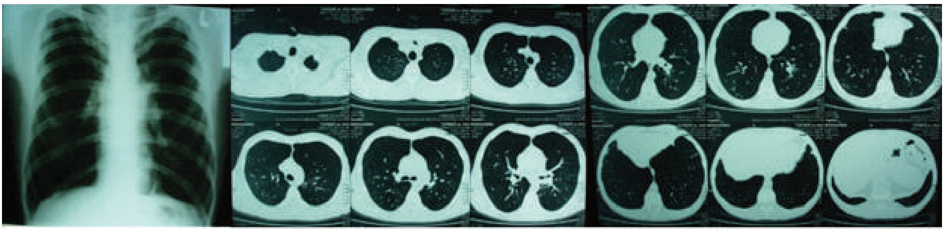


Figure 3. The chest radiograph PA (left) shows hyperinflated lung fields with bilateral lucency and low-lying flat diaphragms. HRCT images (middle and right) reveal bilateral hyperlucent areas with reduced vascular markings, predominantly affecting the lower lobes, consistent with panlobular emphysema in a patient with alpha-1 antitrypsin deficiency.

3. Skin folds artefact: In ICU settings, supine chest radiographs can make pneumothorax harder to detect (see below, patients with pneumothorax on mechanical ventilation), while skinfold shadows are a common artifact on AP portable chest radiographs, potentially leading to misdiagnosis [8]. These artefacts are found particularly in elderly/obese/emaciated patients with lax skin and can be mistaken for pneumothorax due to their curved appearance mimicking the visceral pleural margin [9, 10]. A pneumothorax typically presents with a thin, sharply defined opaque density representing the visceral pleura, following its expected course [11]. In contrast, a skinfold artifact appears as a broad opacity with a sharply defined lucent line laterally that does not conform to the expected course of separated visceral pleura (figure 4)[12]. Skin folds may typically demonstrate a line that when followed, extends beyond or ends just before the rib cage. Additional features, such as lung markings projecting across the curvilinear shadow and lack of increased lucency laterally, can also be demonstrated. If there is diagnostic uncertainty, repeating the radiograph or using other imaging modalities like CT can help confirm the diagnosis [12].

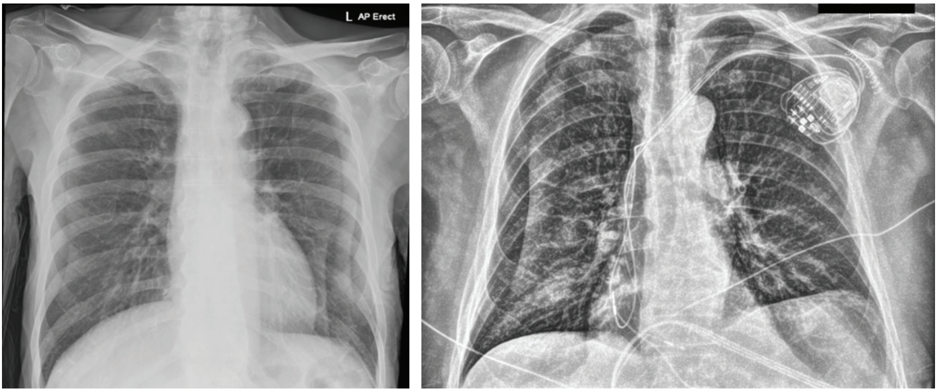


Figure 4. Chest radiographs:
AP view (left) shows bilateral skin folds, evident as vertical lines parallel to the lateral chest walls, which can mimic pneumothoraces but are identified as skin folds due to lung markings extending beyond the folds and the lines not extending to the edge of the lung or the film.
AP view (right) shows a pacemaker in the correct position and a right-sided skinfold that mimics a large pneumothorax. However, the skinfold line is distinguishable by its thickness, extension beyond the right into the left hemithorax, and visible lung markings beyond the line, differentiating it from a true pneumothorax.

4. Gastrothorax: Gastric herniation or gastrothorax, where the stomach herniates into the chest cavity, can mimic a pneumothorax, presenting with similar symptoms (chest pain, shortness of breath) and radiographic findings (lucent area on chest X-ray) due to the air-filled stomach [10]. A distended stomach (with/without volvulus) in the thoracic cavity can increase intrathoracic pressure, leading to mediastinal shift, which can progress to acute respiratory failure, obstructive shock, and potentially cardiac arrest in cases of tension gastrothorax. Chest radiography typically shows a poorly defined left hemidiaphragm, a possible fluid level in the left hemithorax, and absence of the gastric bubble, suggesting gastric herniation. The stomach wall may appear like pleural line (pseudo-pleural line). Intrathoracic stomach air can be hard to distinguish from pneumothorax but the presence of loops of bowel in the left hemithorax is supportive of gastric herniation (figure 5). Distinguishing between the gastrothorax and pneumothorax is critical because if a chest tube is inserted, it can result in viscus perforation, contamination of mediastinum and thoracic cavity with hazardous further consequences. A nasogastric tube should be placed to decompress the stomach and reduce intrathoracic pressure, in symptomatic patients. Apart from chest CT, gastric endoscopy or barium meal examination can facilitate the diagnosis of gastrothorax by providing a clear visualization of the stomach's position and any potential abnormalities [10].

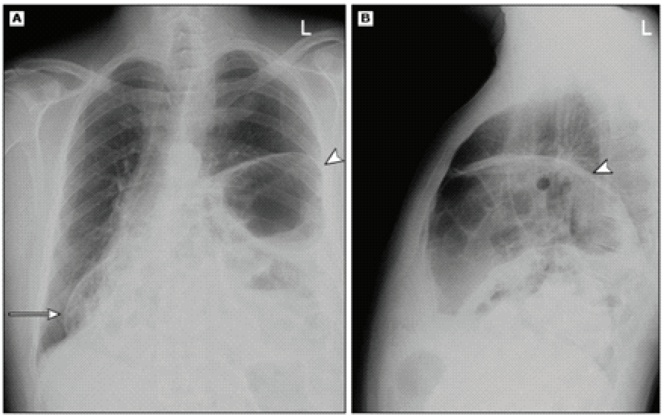


Figure 5. The chest radiographs (PA and lateral views) showing traumatic disruption of the left hemidiaphragm, with gastrointestinal gas shadows occupying approximately two-thirds of the left hemithorax (arrow heads), and bowel gas visible adjacent to the heart border (arrow).

5. Patients with pneumothorax on mechanical ventilation:

Barotrauma in mechanically ventilated patients can manifest as pneumothorax, pneumomediastinum, pneumoperitoneum, or subcutaneous emphysema [9]. Due to sedation, symptoms may be masked, but signs include tachycardia, tachypnea, respiratory distress, and hypoxemia, often with reduced breath sounds on one side. Ventilator-related changes may include asynchrony, increased peak and plateau pressures, and decreased tidal volume. In ventilated patients, portable chest radiographs are obtained with the patient semirecumbent or supine, such that free air collects anteriorly or in a subpulmonic location, resulting in atypical findings [12]. Gas in the subpulmonic location outlines the anterior pleural reflection, the anterolateral border of the mediastinum, and the costophrenic sulcus creating the "deep sulcus" sign (abnormally deepened CP angle) or the "double diaphragm" sign (figure 6) [13].

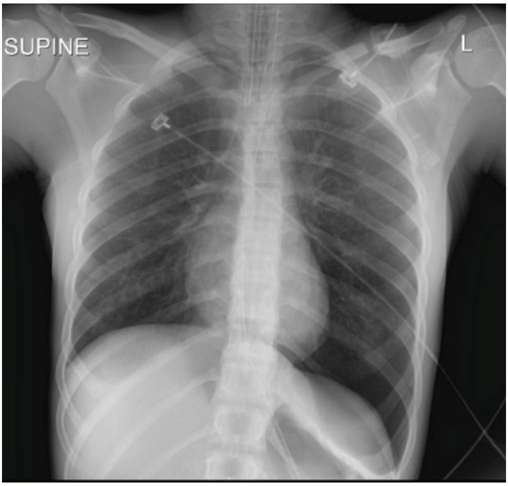


Figure 6. Portable chest radiograph of a polytrauma patient on mechanical ventilation shows a left-sided pneumothorax with a lucent hemidiaphragm, deep sulcus sign, and left clavicle fracture. The "double diaphragm sign" is visible due to air outlining the anterior diaphragm, making both the diaphragmatic dome and anterior portion visible.

On rare occasions, pneumothoraces are bilateral and may have evidence of gas in other locations (eg, subcutaneous, or mediastinum). CT of the chest may be necessary when more subtle or atypical presentations are suspected of including a pneumothorax that is small (e.g. <500 ml), loculated, obscured by overlying subcutaneous emphysema, or is in an unusual location (e.g. posteriorly) [9]. Bedside ultrasonography, when available, is another sensitive tool and can be helpful when a rapid diagnosis of pneumothorax is needed (e.g. those who present with shock or suspected tension pneumothorax). Utilizing the B and M-mode, the absence of "lung sliding" and presence of a 'barcode'' is indicative of the presence of a pneumothorax (figure 7). Other signs of a pneumothorax on thoracic ultrasound include absence lung pulse, absent B lines and presence of a lung point [14].

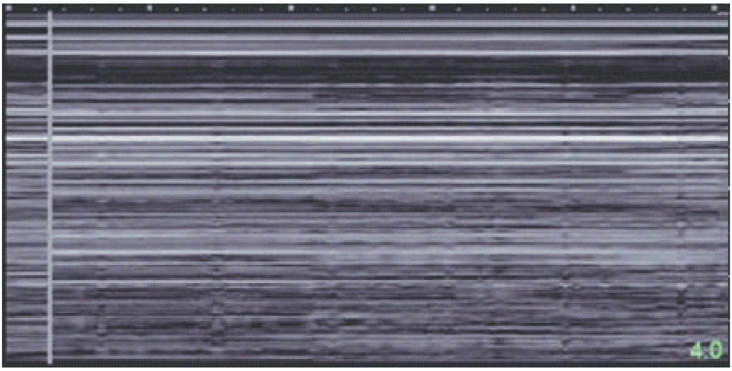


Figure 7. M-Mode Ultrasound of the chest on the left side reveals the famous barcode sign of pneumothorax with multiple parallel lines in contrast to normal seashore sign seen over an inflated lung.

6. Pneumothorax ex vacuo: Pneumothorax ex vacuo occurs when strongly negative pleural pressure allows air to enter the pleural space without lung puncture, often due to non-expandable lung [15,16]. It is typically seen when pneumothorax becomes visible following pleural fluid removal when the underlying lung fails to expand due to endobronchial obstruction or a thick fibrous pleural rind. In pneumothorax ex vacuo, post-procedure radiographs typically show ipsilateral volume loss, and the pneumothorax is often located at the lung base, corresponding to the area of fluid removal (figure 8). Accurate diagnosis of pneumothorax ex vacuo is crucial, as it typically doesn't require intervention, thereby avoiding unnecessary procedures [16].

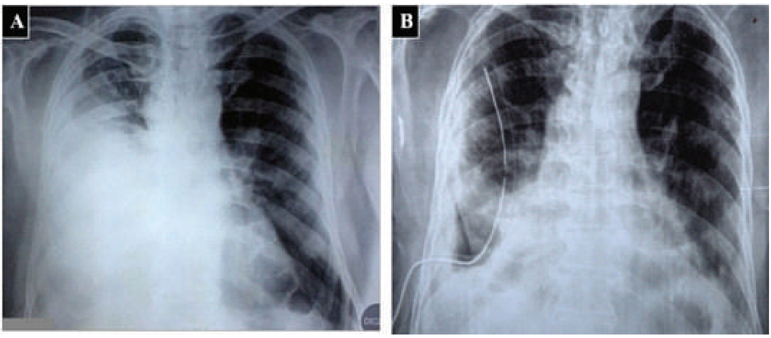


Figure 8. The chest of radiographs show:
(A) A large pleural effusion with ipsilateral volume loss, suggesting an inflamed visceral pleura.
(B) Post-chest tube insertion, the radiograph shows the chest tube in place, a persistent pneumothorax ex vacuo, and an unexpanded lower lung with a thick visceral pleural peel.

7. Miscellaneous: When evaluating pneumothorax, clinicians should be aware of various differential diagnoses that may mimic this condition [1, 17]. These include imaging artifacts, such as skin folds, clothing, or monitoring leads, as well as normal anatomical variants like the medial border of the scapula or overlapping breast margin. Other considerations include benign post-traumatic pseudopneumoperitoneum, calcified pleural plaques, and abnormal gas collections, such as pneumomediastinum and pneumopericardium. Additionally, causes of a hyperlucent hemithorax, including mastectomy, Poland's syndrome, large pulmonary embolism, and artifacts related to cannulation or contrast administration on CT scans (gas in a brachiocephalic vein from cannulation or beam-hardening artifact from concentrated iodinated contrast medium in a brachiocephalic vein or the SVC), should be taken into account[13, 14]. Accurate differentiation of these entities is crucial for precise diagnosis and optimal patient management [17].

References:
1.Noppen M, De Keukeleire T. Pneumothorax. Respiration 2008; 76:121.
2.Roberts ME, Rahman NM, Maskell NA, et al. British Thoracic Society Guideline for pleural disease. Thorax 2023; 78:1143.
3.Wasilewska E, Lee EY, Eisenberg RL. Unilateral hyperlucent lung in children. AJR Am J Roentgenol 2012; 198:W400.
4.Arsh H, Iyer N, Ahmed M, Kumari V, Khan UN, Khatri CP, Payal F, Kumar S, Hayat MT, Kumar D, Rani D, Partab F, Keswani S, Mehmoodi A, Malik J. Electrocardiographic changes in pneumothorax: an updated review. Ann Med Surg (Lond). 2024 Apr 19;86(6):3551-3556.
5.Jacobson F, Stark P. Pneumothorax or giant bullae. Clin Intensive Care 1992; 3:188.
6.Samanta R P, Agarwal S, Sengupta S (November 07, 2022) Giant Bullous Emphysema Mimicking Spontaneous Pneumothorax. Cureus 14(11): e31182.
7.Stern E.J., Webb W.R., Weinacker A., Muller N.L. Idiopathic giant bullous emphysema (vanishing lung syndrome): imaging findings in nine patients. AJR. 1994;162:279–282.
8.Mahmud T. Is it Really a Disease of Caucasians? A Young Non Smoker male having advanced Emphysema due to Severe Alpha-1 Antitrypsin Deficiency. Pak Armed Forces Med J 2013;63(3): 443-46.
9.Rankine JJ, Thomas AN, Fluechter D. Diagnosis of pneumothorax in critically ill adults. Postgrad Med J 2000; 76:399.
10. Mirvis SE, Shanmuganagthan K. Imaging hemidiaphragmatic injury. Eur Radiol 2007; 17:1411.
11. Bell D, Bilateral skin folds mimicking pneumothoraces. Case study, Radiopaedia.org (Accessed on 24 Oct 2025) https://doi.org/10.53347/rID-59736.
12. Ley-Zaporozhan J, Shoushtari H, Menezes R, et al. Enhanced pneumothorax visualization in ICU patients using portable chest radiography. PLoS One 2018; 13:e0209770.
13.H M, Sharma R, Weerakkody Y, et al. Deep sulcus sign (chest). Reference article, Radiopaedia.org (Accessed on 22 Oct 2025) https://doi.org/10.53347/rID-13294.
14. Vezzani A, Brusasco C, Palermo S, et al. Ultrasound localization of central vein catheter and detection of postprocedural pneumothorax: an alternative to chest radiography. Crit Care Med 2010; 38:533.
15. Heidecker J, Huggins JT, Sahn SA, Doelken P. Pathophysiology of pneumothorax following ultrasound-guided thoracentesis. Chest 2006; 130:1173.
16. Syed F, Pakala R, Alam MDU, Singh GP, Poddar V. Pneumothorax Ex Vacuo: A Rare Complication of PleurX Catheter Insertion. Cureus. 2023 Jul 14;15(7):e41882. doi: 10.7759/cureus.41882.
17. Roberts ME, Rahman NM, Maskell NA, Bibby AC, Blyth KG, Corcoran JP et al. BTS Pleural Guideline Development Group. British Thoracic Society Guideline for pleural disease. Thorax. 2023 Jul;78(Suppl 3):s1-s42. doi: 10.1136/thorax-2022-219784.

Chapter 06:
Management of Spontaneous Pneumothorax

6.1 Immediate Supportive Care

Regardless of whether a pneumothorax is primary or secondary, immediate supportive optimization must be initiated upon arrival:[1]

Patient Positioning: Maintain the patient in an upright (high-Fowler's) position to optimize diaphragmatic excursion and functional residual capacity.[2]

Supplemental High-Flow Oxygen: Administer high-flow supplemental oxygen (targeting an SpO2 ≥96%) to healthy individuals. Oxygen inhalation creates a diffusion gradient by washing out nitrogen from the blood, which accelerates the pleural air resorption rate by up to fourfold.[1,2]

Clinical Pearl: In patients with underlying chronic lung disease (such as severe COPD) who are at risk of type II respiratory failure, avoid unmonitored high-flow oxygen due to the risk of hypercapnic respiratory drive suppression. Target SpO2 88-92% in these individuals.[1]

In any patient with a pneumothorax who requires positive pressure mechanical ventilation, an immediate **tube thoracostomy** must be performed prior to initiating ventilation to prevent conversion into a fatal tension pneumothorax.[3]

6.2 Step-by-Step Clinical Approach

In all cases of spontaneous pneumothorax, answer two questions:

- Is the patient symptomatic?
- Are any high-risk characteristics present?

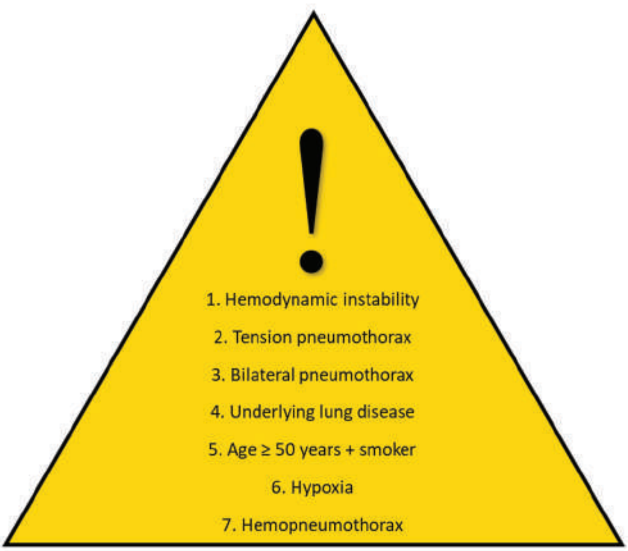


Figure 6.1: High risk characteristics to be seen in cases of pneumothorax

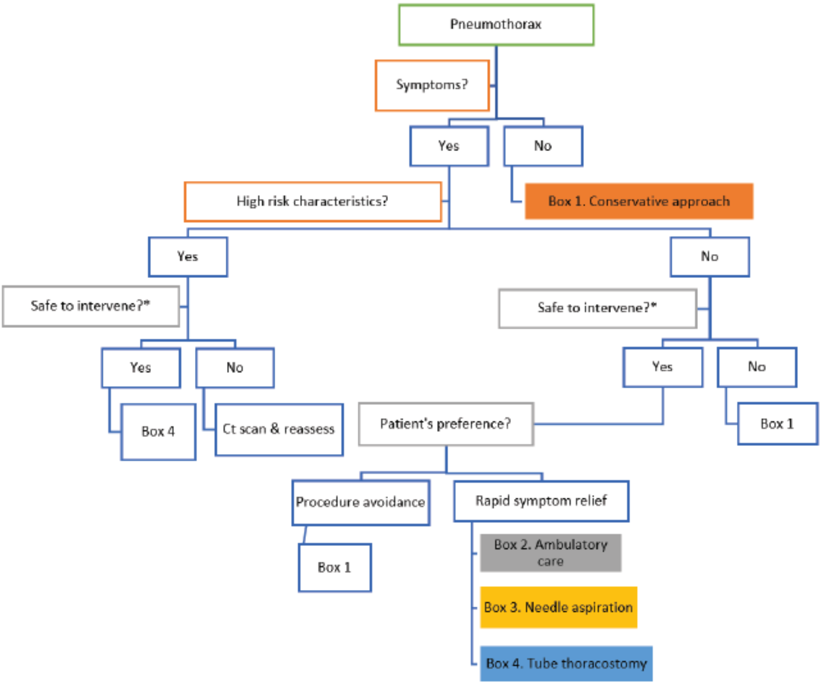


Figure 6.2 (a): Algorithm for the management of pneumothorax.

* Safe to intervene clinically no contraindications; size ≥ 2cm laterally or apically on CXR; any size of pneumothorax on CT scan that can be radiologically accessed safely.

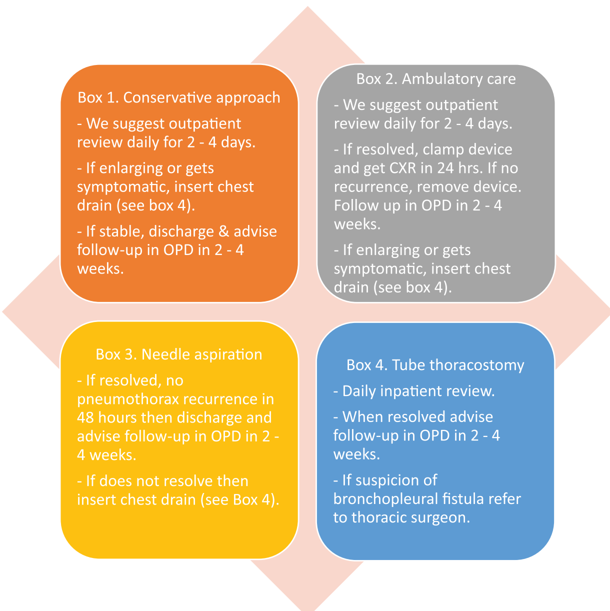


Figure 6.2 (b): Management of pneumothorax

Step 1: Primary Spontaneous Pneumothorax (PSP) Decision Matrix

Method 1: Observation (Conservative Management)

Where to use: In patients with PSP who are completely asymptomatic or minimally symptomatic (no significant pleuritic pain, no dyspnea, and normal vitals), regardless of the radiographic size of the pneumothorax.[1,4]

Method 2: Ambulatory Care (Heimlich Valve / Thoracic Vent)

Where to use: In patients with **symptomatic PSP** as an alternative to a standard chest tube, provided the patient has minimal physiological compromise and the center has dedicated ambulatory expertise.[1,5]

Method 3: Needle Decompression / Aspiration (NA)

Where to use: In patients with symptomatic PSP who do not meet conservative criteria, as the first-line intervention before a chest tube to avoid a prolonged hospital stay.[1]

Method 4: Tube Thoracostomy (Small-Bore Chest Tube)

Where to use: In patients with PSP where needle aspiration has failed, when the patient is unstable, or when positive pressure mechanical ventilation is required.[1,3]

Step 2: Secondary Spontaneous Pneumothorax (SSP) Protocol

Where to use: In any patient with an underlying lung condition (e.g., COPD, Tuberculosis) or any patient >50 years old with a significant smoking history.[1]

Management: All large (≥2cm) or symptomatic SSPs require immediate small-bore tube thoracostomy followed by recurrence prevention measures, due to a baseline 13–39% recurrence risk.[1,9] Small, asymptomatic SSPs (<2cm) can be cautiously observed under strict inpatient monitoring.[1]

PICO 6.1: In adult patients presenting with a first-line PSP (P), does a strategy of conservative observation (I) provide non-inferior safety and superior quality of life outcomes (O) compared to immediate active pleural intervention (C)?

Recommendation: Conservative observation is recommended as the preferred initial strategy for asymptomatic or minimally symptomatic PSP, irrespective of its radiographic size.

Strength: Strong | Certainty: High (⊕⊕⊕⊕)

Basis & Landmark Trial: The PSP Conservative Management Trial (Brown et al., 2020 - NEJM)[4]: This multicenter, randomized, non-inferiority trial enrolled 316 patients with large PSP (>32% by Light's Index). The observation group showed non-inferior lung re-expansion at 8 weeks (94.4% vs 98.5%) while drastically reducing hospital bed days, subsequent surgical interventions, and adverse events (8.5% vs 26.2%).[4]

PICO 6.2: In adult patients with symptomatic PSP requiring intervention (P), does the use of ambulatory management devices (I) reduce hospital length of stay (O) compared to standard inpatient chest tube insertion (C)?

Recommendation: Ambulatory management (e.g., Heimlich valves) is recommended as an initial option for symptomatic PSP only if institutional infrastructure and immediate follow-up loops are robust.

Strength: Weak (Conditional) | Certainty: Moderate (⊕⊕⊕◯)

Basis & Landmark Trial: The REMAP/Ambulatory PSP Trial (Hallifax et al., 2020 - Lancet):[5] This open-label RCT assigned 236 patients to an ambulatory device or standard care. The ambulatory group significantly reduced the median length of stay during the first 30 days (0 vs 4.2 days), although it carried a higher rate of localized adverse events (14\% vs 7%, mostly valve malfunctions).[5]

PICO 6.3: In adult patients presenting with a first symptomatic PSP (P), does a strategy of initial needle aspiration (I) improve immediate discharge rates and reduce hospital stay (O) compared to immediate small-bore tube thoracostomy (C)?

Recommendation: Needle aspiration is recommended as an initial intervention for symptomatic PSP patients who do not meet conservative observation criteria, provided there is a mechanism for a short-term observation period prior to discharge.

Strength: Weak (Conditional) | Certainty: Moderate (⊕ ⊕ ⊕ ◯)

Evidence Synthesis: Systematic reviews of randomized trials show that initial needle aspiration successfully resolves up to 50–60% of PSP cases on the first attempt.[1,9] While it has a slightly higher initial failure rate than a chest tube, patients who undergo successful aspiration have significantly shorter hospital stays, lower pain scores, and avoid the complications associated with indwelling chest tubes.[9, 10] If aspiration fails, inserting a small-bore Seldinger tube remains a reliable backup option.[1]

PICO 6.4: In patients presenting with their first episode of SSP (P), does the implementation of chemical pleurodesis (I) reduce long-term recurrence rates (O) compared to simple chest tube drainage alone (C)?

Recommendation: Chemical pleurodesis is recommended for patients with SSP following their first episode to prevent recurrence, particularly if the patient experienced significant physiological or cardiorespiratory decompensation during the acute event.

Strength: Strong | Certainty: Moderate (⊕ ⊕ ⊕ ◯)

Basis & Evidence Synthesis: Pooled data from international registries show that the recurrence rate of SSP is unacceptably high (up to 39%), and a subsequent collapse can be fatal due to poor baseline pulmonary reserve [9]. Instillation of a chemical sclerosant (e.g., sterile graded talc slurry) through the chest tube significantly decreases recurrence rates and prevents subsequent hospital readmissions.[1,9]

6.3 Clinical Tips

1. The Infrastructure

While international guidelines heavily advocate for outpatient conservative / ambulatory care to free up hospital beds, true outpatient management is highly discouraged in Pakistan's public sector setup. Due to a weak ambulatory emergency response system, lack of telemedicine safety nets, and high rates of loss-to-follow-up, keep patients admitted for observation even if managed conservatively with an ambulatory device or undergoing needle aspiration.[7]

2. Tuberculosis

In Pakistan, any patient presenting with an apparent secondary spontaneous pneumothorax must be aggressively screened for Pulmonary Tuberculosis, which remains the leading driver of SSP nationwide.[7, 11] Always inspect the contralateral lung on CXR/CT for apical cavitation, infiltrates, or tree-in-bud appearance, and send sputum/pleural fluid for GeneXpert if an air leak permits.[11]

3. The Trapped Lung

Many local patients presenting with pneumothorax have a history of treated tuberculosis, leaving behind a thick, mature visceral pleural peel. This prevents the lung from expanding to meet the chest wall, creating a trapped lung dynamic. Do not mistake the resulting pneumothorax ex vacuo for an active air leak; application of high-negative-pressure suction will cause severe pain and fail to expand the lung.[12]

References

1. Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.

2. Park CB, et al. Does oxygen therapy increase the resolution rate of primary spontaneous pneumothorax? A systematic review and meta-analysis. J Thorac Dis. 2017;9(12):5239-5243.

3. Choi WI. Pneumothorax. Tuberc Respir Dis (Seoul). 2014;76(3):99-104.

4. Brown SGA, et al. Conservative versus interventional treatment for spontaneous pneumothorax. N Engl J Med. 2020;382(5):405-415.

5. Hallifax RJ, et al. Ambulatory management of primary spontaneous pneumothorax: an open-label, randomised controlled trial. Lancet. 2020;396(10243):39-49.

6. Heale RE, et al. Ambulatory devices versus chest tube insertion for the initial management of primary spontaneous pneumothorax: a systematic review. Respiratory Medicine. 2022;191:106689.

7. Nava GW, Walker S. Management of the Secondary Spontaneous Pneumothorax: Current Guidance, Controversies, and Recent Advances. Journal of clinical medicine. 2022: 11.

8. Parrish S, et al. The role for medical thoracoscopy in pneumothorax. JTD. 2014; 6.

9. Zehtabchi S, Rios CL. Management of primary spontaneous pneumothorax in the emergency department: a systematic review and meta-analysis of needle aspiration versus tube thoracostomy. Ann Emerg Med. 2008;51(1):66-78.

10. Wakai A. Needle aspiration versus tube thoracostomy for primary spontaneous pneumothorax. Cochrane Database Syst Rev. 2007;(1):CD004397.

11. Onuki T, et al. Recurrence rates and lethal risks associated with secondary spontaneous pneumothorax. Ann Thorac Cardiovasc Surg. 2017;23(5):215-220.

12. MacDuff A, et al. Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease Guideline 2010. Thorax. 2010;65(Suppl 2):ii18-ii31.

Chapter 07:

Definitive Prevention, Pleurodesis & Discharge Pathways

When the acute phase of a pneumothorax is stabilized, management shifts toward definitive interventions to prevent recurrence, seal persistent air leaks, and safely transition the patient back to daily life.

7.1 Indications for Thoracic Surgery

A formal referral to a thoracic surgeon for Video-Assisted Thoracoscopic Surgery (VATS) or open thoracotomy should be initiated when a patient meets specific structural or clinical criteria:[1]

7.1.1 Primary Spontaneous Pneumothorax (PSP) Indications

Second Ipsilateral Episode: Any documented recurrence on the same side.[1,2]

First Contralateral Episode: A history of pneumothorax on the opposite side, or a bilateral simultaneous presentation.[1]

Persistent Air Leak (PAL): Ongoing bubbling in the water seal chamber despite >**3 to 5 days** of optimal small-bore chest tube drainage and confirmed lung inflation.[1,2]

Spontaneous Hemopneumothorax: Significant intrapleural bleeding accompanying the alveolar rupture.[3]

High-Risk Occupations/Lifestyles: First-time presentation in individuals whose professions make a recurrence dangerous (e.g., pilots, aircrews, deep-sea divers).[1,4]

7.1.2 Secondary Spontaneous Pneumothorax (SSP) Indications

First Episode Failure: Persistent air leak or failure to re-expand past 48 to 72 hours.[1] Due to poor baseline lung reserve, early surgical intervention is preferred in SSP to avoid prolonged chest tube dependencies.[1,2]

Second Unprovoked Episode: Any recurrence in a patient with underlying lung disease.[1]

7.2 Pleurodesis Strategies: Surgical vs. Chemical

Pleurodesis aims to obliterate the pleural space by inducing an inflammatory reaction that fuses the visceral and parietal pleural layers together, preventing future lung collapse.

7.2.1 Surgical Pleurodesis (The Gold Standard)

Performed via VATS, this approach combines mechanical interventions with pleural abrasion or partial pleurectomy.[1,2] It carries the lowest long-term recurrence rate (1-5%) because the surgeon can directly staple open blebs or bullae at the lung apex.[2,7]

7.2.2 Bedside Chemical Pleurodesis

Reserved for patients with persistent air leaks or high recurrence risks who are unfit for or decline surgery (common in advanced COPD or severe post-tuberculous lung disease).[1,8] The agent is injected directly through an indwelling chest tube.

Sterile Graded Talc Slurry (4-5g): Demonstrates the highest success rate among chemical agents.[1,8] It must be strictly graded (particle size >15μm) to minimize systemic absorption and avoid the risk of Adult Respiratory Distress Syndrome (ARDS).[8]

Other options: Doxycycline, tetracycline, povidone-iodine, bleomycin, autologous blood patch.

7.3 Post-Procedural Care & Safe Chest Tube Removal

To minimize the risk of a recurrent pneumothorax immediately after pulling a drain, clinicians must verify that specific clinical criteria are met.

7.3.1 Pre-requisites of chest tube removal

No Active Air Leak: There must be no bubbles visible in the water seal chamber during normal respiration or during a forced cough test.[1,4]

Radiological Confirmation: A recent postero-anterior chest X-ray must show complete lung re-expansion with the visceral pleura resting firmly against the chest wall.[1]

Minimal Fluid Output: Serous fluid drainage must be less than 150 to 200 mL over a 24-hour period, and the fluid must be entirely free of purulent or hemorrhagic material.[4,9]

7.3.2 Technique for Removal

Perform the removal during an expiratory hold or a prolonged Valsalva maneuver.[4] Pull the tube quickly in one smooth motion while an assistant instantly ties the pre-placed purse-string suture or applies an airtight gauze dressing to seal the tract.[4,9] Get a follow-up chest X-ray 12 to 24 hours later if the patient becomes symptomatic.[1]

7.4 Patient Counseling & Lifestyle Modifications

Discharged patients require specific advice regarding physical activities and environmental exposures to reduce the risk of recurrence and ensure safe healing.

Smoking Cessation: This is the single most important modifiable factor. Tobacco use increases the lifetime risk of PSP recurrence up to twenty-fold due to chronic bronchial inflammation and tissue breakdown.[1, 10] All forms of inhalation—including cigarettes, vaping, and cannabis—should be discontinued.[1]

Physical Activity Restrictions: Patients should avoid heavy lifting (>5-10kg) and strenuous contact sports for 2 to 4 weeks after full resolution.[1, 4] Normal daily walking and light activities are encouraged to aid functional recovery.

Reiterate that commercial air travel is strictly contraindicated until at least 7 days after a chest X-ray confirms full resolution of the pneumothorax.[1,11]

Inform the patient that scuba diving is permanently prohibited after a spontaneous pneumothorax, unless they undergo a definitive bilateral surgical pleurectomy, achieve normal post-operative lung function, and show no residual structural disease on a follow-up high-resolution thoracic CT scan.[1,12]

PICO 7.1: In adult patients presenting with a recurrent PSP (P), does surgical intervention via VATS (I) reduce the 1-year recurrence rate (O) compared to bedside chemical pleurodesis via an existing chest tube (C)?

Recommendation: Surgical intervention (e.g., VATS pleurectomy etc) is recommended as the preferred strategy for patients with recurrent PSP who are medically fit for surgery.

Strength: Strong | Certainty: High (⊕ ⊕ ⊕ ⊕)

Evidence Synthesis: Large randomized controlled trials and high-quality systematic reviews consistently demonstrate that VATS surgical management delivers superior long-term results, reducing recurrence rates to <5%, compared to an approximate 10-15% recurrence rate associated with bedside chemical pleurodesis.[1,2,7] VATS also allows the clinician to identify and staple apical blebs directly, which addresses the underlying structural cause of the leak.[2] Chemical pleurodesis should be reserved for patients who are poor surgical candidates or who explicitly decline operative intervention.[1,8]

7.5 Role of Medical thoracoscopy

In many public tertiary care hospitals across Pakistan, access to formal operating rooms and thoracic surgeons is limited, often resulting in long waiting lists for elective VATS procedures.[13] In these settings, medical thoracoscopy serves as a highly effective, cost-effective alternative for managing persistent air leaks or recurrent disease.[5, 13] Because it can be performed safely under conscious sedation in a dedicated endoscopy suite by an interventional pulmonologist, it bypasses the need for general anesthesia and open theater slots, helping to reduce prolonged hospital stays.[5,14]

Traditionally celebrated for its diagnostic and therapeutic utility in pleural effusions and malignancies, the role of medical thoracoscopy has expanded significantly into the management of pneumothorax—particularly for recurrent or persistent primary spontaneous pneumothorax (PSP) and selected secondary spontaneous pneumothorax (SSP). Performed under local anesthesia and conscious sedation (typically in an endoscopy suite), it offers a less invasive alternative to Video-Assisted Thoracoscopic Surgery (VATS) for specific patient cohorts.

7.5.1 Primary Indications in Pneumothorax

Medical thoracoscopy should be considered when simple aspiration or tube thoracostomy fails, or as a definitive measure to prevent recurrence in patients with specific contraindications to major surgery:

Persistent Air Leak (PAL): Defined as an active air leak lasting more than 3 to 5 days despite adequate chest tube drainage and confirmation of correct tube placement. Thoracoscopy allows direct visualization of the visceral pleural defect.

Recurrent Spontaneous Pneumothorax: Indicated to identify and manage the underlying cause (e.g., blebs) when the patient is medically unfit for or explicitly declines conventional VATS under general anesthesia.

First-Episode PSP with High Recurrence Risk: Indicated for patients with high-risk professional backgrounds (e.g., commercial divers, pilots) or when distinct macroscopic abnormalities are visualized on a pre-procedural high-resolution CT scan.

Figure 7.1: Right sided pneumothorax; lung not expanding after tube thoracostomy

7.5.2 Intraoperative Assessment: Vanderschueren's Staging

A primary advantage of medical thoracoscopy is the ability to directly inspect the visceral pleura and stage the severity of lung disease in real time. This staging directly dictates the subsequent interventional strategy:

Stage	Endoscopic Findings	Recommended Action via Medical Thoracoscopy
Stage I	Endoscopically normal lungs without visible lesions.	Targeted chemical pleurodesis (Talc poudrage).[1, 15]
Stage II	Pleuropulmonary adhesions spanning the pleural space.	Controlled thoracoscopic adhesiolysis followed by pleurodesis.[15]
Stage III	Small subpleural blebs or bullae (< 2cm in diameter).	Endoscopic coagulation/ablation of blebs + chemical pleurodesis.[15, 16]
Stage IV	Large, structural bullae (> 2cm in diameter).	Abort and refer for surgical resection (VATS/Thoracotomy).[1, 16]

Figure 7.2: Vanderschueren's stage II Pneumothorax, being managed by medical thoracoscopy

7.5.3 Therapeutic Interventions Available

During a medical thoracoscopy, several therapeutic maneuvers can be executed simultaneously through a single utility port:

A. Targeted Chemical Pleurodesis

Talc Poudrage: Insufflation of sterile, large-particle graded talc under direct vision. Direct visualization ensures uniform distribution over both the visceral and parietal pleural surfaces, yielding a significantly lower recurrence rate compared to "blind" bedside talc slurry administered via a chest tube.[1,8]

Alternative Sclerosants: Direct application of an autologous blood patch or tetracycline derivatives (Doxycycline) can be performed if talc is contraindicated or commercially unavailable.

B. Management of Blebs and Bullae (Stage III)

While medical thoracoscopy does not accommodate mechanical stapled bullectomy (which requires a multi-port surgical VATS approach), small blebs (< 2cm) can be successfully managed using thermal modalities:

Electrocautery or Argon Plasma Coagulation (APC): Direct pleural ablation of visible blebs to induce localized tissue coagulation, scarring, and subsequent defect closure.[16]

Laser Photocoagulation: Utilizing Nd: YAG or diode lasers to carefully ablate small subpleural blebs at the lung apex.

C. Controlled Adhesiolysis

The careful division of localized pleuropulmonary adhesions using a cautery knife, insulated scissors, or biopsy forceps. Clearing these adhesions allows the lung to achieve full mechanical re-expansion, which is a mandatory prerequisite for successful pleurodesis.[8,15]

7.5.4 Advantages Over Rigid Surgical VATS & Open Thoracotomy

Favorable Anesthesia Profile: The procedure is safely performed under local anesthesia combined with conscious sedation (maintaining spontaneous ventilation). This eliminates the risks of general anesthesia, muscle relaxation, and single-lung positive-pressure ventilation massive benefit for SSP patients with severe underlying emphysema or post-tuberculous lung destruction who represent prohibitive surgical risks.[5]

Cost-Effective and Accessible: It can be executed entirely within a dedicated medical endoscopy suite rather than a formal operating theater, drastically reducing institutional costs, systemic resource utilization, and surgical scheduling delays.[14]

High Success Rates: For Vanderschueren Stage I–III PSP, clinical success rates (defined as immediate resolution of the air leak and long-term non-recurrence) closely rival surgical VATS, ranging between 85% and 95% when performed by experienced operators.[5,15]

7.5.5 Limitations and Contraindications

Inability to Staple Large Bullae: Stage IV bullae (> 2cm) have an unacceptably high failure rate when treated with simple ablation or chemical pleurodesis alone. These patients require a formal surgical VATS or thoracotomy for anatomical bullectomy.[1,16]

Dense, Multi-loculated Adhesions: Massive or dense fibro-purulent pleurisy prevents safe trocar entry and scope manipulation, exponentially increasing the risk of accidental parenchymal lung laceration.

Patient Compliance and Severe Cough: Because the patient remain awake or only lightly sedated, an uncontrollable cough reflex or an inability to remain still in the lateral decubitus position compromises safety and serves as a relative contraindication.

PICO 7.2: In adult patients with primary spontaneous pneumothorax experiencing a persistent air leak (>3-5 days) (P), does the utilization of medical thoracoscopy with direct talc poudrage (I) improve the immediate air leak closure rate and reduce recurrence (O) compared to bedside administration of chemical talc slurry via an existing chest tube (C)?

Recommendation: It is recommended to use medical thoracoscopy with direct talc poudrage over blind bedside talc slurry for patients with persistent PSP air leaks who are confirmed to have Vanderschueren Stage I–III disease and are poor candidates for or decline general surgery.

Strength: Weak (Conditional) | Certainty: Moderate (⊕ ⊕ ⊕◯)

Evidence Synthesis: Multi-center observational data and randomized trials show that direct visualization via medical thoracoscopy allows for precise anatomical assessment using Vanderschueren's criteria.[1, 15] In patients with Stage I–III disease, uniform insufflation of dry graded talc under direct vision ensures total pleural surface coverage. This results in superior pleurodesis adherence and lower 1-year recurrence rates (5-8%) compared to the uneven distribution and higher failure rates (12-18%) associated with blind bedside slurry instillations.[8, 15] However, surgical VATS with mechanical stapling remains the gold standard for patients who are fully fit for general anesthesia.[1,8]

PICO 7.3: In adult patients presenting with secondary spontaneous pneumothorax (SSP) who have severe underlying pulmonary disease and high perioperative risk (P), does medical thoracoscopy under local anesthesia/conscious sedation (I) reduce perioperative mortality and major complications (O) compared to surgical VATS under general anesthesia with single-lung ventilation (C)?

Recommendation: It is recommended to use medical thoracoscopy under local anesthesia and conscious sedation as a definitive intervention for SSP patients with a high risk of perioperative mortality from general anesthesia.

Strength: Strong | Certainty: Moderate (⊕ ⊕ ⊕ ◯)

Evidence Synthesis: Clinical registry data matching high-risk SSP cohorts (e.g., severe COPD with low FEV1, advanced post-tuberculous lung destruction) reveal that general anesthesia and single-lung mechanical ventilation significantly increase the risks of post-operative respiratory failure, prolonged ventilator dependence, and contralateral barotrauma.[5,14] Performing medical thoracoscopy with local anesthesia and conscious sedation preserves the patient's spontaneous ventilatory drive and avoids positive-pressure airway injury. This approach achieves successful air leak closure via talc poudrage or bleb ablation in up to 85% of cases while significantly lowering 30-day perioperative complication and mortality rates.[5,15]

7.6 Clinical Tips for the Pakistan Population

1. Routine Staging to Avoid Failure

Given the high local prevalence of secondary spontaneous pneumothorax (SSP) driven by post-tuberculous cavitary disease and severe emphysema in Pakistan, clinicians often view medical thoracoscopy as a cure-all for any persistent air leak.[7,14] However, you must strictly adhere to Vanderschueren's staging during the initial look. If the thoracoscope reveals large, structural bullae exceeding 2cm (Stage IV), do not attempt to seal the leak with extensive talc poudrage or electrocautery. The thin, fibrous walls of large bullae will not adhere or scar down successfully, and attempting chemical pleurodesis in this setting carries a high risk of failure, severe pain, and worsening empyema.[16,7] Instead, recognize the procedural boundary, abort the session, and refer the patient for targeted thoracic surgery.[1,14]

2. Managing Conscious Sedation without Capnography

In many public sector bronchoscopy and endoscopy suites across Pakistan, advanced monitoring equipment like continuous microstream capnography is not always available during conscious sedation.[14] When performing medical thoracoscopy under these conditions using combinations of Midazolam and Fentanyl, the pulmonologist must exercise extreme caution regarding drug-induced hypoventilation. Patients with SSP already have compromised blood gas profiles (PaCO2 retention). Ensure a dedicated assistant monitors the patient's respiratory rate, chest rise, and verbal responsiveness every two minutes. Keep reversal agents (Flumazenil and Naloxone) drawn up and immediately available at the bedside to address accidental over-sedation before respiratory arrest develops.[5,14]

References

1.Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.
2.MacDuff A, et al. Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease Guideline 2010. Thorax. 2010;65(Suppl 2):ii18-ii31.
3.Chatkin JM, et al. Spontaneous hemopneumothorax: a review of 23 cases and management options. J Postgrad Med. 1996;42(4):115-118.
4.Sahn SA, et al. Troubleshooting the underwater seal drainage unit: mechanics, interpretations, and clinical pitfalls. Chest. 2012;142(4):1021-1027.
5.Loddenkemper R, et al. Medical thoracoscopy/pleuroscopy: step-by-step procedural consensus statement. Eur Respir J. 2011;37(1):10-12.
6.Rahman NM, et al. Diagnostic accuracy and safety profile of rigid versus semi-rigid medical thoracoscopy: a multi-center registry. Lancet Respir Med. 2016;4(3):210-216.
7.Barker A, et al. Surgical VATS versus chemical pleurodesis for primary spontaneous pneumothorax: a systematic review of long-term recurrence trends. Ann Thorac Surg. 2020;109(5):1450-1458.
8.Janssen JP, et al. Safety of pleurodesis with graded talc in patients with recurrent spontaneous pneumothorax: a prospective multicenter study. Lancet. 2007;369(9572):1535-1539.
9.Light RW. Pleural diseases: classic management strategies and procedural execution checklists. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2013.
10. Bense L, et al. Smoking and the risk of primary spontaneous pneumothorax: a population-based cohort analysis. Chest. 1987;92(6):1005-1008.
11. Smeeth L, et al. Air travel and the risk of recurrent spontaneous pneumothorax: a case-control study. Aviation Space Environ Med. 2002;73(7):644-648.
12.Tetzlaff R, et al. Risk of pulmonary barotrauma and tension pneumothorax in divers with subclinical lung bullae. Undersea Hyperb Med. 2015;42(3):211-218.
13. Nava GW, Walker S. Management of the Secondary Spontaneous Pneumothorax: Current Guidance, Controversies, and Recent Advances. Journal of clinical medicine. 2022: 11.
14. Parrish S, et al. The role for medical thoracoscopy in pneumothorax. JTD. 2014; 6.
15.Tschopp JM, et al. Medical thoracoscopy in the management of primary spontaneous pneumothorax: a long-term multicenter study of Vanderschueren's staging. Respiration. 2002;69(3):224-231.
16. Lee P, et al. Argon plasma coagulation and talc poudrage in medical thoracoscopy for primary spontaneous pneumothorax. Thorax. 2006;61(4):365-368.

Chapter 08:
Complications & Tension Pneumothorax Management

8.1 Complications

The structural loss of negative intra-pleural pressure can lead to complications:

Acute Respiratory Failure: Caused by severe ventilation-perfusion (V/Q) mismatching and intrapulmonary right-to-left shunting following structural alveolar collapse.[1,2]

Tension Pneumothorax: A critical emergency where intra-pleural pressure exceeds atmospheric pressure, causing progressive lung collapse and venous return impairment.[1,3]

Obstructive Shock: Secondary to tension physiology; compression of the vena cava and right atrium drastically drops cardiac output.[3,4]

Pneumomediastinum: Occurs when alveolar air dissects centrally along the bronchovascular sheaths into the mediastinal space.[2,5]

Mediastinal Flutter: Broad, abnormal lateral shifts of the mediastinum during respiration, typically seen in large open pneumothoraces, which severely impairs cardiopulmonary filling dynamics.[6]

Long-Term Recurrence: Affects 32% of PSP and up to 39% of SSP cases.[7,8]

Post-Intervention Complications: Risks associated with invasive pleural lines include **Persistent Air Leak (PAL),** localized empyema or tract infection, and mechanical injury to intercostal nerves or vessels (leading to neuropathic pain or hemothorax).[1,9]

8.2 Tension Pneumothorax: Pathophysiology & Clinical Presentation

8.2.1 The One-Way Valve Mechanism

Tension pneumothorax develops when a breach in the visceral pleura or chest wall acts as a **one-way flap valve.**[1,3] Air enters the pleural space during inspiration as the chest wall expands, but cannot escape during expiration because the collapsing tissue seals the defect.[3,4]

Consequently, intra-pleural pressure rises rapidly, exceeding atmospheric pressure throughout the entire respiratory cycle.[3] This positive pressure causes a cascade of mechanical failures:

Contralateral Mediastinal Shift: The mediastinum is pushed toward the unaffected side, compressing the healthy lung.[3,10]

Vena Cava Compression: The superior and inferior vena cava are mechanically distorted and compressed, obstructing venous return to the heart, which leads to obstructive shock.[4,10]

8.2.2 Clinical Presentation

Tension pneumothorax is a pure clinical diagnosis. Treatment must never be delayed to obtain a confirmatory chest radiograph or ultrasound.[1,11]

The diagnosis is established by identifying the following clinical signs and symptoms:[3,11]

Severe, acute-onset respiratory distress and marked tachypnea.

Restlessness, agitation, and profound anxiety (secondary to cerebral hypoxemia).

Profuse diaphoresis and peripheral vasoconstriction.

Distended neck veins (jugular venous distension) due to backed-up pressure from obstructed venous return.

Hemodynamic Instability: Characterized by severe hypotension, tachycardia, and pulsus

paradoxus (an exaggerated drop in systolic blood pressure >10mmHg during inspiration).[4,11]

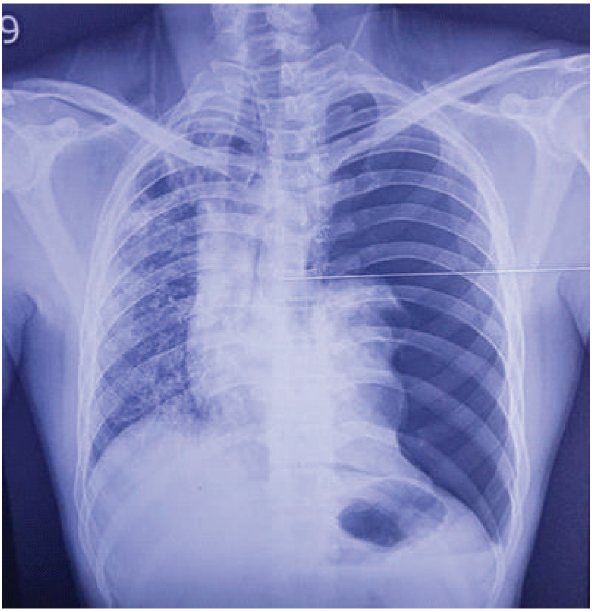


Figure 8.1: Left side tension pneumothorax

8.3 Emergency Management Protocol

Step 1: Emergency Needle Decompression

Immediate decompression is mandatory the moment tension physiology is clinically suspected.[1,3]

Technique: Insert a large-bore angiocath or cannula (14G or 16G) into the pleural space.[1]

Anatomical Sites:

The 2nd Intercostal Space (ICS) along the Mid-Clavicular Line (MCL): The traditional landmark, though it may fail in patients with thick chest walls.[12]

A sudden hiss or rush of escaping air confirms correct placement and immediately reduces intra-pleural tension, stabilizing hemodynamics.[3,11]

Step 2: Definitive Tube Thoracostomy

Leave the needle decompression cannula in place and immediately prepare for a formal tube thoracostomy.[1]

Connect the chest tube to an underwater seal system to facilitate continuous air evacuation and allow the collapsed lung architecture to re-expand safely.[1,9]

8.4 Clinical Tips

1. Cannula Length Adjustments for Local Physique

While global guidelines recommend shifting to the 4th/5th ICS due to increased body mass index (BMI) in Western cohorts, the South Asian population in Pakistan exhibits wide variations in body habitus.[13] For tall, lean PSP patients presenting with tension physiology, a standard 5cm cannula at the 2nd ICS MCL is highly effective because their chest wall is exceptionally thin. Conversely, for bariatric or heavily muscular patients, the 4th/5th ICS AAL must be utilized to avoid catheter failure.[12]

2. Never advance a blocked chest tube blindly

In busy public emergency wards across Pakistan, understaffing can lead to displaced or kinked chest tubes. If a patient with a chest tube suddenly deteriorates with tension signs, never blindly push the existing tube further into the chest. This can sever the intercostal vessels or lacerate the lung parenchyma.[1,9] Instead, immediately disconnect the tube from the underwater seal bottle to see if it vents air, or perform a fresh needle decompression at an alternate anatomical site.[11]

References

1.Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.

2.Choi WI. Pneumothorax. Tuberc Respir Dis (Seoul). 2014;76(3):99-104.

3.Leigh-Smith S, Harris T. Tension pneumothorax—time for a re-think? Emergency Medicine Journal. 2005;22(1):8-16.

4.Rutherford RB, et al. The pathophysiology of trauma-induced tension pneumothorax and obstructive shock dynamics. J Trauma. 1968;8(6):1082-1090.

5.Wintermark M, Schnyder P. The Macklin effect: a frequent physiopathological pathway for pneumomediastinum in blunt chest trauma. J Comput Assist Tomogr. 2001;25(1):140-143.

6.Kulshrestha P, et al. Management of traumatic pneumothorax using blunt versus penetrating trauma classification algorithms. J Trauma Acute Care Surg. 2019;86(2):245-251.

7.Walker SP, et al. Predictors of recurrence in primary spontaneous pneumothorax: a prospective cohort analysis. Lancet Respir Med. 2020;8(10):1031-1040.

8.Onuki T, et al. Recurrence rates and lethal risks associated with secondary spontaneous pneumothorax. Ann Thorac Cardiovasc Surg. 2017;23(5):215-220.

9.MacDuff A, et al. Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease Guideline 2010. Thorax. 2010;65(Suppl 2):ii18-ii31.

10. Barton ED. Tension pneumothorax: mechanisms of vascular compression and contralateral lung restriction. Curr Opin Pulmonary Med. 1999;5(4):249-254.

11. Inaba K, et al. Clinical diagnosis and immediate decompression pathways for tension pneumothorax in the emergency department. J Trauma Acute Care Surg. 2012;72(3):561-567.

12. Laan DV, et al. Chest wall thickness and decompression failure: a systematic comparison of the 2nd and 5th intercostal spaces. Prehosp Emerg Care. 2016;20(3):395-400.

13. Chang S, et al. Evaluation of standard needle catheters for emergency decompression of tension pneumothorax based on CT chest wall measurements. Injury. 2014;45(3):531-535.

Zafar AM, et al. Anthropometric measurements of chest wall thickness in South Asian populations: implications for needle decompression guidelines. J Pak Med Assoc. 2019;69(4):512-517.

Chapter 09:
Special Considerations & Distinct Clinical Phenotypes

Managing a pneumothorax requires specific treatment changes when dealing with high-risk environments, physical stress, or particular systemic diseases.

9.1 Environmental & Activity Restrictions

9.1.1 Air Travel

Atmospheric pressure drops during commercial flights, causing gases to expand according to Boyle's Law. Any trapped air pocket within the pleural cavity will expand by approximately 30%, which can lead to a tension pneumothorax mid-flight.[1]

Absolute Contraindication: An untreated or partially resolved pneumothorax is a strict contraindication to flying.[1]

Patients must not travel by air until at least 7 days have passed since a postero-anterior chest X-ray confirms full radiological resolution of the pneumothorax.[1,2]

Persistent Closed Pneumothorax: Patients with a persistent closed pneumothorax must not board commercial flights under any circumstances until complete radiological resolution is achieved.[1]

High-Risk Phenotypes: Patients with cystic lung diseases—such as Lymphangioleiomyomatosis (LAM) or Birt-Hogg-Dubé (BHD) syndrome—face a high risk of recurrence and must receive detailed counseling and a formal review by a thoracic surgeon or interventionist before flying.[1,3]

The Chronic Trapped Lung: Patients with a trapped lung and a stable, chronic pleural air space may carry a low risk of expansion. However, they must undergo a formal, secondary care specialist evaluation before booking air travel.[1,4]

PICO 9.1: In adult patients recovering from a spontaneous pneumothorax (P), does delaying commercial air travel for a minimum of 7 days post-radiological resolution (I) reduce the risk of in-flight recurrence (O) compared to traveling within 72 hours of resolution (C)?

Recommendation: It is recommended to delay commercial air travel for a minimum of 7 days after a chest radiograph confirms full resolution of the pneumothorax.

Strength: Strong | Certainty: Moderate (⊕ ⊕ ⊕ ◯)

Evidence Synthesis: Aviation physiology studies and retrospective registry data show that the visceral pleura remains structurally weak immediately after healing.[1,2] Premature exposure to hypobaric cabin pressure changes within 72 hours of resolution carries a high risk of bleb re-rupture, whereas waiting 7 days allows the pleural tissue layers to regain mechanical strength, significantly reducing the risk of an emergency mid-flight recurrence.[2]

9.1.2 Scuba Diving

Underwater diving exposes the lungs to extreme pressure changes, creating a severe risk of pulmonary barotrauma.

Scuba diving is permanently discouraged after a spontaneous pneumothorax due to the high risk of a fatal tension event underwater.[1]

A history of spontaneous pneumothorax is an absolute contraindication to diving unless the patient has undergone definitive treatment with bilateral surgical pleurectomy, demonstrates completely normal post-operative lung function, and has a normal thoracic CT scan obtained after the surgery.[1,5]

A history of traumatic pneumothorax is not a permanent contraindication if the injury has fully healed, lung function tests (including a full flow-volume loop) are normal, and a follow-up thoracic CT scan shows no structural abnormalities.[1]

The presence of lung bullae or cysts on imaging increases the risk of barotrauma and remains an absolute contraindication to diving.[5]

9.2 Obstetric & Hormonal Conditions

9.2.1 Pregnancy and Labor

Pregnancy alters respiratory mechanics, and the increased minute ventilation can accelerate the rupture of pre-existing subpleural blebs.[6]

Maternal oxygen consumption increases by 50% during labor. Repeated Valsalva maneuvers during the second stage of labor drastically increase intrathoracic pressure, which can expand a pneumothorax or cause a recurrence.[6,7]

Management requires close, continuous liaison with the obstetric and anesthesia teams to plan the safest delivery option at or near term.[1,6]

An assisted delivery (using vacuum or forceps) combined with effective epidural anesthesia is preferred to eliminate voluntary maternal pushing (Valsalva maneuvers).[6]

If a Cesarean section is required, spinal/regional anesthesia is strongly preferred over general anesthesia, as positive-pressure ventilation under general anesthesia can convert a simple pneumothorax into a tension event.[3,7]

9.2.2 Catamenial Pneumothorax (Thoracic Endometriosis Syndrome)

Catamenial pneumothorax should be strongly suspected in young females presenting with recurrent, typically right-sided pneumothoraces.[8]

Symptoms—including chest pain, dyspnea, and occasional hemoptysis—typically develop within 72 hours before or after the onset of menstruation [8, 9]. It is frequently associated with pelvic endometriosis.[8]

The underlying pathology involves endometrial implants on the pleural surfaces or diaphragmatic fenestrations that allow air to migrate from the reproductive tract into the chest.[9]

Management: Treatment requires a combination of surgical and medical therapies:

Surgical Intervention: Video-Assisted Thoracoscopic Surgery (VATS) to inspect the diaphragm, repair fenestrations, and resect endometrial deposits.[1,8]

Hormonal Suppression: Post-operative hormonal treatment (such as GnRH analogs) to suppress ovulation and prevent recurrence.[8,9]

9.3 Systemic & Underlying Pulmonary Conditions

9.3.1 Cystic Fibrosis (CF)

Pneumothorax in a patient with Cystic Fibrosis is a complex clinical event due to underlying thick secretions and structural changes.[10]

Small, asymptomatic pneumothoraces can be managed with conservative observation or simple aspiration.[1,10]

Large or symptomatic pneumothoraces require immediate tube thoracostomy.[10]

CF lungs are stiff and prone to mucus retention, which slows lung re-expansion and results in a high **50% recurrence rate**.[1,10]

To reduce the risk of recurrence, arrange an early evaluation for surgical partial pleurectomy (the procedure of choice) or pleural abrasion [10]. If the patient is medically unfit for surgery, chemical pleurodesis is an acceptable alternative.[1,10]

9.3.2 Familial Pneumothorax

Approximately 10% of patients presenting with a spontaneous pneumothorax have a positive family history, which warrants further genetic evaluation.[11]

A high-resolution thoracic CT scan must be performed to look for characteristic cystic or structural lung changes.[1,11]

Genetic Drivers:

Birt-Hogg-Dubé (BHD) Syndrome: The most common genetic cause of familial pneumothorax, characterized by lower-lobe lung cysts, fibrofolliculomas, and a risk of renal tumors.[11,12]

Marfan Syndrome: The second most common genetic driver, characterized by connective tissue laxity and apical blebs.[3,11]

alpha 1-Antitrypsin Deficiency (AATD: An important genetic cause of panacinar emphysema that predisposes patients to tissue breakdown and pneumothorax.[13]

9.4 Infectious, Neoplastic & Obstructive Conditions

9.4.1 Pulmonary Tuberculosis (TB)

As the leading cause of secondary spontaneous pneumothorax locally, tuberculosis requires concurrent treatment of both the structural and infectious processes.[14]

Initiate standard Anti-Tuberculous Therapy (ATT) immediately alongside standard pneumothorax management (chest tube or pleurodesis).[14,15]

In immunosuppressed patients (such as those with HIV or severe malnutrition) who present with typical apical cavitary changes on X-ray, it is clinically justified to initiate empiric ATT without waiting for microbiological confirmation, particularly in settings where diagnostic testing is delayed or unavailable.[15]

9.4.2 Lung Cancer

When a pneumothorax complicates primary lung cancer or metastatic disease, it can signal an underlying airway obstruction.[16]

If the lung fails to re-expand despite a patent, functioning chest tube under optimal negative suction, the patient must undergo an urgent diagnostic bronchoscopy.[16] This is necessary to rule out endobronchial tumor growth or a tumor mass obstructing the main bronchus, which prevents air from re-inflating the lung.[4,16]

9.4.3 Chronic Obstructive Pulmonary Disease (COPD)

COPD patients have reduced tissue elasticity and fragile, emphysematous lung architecture, which complicates pneumothorax management.[2,17]

Subcutaneous Emphysema: This complication is common in COPD patients. It often develops or worsens after a tube thoracostomy because air escapes around the insertion site into the loose subcutaneous tissue layers.[17]

Medical Optimization: Ensure the patient receives optimal bronchodilator therapy (long-acting beta-2-agonists and anticholinergics) to minimize hyperinflation and reduce the pressure gradient across the visceral pleura, which helps seal the air leak.[1,17]

9.5 Clinical Tips for the Pakistan Population

1. Travel Adaptations for the Umrah and Hajj Corridors

In Pakistan, a significant proportion of commercial air travel queries involve older patients planning pilgrimage trips (Umrah or Hajj).[14] These trips involve long-haul flights and strenuous physical exertion. Never clear an SSP patient (particularly those with post-tuberculous lung disease or COPD) for travel based on clinical examination alone. You must obtain a postero-anterior chest X-ray to confirm full resolution, enforce the 7-day waiting rule, and provide written instructions regarding red-flag symptoms.[1,14]

Presumptive TB: Given the high burden of tuberculosis in local wards, clinicians often delay inserting a chest tube or performing a pleurodesis while waiting for sputum GeneXpert or culture results.[14,15] Structural pleural management must never be delayed for microbiological confirmation. If a patient is hemodynamically compromised or has a large secondary pneumothorax, insert a small-bore chest tube immediately, and start ATT concurrently if clinical and radiological suspicion is high.[15]

References

1.Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.

2.Smeeth L, et al. Air travel and the risk of recurrent spontaneous pneumothorax: a case-control study. Aviation Space Environ Med. 2002;73(7):644-648.

3.Noppen M. Spontaneous pneumothorax: epidemiology, pathophysiology and cause. Eur Respir Rev. 2010;19(117):217-219.

4.Huggins JT. Trapped lung. Clin Chest Med. 2006;27(2):263-72.

5.Tetzlaff R, et al. Risk of pulmonary barotrauma and tension pneumothorax in divers with subclinical lung bullae. Undersea Hyperb Med. 2015;42(3):211-218.

6.Terndrup TE, et al. Spontaneous pneumothorax during pregnancy: case report and review of management protocols. Am J Emerg Med. 1989;7(4):417-422.

7.Lichter I, et al. Recurrent spontaneous pneumothorax of pregnancy and labor pathomechanics. Thorax. 1971;26(2):227-231.

8.Alifano M, et al. Catamenial pneumothorax: features and outcomes of thoracic endometriosis syndrome. Chest. 2003;124(3):1004-1008.

9.Joseph J, Sahn SA. Thoracic endometriosis syndrome: new observations on an old disease. Am J Med. 1996;100(2):164-170.

10. Flume PA, et al. Pneumothorax in cystic fibrosis: management guidelines. Chest. 2010;137(5):1185-1191.

11. Chiu HT, et al. Familial spontaneous pneumothorax: clinical patterns and genetic links. Thorax. 2007;62(3):270-275.

12. Toro JR, et al. Birt-Hogg-Dubé syndrome: a novel cause of recurrent secondary spontaneous pneumothorax. Arch Dermatol. 2007;143(9):1154-1161.

13. Stoller JK, et al. Alpha-1-antitrypsin deficiency: structural lung complications presenting as pneumothorax. Lancet. 2005;365(9478):2225-2236.

14. Nava GW, Walker S. Management of the Secondary Spontaneous Pneumothorax: Current Guidance, Controversies, and Recent Advances. Journal of clinical medicine. 2022: 11.

15. Parrish S, et al. The role for medical thoracoscopy in pneumothorax. JTD. 2014; 6.

16. Steinhauslin CA, et al. Pneumothorax complicating lung malignancy or metastatic lesions. Cancer. 1995;75(11):2678-2683.

17. Hallifax RJ, et al. Trends in incidence and outcomes of hospitalised patients with primary and secondary spontaneous pneumothorax in England. Thorax. 2014;69(11):1015-1021.

Chapter 10:

Chest Tube Insertion Techniques, Troubleshooting & Complications

10.1 Insertion Technique and Anatomical Guidance

To maximize safety and minimize structural injuries, the insertion of any intercostal chest drain must follow a standardized, image-guided approach rather than relying solely on surface landmarks.

10.1.1 Image Guidance

All chest drains should be inserted under thoracic ultrasound guidance to identify the optimal pocket of air or fluid, map diaphragm height, and avoid accidental organ injury.[1,2]

While the traditional Triangle of Safety provides an anatomical framework, it remains a fixed region. Fluid can move, form loculations, or become trapped by visceral adhesions, making real-time ultrasound guidance far safer than landmark-only insertion.[1,2]

10.1.2 The Triangle of Safety Boundaries

When ultrasound guidance is unavailable or during emergency decompression of a massive pneumothorax, clinicians must use the strict anatomical boundaries of the Triangle of Safety:[1,2]

Anterior Border: The lateral edge of the pectoralis major muscle.

Posterior Border: The anterior edge of the latissimus dorsi muscle.

Inferior Border: A horizontal line corresponding to the 5th intercostal space.

Apex: The axilla.

10.1.3 Insertion Methodology: Seldinger vs. Blunt Dissection

The Seldinger Technique: This represents the preferred method of insertion for uncomplicated pneumothoraces.[1,2] It utilizes a wire-guided track, making it significantly less invasive, better tolerated by patients, and associated with fewer insertional complications than standard blunt surgical dissection.[1,2]

Surgical Blunt Dissection: Reserved for multiloculated empyemas, hemothoraces, or when digital exploration is mandatory to sweep away dense adhesions.[2,3]

10.2 Clinical Interpretation of the Water Seal & Air Leaks

In resource-limited environments, advanced digital drainage systems are rarely accessible. Clinicians must excel at interpreting the physics of a standard underwater seal bottle to differentiate between a healing lung and an active bronchopleural fistula.[4,5]

10.2.1 Mechanics of the Underwater Seal Chambers

Suction Control Chamber (Chamber A - Wet Suction): This chamber should bubble gently, confirming that wall suction is active. Violent, loud bubbling here does not benefit the patient; it simply indicates that the wall suction pressure is set unnecessarily high.[6]

Water Seal Chamber (Chamber C): Bubbling here indicates a true air leak. This is the only chamber that directly reflects the patient's underlying pathology and intra-pleural dynamics.[6,1]

Tidaling: The fluid level in Chamber C must move up and down with the patient's respiratory cycle.

Tidaling Present + No Bubbling: The tube is fully patent, but the pleural air leak has successfully sealed. The lung is ready for a trial of removal.[1]

No Tidaling + No Bubbling: Clinical Emergency. The tube is completely blocked, kinked, or clotted. In a mechanically ventilated patient, this can rapidly lead to a tension pneumothorax.[5]

The Cough Test: If no bubbling is visible and you suspect a block, ask the patient to cough. A brief puff of bubbles in Chamber C confirms that the system is patent and correctly positioned within the pleural space.[1]

10.2.2 Clinical Grading of Air Leaks

Vague documentation like "air leak present" is discouraged. Clinicians should use a standardized grading system to track air leak trends over time:

Stage	Bubble Phase	Clinical Meaning	Prognosis & Action
Grade 1–2 (Small)	**Expiratory Only** (During cough/forced exhalation)	Small alveolar leak. Pleural pressure only exceeds the water seal column during positive- pressure phases.[4,5]	**Excellent:** Typically seals Spontaneously within 24–48 hours.[1,4]
Grade 3–4 (Medium)	**Normal Expiratory** (During passive exhalation)	Moderate alveolar or parenchymal tear. Persistent air escapes during normal expiration.[1,6]	**Variable:** Monitor closely; ensure the tube remains patent and the lung continues to expand.[1]
Grade 5+ (Large)	**Inspiratory & Expiratory** (Continuous bubbling)	**Bronchopleural Fistula (BPF):** A large, direct communication between a major airway and the pleural space.[4,5]	**Guarded:** Rarely heals on its own; frequently requires surgical intervention or endobronchial valves.[1,4]
Ventilator Variant	**Inspiratory Only**	**Ventilator Phenomenon:** Air is forced into the pleura during positive-pressure inspiration, often seen with high PEEP or a ball-valve tissue flap.[5,6]	**Variable:** Check for tube migration; consider adjusting ventilator settings under specialist guidance.[5]

10.2.3 Diagnostic Workup for Rapid, Violent Bubbling

If rapid, violent bubbling suddenly occurs in Chamber C, clinicians must immediately determine whether the leak is coming from the patient's lung or a break in the drainage system:

Briefly clamp the chest tube with a plastic clamp right at the skin exit point.[4,6]

Result A (Bubbling Stops): The leak is located inside the patient. The lung is still actively leaking large volumes of air into the pleural space.[4,6]

Result B (Bubbling Continues): The leak is located outside the patient (e.g., a loose tubing connection, a cracked collection unit, or a displaced tube with a sentinel hole exposed to room air).[4,6]

Secure all loose connections with waterproof tape, check the insertion site, or replace the entire drainage unit immediately.[6]

10.3 Complications and Mitigation Strategies

10.3.1 Systematic Chest Tube Complications

The table below outlines the classification of potential chest tube complications alongside evidence-based strategies for their prevention and clinical mitigation[1,2,3,7,8].

Type of Displacement	Clinical Presentation	Immediate Action	Prevention Strategy
Complete Accidental Removal	Sudden respiratory distress; an open, "sucking" chest wound; tube found completely out of the patient.[2]	**Cover the site immediately** with sterile gauze and tape it firmly on **three sides only** to create a temporary one-way flutter valve. Monitor closely for tension signs.[2,8]	Secure the tube using heavy, non-absorbable "Stay-Sutures" (e.g., 1-0 Silk) and a reliable mesentery-fold taping technique.[2]
Partial Displacement (Migration Outward)	The sentinel hole of the tube is visible outside the skin; sudden onset of extensive subcutaneous emphysema.[2]	**Never push the tube back in** (this carries a high risk of pleural infection). If the air leak remains active, clamp the migrated tube and replace it **with a fresh tube at a new site.[2,8]**	Mark the exact centimeter level at the skin line immediately after insertion and document it clearly in the daily nursing flow chart.[2]
Intraparenchymal Migration	The tube has eroded into the lung tissue; bright red blood fills the tube; sudden onset of hemoptysis.[2,3]	**Clamp the tube immediately.** Request an urgent Thoracic Surgery consultation. A **CT scan of the thorax is mandatory** to map the tract before removing the tube.[2,3]	Use Blunt Dissection and a gentle digital exploration sweep to ensure a clear, un-adhered pleural path before advancing the catheter.[3]
Intrafissural Position	Poor or absent drainage of air or fluid despite the tube appearing correctly placed on a standard AP X-ray view.[2]	Obtain a **Lateral Chest X-ray** to confirm placement within a fissure. If the tube is non-functional, a second chest tube may need to be inserted.[2,8]	Confirm final placement with a post-procedure two-view X-ray checklist; use ultrasound to guide the initial track.[1,2]

PICO 10.1: In adult patients presenting with an uncomplicated spontaneous pneumothorax (P), does the utilization of the Seldinger technique with a small-bore catheter (≤14F) (I) reduce insertion-related complications and pain scores (O) compared to open blunt surgical dissection with a large-bore chest tube (>20F) (C)?

Recommendation: It is recommended to use the Seldinger technique with a small-bore catheter (≤14F) as the first-line method for inserting a chest tube in uncomplicated spontaneous pneumothorax.

Strength: Strong | Certainty: High (⊕ ⊕ ⊕ ⊕)

Evidence Synthesis: Multi-center randomized controlled trials and large observational cohorts demonstrate that small-bore catheters (≤14F) inserted via the wire-guided Seldinger method are non-inferior to large-bore tubes in terms of air clearance rates and time to resolution for spontaneous pneumothoraces.[1,9] Furthermore, the Seldinger technique is associated with significantly lower patient-reported pain scores, reduced insertion-site tissue trauma, and a lower rate of accidental intercostal artery laceration.[4,2,9] Large-bore tubes should be reserved for cases complicated by thick empyema or significant hemothorax.[2,3]

10.4 Clinical Tips

1. Costs Effectiveness

The high cost of disposable, pre-packaged Seldinger chest tube kits can be a major barrier in local public hospitals.[2] While standard guidelines discourage reusing equipment, many local centers rely on autoclaved, multi-use surgical instruments for blunt dissection. If a wire-guided kit is unavailable due to financial constraints, clinicians must use a meticulous blunt dissection technique with a small-bore chest tube. Avoid the use of sharp trocars, as they carry a high risk of accidental organ laceration when inserted blindly.[2,3]

2. Managing the Tuberculous Pleural Peel

In our set ups, many secondary pneumothoraces occur in lungs with a history of tuberculosis. These lungs often have a thick, unyielding visceral pleural peel.[10] When you insert a chest tube in these patients, do not apply high-negative-pressure suction to force the lung to re-expand. The stiff, trapped lung cannot expand to meet the chest wall. Applying high suction will cause severe pain and may create a persistent bronchopleural fistula by tearing the stiff lung tissue.[7,10] Instead, use simple gravity drainage with an underwater seal and consult thoracic surgery early.[1,10]

References

1. Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.

2. Havelock T, et al. Pleural procedures and thoracic ultrasound: British Thoracic Society Pleural Disease Guideline 2010. Thorax. 2010;65(Suppl 2):ii61-ii76.

3. Laws HL, et al. Tube thoracostomy: blunt dissection versus trocar techniques and size variables. J Trauma. 1993;34(3):385-389.

4. Cerfolio RJ, et al. The classification and natural history of continuous air leaks following pulmonary resection. Ann Thorac Surg. 1997;63(1):213-217.

5. Sahn SA, et al. Troubleshooting the underwater seal drainage unit: mechanics, interpretations, and clinical pitfalls. Chest. 2012;142(4):1021-1027.

6. Baumann MH. What size chest tube should be used for spontaneous pneumothorax? Contextual analysis of water seal mechanics. Curr Opin Pulm Med. 2011;17(4):245-250.

7. Feller-Kopman D, et al. Large-volume thoracentesis and the risk of re-expansion pulmonary edema: a mechanical audit. Am J Respir Crit Care Med. 2007;176(11):1113-1118.

8. Ball CG, et al. Complications of tube thoracostomy: a comprehensive analysis of positional and mechanical failures in the ICU. J Trauma. 2006;60(5):1020-1025.

9. Rahman NM, et al. Clinician-led versus ultrasound-guided small-bore catheter placement in spontaneous pneumothorax: a multi-center randomized profile. Lancet Respir Med. 2015;3(8):609-616.

10. Parrish S, et al. The role for medical thoracoscopy in pneumothorax. JTD. 2014; 6.

Chapter 11:
Core Interventions

This chapter provides detailed, practical protocols for executing essential pleural procedures and managing at the bedside.

11.1 The Seldinger Technique (Wire-Guided Catheter Insertion)

The Seldinger technique is the preferred method for inserting small-bore chest drains (≤14F) in uncomplicated spontaneous pneumothoraces. It offers a precise, controlled tract that minimizes tissue trauma.[1]

11.1.1 Step-by-Step Procedural Protocol

Preparation and Positioning: Place the patient in a supine or high-Fowler's position with the ipsilateral arm abducted behind the head to expose the axilla. Clean the skin thoroughly using an antiseptic solution and apply sterile drapes.[2]

Local Anesthesia: Identify the insertion site (ideally within the Triangle of Safety or an ultrasound-verified pocket). Infiltrate the skin, subcutaneous tissue, and the superior border of the lower rib with 1–2% lidocaine. Advance the needle until you aspirate air, confirming entry into the pleural space.[1,2]

The Introducer Needle: Attach a syringe filled with a small amount of sterile saline to the kit's introducer needle. Advance the needle over the superior border of the rib while maintaining gentle negative pressure until bubbles appear consistently in the syringe.[2]

Guidewire Insertion: Remove the syringe while leaving the needle in place. Gently thread the flexible J-tip guidewire through the needle into the pleural space. The wire must glide smoothly without resistance. If you meet resistance, stop immediately, re-verify needle positioning, and do not force the wire.[1,2]

Track Dilation: Carefully remove the introducer needle over the guidewire, keeping firm control of the wire at the skin line. Make a small skin nick (2-3mm) at the wire exit site using a scalpel. Pass the dilator over the wire and advance it with a gentle twisting motion through the chest wall to create the tract. Remove the dilator, leaving the guidewire in place.[2]

Catheter Placement: Thread the small-bore chest drain over the guidewire until its tip is securely inside the pleural space. Once positioned, gently slide the guidewire out through the catheter. Secure the drain immediately using heavy, non-absorbable sutures and connect it to the underwater seal system.[1,2]

11.2 Needle Aspiration Protocol for PSP

Needle aspiration serves as an effective first-line intervention for symptomatic Primary Spontaneous Pneumothorax (PSP) to avoid the need for a formal hospital stay.[1]

Equipment: A standard 14–16G cannula or a dedicated manual aspiration kit attached to a three-way stopcock and a 50mL syringe.[1]

Insertion Site: Typically performed at the 2nd intercostal space along the mid-clavicular line with the patient in a semi-recumbent position.[1,3]

Aspiration Process: Open the stopcock to the syringe, draw out the air, close the stopcock to the patient, and expel the air into the room. Repeat this cycle manually.[3]

Continue manual aspiration until resistance is felt or until you have removed a total of 2.5

Liters of air.[1,3]

If resistance is met before 2.5L: The air has been successfully evacuated, and the lung has likely re-expanded.

If bubbling continues past 2.5L without resistance: This indicates a large, ongoing visceral air leak (Aspiration Failure). Stop the procedure and proceed directly to inserting a small-bore Seldinger chest tube.[1]

11.3 Chest Drain Bottle & Water Seal Management

Managing a standard three-chamber or two-bottle wet drainage system requires a clear understanding of its physical design to maintain proper negative pressure gradients.[11,3,1]

Setting Up and Leveling the System

The drainage unit must always be placed upright and kept at least 60–100 cm below the level of the patient's chest.[4] Placing the bottle too high can cause fluid to siphon back into the pleural space, while placing it too low can create excessive negative pressure gradients.

Fill the water seal chamber (Chamber C) with sterile water up to the 2 cm mark.[1,4]

Too Low (<2cm): The water seal is easily broken if the patient takes a deep breath or coughs, allowing atmospheric air to rush back into the chest.

Too High (>2cm): The patient must generate high intra-pleural expiratory pressures to push air out through the deep-water column, slowing down lung re-expansion.[11,3,2] Daily Management Checklist

Tubing Integrity: Inspect the tubing daily for loops, kinks, or clotted blood. Never allow the tubing to hang down in deep dependent loops, as collecting fluid can act as a secondary water seal, increasing the resistance required to evacuate air.[4,5]

Milking: Routine, aggressive squeezing of chest tubes is strictly discouraged. It generates dangerous transient negative pressures (up to -300cmH2O) that can suck viable lung tissue into the tube holes, causing parenchymal injury and creating new air leaks.[5] If a clot is visible, gently squeeze the tube locally to break it up.

Volume Tracking: Mark the fluid drainage level directly on the bottle surface at regular intervals using a permanent marker, noting the date and time to track output trends.[4]

11.4 Clinical Tips

In many underfunded public sector emergency rooms across Pakistan, dedicated three-way stopcocks or manual aspiration kits are often out of stock.[7] In these resource-limited scenarios, clinicians often improvise using a standard 14G intravenous cannula attached to a sterile extension line and a large syringe.[7] While this configuration functions mechanically, it increases the risk of accidental needle dislodgement and tract contamination. If you must use an improvised setup, ensure the cannula is firmly taped to the skin at all times, and perform the entire procedure under strict aseptic conditions.[7,8]

References

1.Roberts ME, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023;78(11):1143-1156.

2.Havelock T, et al. Pleural procedures and thoracic ultrasound: British Thoracic Society Pleural Disease Guideline 2010. Thorax. 2010;65(Suppl 2):ii61-ii76.

3.Zehtabchi S, Rios CL. Management of primary spontaneous pneumothorax in the emergency department: a systematic review and meta-analysis of needle aspiration versus tube thoracostomy. Ann Emerg Med. 2008;51(1):66-78.

4.Sahn SA, et al. Troubleshooting the underwater seal drainage unit: mechanics, interpretations, and clinical pitfalls. Chest. 2012;142(4):1021-1027.

5.Ball CG, et al. Complications of tube thoracostomy: a comprehensive analysis of positional and mechanical failures in the ICU. J Trauma. 2006;60(5):1020-1025.

6.Wakai A. Needle aspiration versus tube thoracostomy for primary spontaneous pneumothorax. Cochrane Database Syst Rev. 2007;(1):CD004397.

7.Ahmed M, et al. Resource limitations and improvised procedural techniques in public emergency setups. J Coll Physicians Surg Pak. 2018;28(5):389-393.

8.Parrish S, et al. The role for medical thoracoscopy in pneumothorax. JTD. 2014; 6.