HANDBOOK OF MECHANICAL VENTILATION

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Foreword
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3rd Edition

JAYPEE

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CHAPTER

Respiratory Assessment and Clinical Manifestations

ANATOMY

The respiratory tract consists of a series of branching airways that extend down to the alveolar region. The initial 16 airway divisions, which include the trachea, bronchi, and bronchioles, along with the nose, pharynx, and larynx, constitute the anatomical dead space because they are not involved in gas exchange. Exchange of gases takes place within the thin-walled alveoli located in the respiratory bronchioles (generations 17–19) and within the alveolar ducts and sacs (generations 20–23).

PHYSIOLOGY

Ventilation, or minute volume, refers to the amount of air moving into and out of the lungs per minute. A portion of this air remains in the anatomical dead space and does not take part in gas exchange. The portion of air that reaches the alveoli is termed alveolar ventilation. In a healthy adult, the anatomical dead space is around 0.15 L. With an average resting tidal volume of 0.5 L and a breathing rate of 15 breaths per minute, the resting minute ventilation equals 7.5 L/min, while effective alveolar ventilation is about 5.25 L/min.

The diaphragm and external intercostal muscles are the primary inspiratory muscles. During times of increased demand, the scalene and sternocleidomastoid muscles assist by elevating the ribs. Contraction of these muscles enlarges the thoracic cavity, making the intrapleural pressure more negative. This increases the pressure gradient across the lung, causing it to expand. As the alveoli expand, alveolar pressure decreases, leading to air inflow.

In quiet breathing, expiration occurs passively due to the elastic recoil of the lungs. Intrapleural pressure becomes less negative compared to inspiration, though it remains below atmospheric pressure. Forced expiration—such as breathing out beyond functional residual capacity (FRC), rapid exhalation, or coughing—involves activation of expiratory muscles. The abdominal wall muscles are especially important, as they increase intra-abdominal pressure and push the diaphragm upward into the thoracic cavity.

The effort required by respiratory muscles to maintain ventilation depends on two main factors—the elastic resistance of the lungs (their stiffness or compliance) and the resistance to airflow within the airways. Under normal conditions, both resistances are low, but they commonly increase in disease states.

Elastic resistance arises from the lung tissue's elastin fibers and the surface tension within alveoli. High elastic resistance reflects reduced compliance, making the lungs stiffer. Airflow resistance, on the other hand, is largely determined by airway radius. Although smaller airways have higher individual resistance, their large number arranged in parallel lowers the overall resistance of peripheral generations. The highest total airway resistance occurs in the segmental bronchi, roughly between generations 2 and 5.

PULMONARY INSUFFICIENCY

The degree of respiratory failure that may occur when the exchange of respiratory gases between the circulating blood and the ambient

atmosphere is impaired. This impairment can be caused by disease or trauma that, to some degree, anatomically alters the lung and chest wall.

Clinical information may be gained by inspecting the chest. The majority of this information should be gathered without the patient's knowledge. This is done by listening and observing the patient.

Things to look for: Is the patient short of breath while talking? Note the use of accessory muscles and retractions. Note the patient's posture, respiratory rate, and pattern, pursed-lip breathing, grunting, or nasal flaring. Also note symmetry of the chest, skin color, nail beds, digital clubbing, and type of cough (dry, hacking, loose, and wet). Is the cough productive, and if it is, how much? When is the cough present (morning, night, and all the time)? What time of the year is the cough present? Note the color of the sputum, audible wheezing or rhonchi, evidence of edema, or dehydration.

This information is the basis for proper treatment of the patient with respiratory problems.

ANXIETY ASSESSMENT FINDINGS

The patient's fear of being in a hospital or physician's office, or just the fear of the unknown, can affect the patient's physical findings. The appearance, conversation, behavior, and vital signs may dramatically increase when anxiety is present. A thorough patient interview and physical examination are necessary for prompt and proper treatment. Help reduce anxiety by asking open-ended questions. When needed, obtain additional information from the patient's family, if possible.

The following are things to look for in patients with anxiety:

- *Appearance:* Muscular tension, pale, clammy skin, fatigue, and restlessness.
- *Conversation:* Does the patient frequently ask questions? Shifts the topic of discussion, describes fears with a sense of helplessness, and avoids focusing on his feelings.

  "Though most of the symptoms listed may be caused by factors other than anxiety, a thorough physical examination with proper laboratory work can give you a good overall clinical picture."
- *Behavior:* Does the patient have a shortened attention span? Does the patient have difficulty following directions?
- *Physiologic signs:* Increased heart rate, respiratory rate, blood pressure, and perspiration.

RESPIRATORY BREATHING PATTERNS

Eupnea: Normal respiratory rate and rhythm with occasional deep breath.

Tachypnea: Increased respiratory rate as seen in patients with fever, pneumonia, respiratory insufficiency, and lesions of the brain's respiratory control center.

Bradypnea: Slower respiratory rate but regular, affected by narcotics, tumors, alcohol, and during regular sleep.

Apnea: Absence of breathing, which may be periodic.

Hyperpnea: Deeper than regular breaths but with a normal rate.

Cheyne-Stokes: Slower respiratory rate that gets faster and deeper than usual, with periods of apnea of 20–60 seconds. Causes are increased intracranial pressure, severe congestive heart failure, meningitis, and drug overdose.

Blot's: Faster and deeper respiratory rate with abrupt pauses between breaths. Each breath has the same depth. Causes are spinal meningitis or other central nervous system conditions.

Kussmaul's: Faster and deeper than normal respiratory rate without pauses. Breathing sounds

are labored, with deep breaths that resemble sighs. Causes may be renal failure or diabetic ketoacidosis.

Apneustic: Prolonged gasping inspiration, followed by short, inefficient expiration. Lesions in the respiratory control center can cause it.

DIAGNOSTIC APPROACH TO A PATIENT WITH DYSPNEA

Causes

Cardiac

- Left ventricular failure
- Mitral valve disease
- Cardiomyopathy
- Pericardial effusion or constriction

Noncardiorespiratory

- Psychogenic
- Anemia
- Hemorrhage
- Acidosis
- Hypothalamic lesions

Respiratory

- *Airways disease:*
  - Chronic bronchiolitis and emphysema
  - Asthma
  - Bronchiectasis and cystic fibrosis
  - Laryngeal or pharyngeal tumor
  - Bilateral lordosis palsy
  - Cricoarytenoid rheumatoid
  - Tracheal obstruction
  - Tracheomalacia
  - Amyloid of airways
- *Parenchymal disease:*
  - Allergic alveolitis
  - Sarcoidosis
  - Fibroses + diffuse alveolitis
  - Obliterative bronchiolitis
  - Pneumonias + toxic pneumonitis
  - Diffuse infections
  - Respiratory distress syndrome
  - Infiltrative + metastatic tumor
  - Pneumothorax
- *Pulmonary circulation*
  - Pulmonary embolism + hypertension
  - Pulmonary arteritis + thrombosis
- *Chest wall and pleura*
  - Effusion or pleural fibrosis
  - Fractured ribs
  - Ankylosing spondylitis
- Kyphoscoliosis
- Neuromuscular and bilateral diaphragm paralysis
- Hematologic
- Psychiatric
- Renal
- Skeletal, endocrine, and rheumatologic

A comprehensive history and physical examination are required for the diagnosis of dyspnea.

History

- Onset/intermittent/progressive
- Nocturnal
- Relation to exercise/provoking factors
- Aggravating + relieving factors
- Orthopnea/paroxysmal nocturnal dyspnea (PND), etc.

Physical Examination

- Respiratory rate
- Body habitus
- Posture
- Use of pursed lips
- Accessory muscles
- Clubbing
- Emotional state
- Chest expansion
- Intensity of breath sounds
- Pedal edema?
- Congestive cardiac failure (CCF)
- Thromboembolic diseases

- Signs of pulmonary arterial hypertension or right
- Ventricular failure

Laboratory Evaluation

Hemoglobin

- Anemic bleeding
- Polycythemia chronic hypoxia
- Renal, metabolic, and thyroid
- Chest X-ray (CXR), spirometry, and electrocardiogram (ECG)

Special Test

- Pulmonary function testing
- Arterial blood gas (ABG)

Cardiopulmonary exercise testing

- Pulmonary or cardiovascular (CVS) system
- Unrelated

Spiral computed tomography

- Ventilation perfusion scan (pulmonary embolism)
- Gallium—*Pneumocystis carinii* infection
- High-resolution computed tomography (HRCT)—interstitial lung disease

Cardiac Cause

- ECG and radionucleotide scan
- Cardiac catheterization
- Rule out psychiatric cause (anxiety)

LOCATIONS FOR PLACEMENT OF THE STETHOSCOPE

Figures 3.1 and 3.2 show the frontal and dorsal placement of the stethoscope.

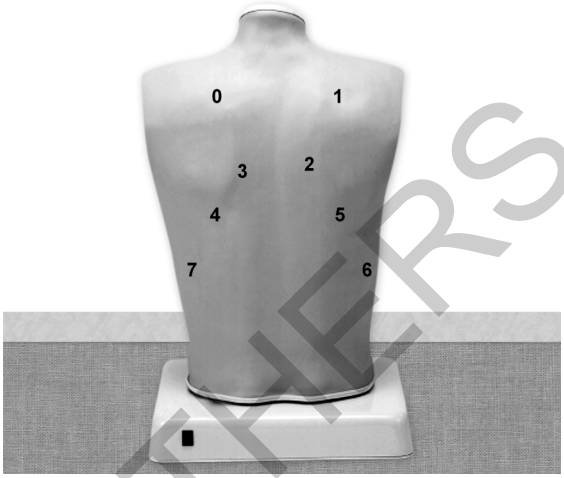

Fig. 3.1: Stethoscope placement—frontal. Note that the locations for placement of the stethoscope are the exact general locations for percussion. (*For color version, see Plate 2*)

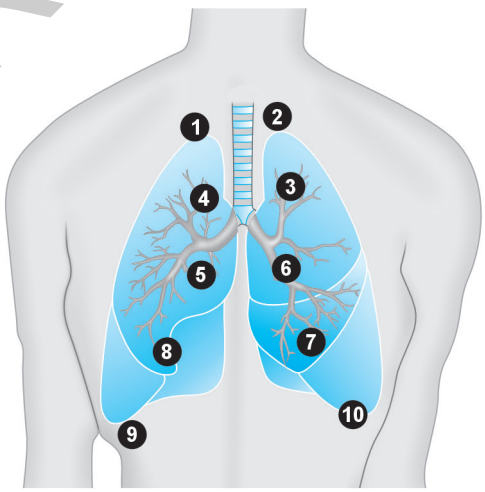

Fig. 3.2: Stethoscope placement—dorsal.

Breath Sounds

Abnormal

The results of a movement by pulmonary physicians 5 years ago to change terms, such as rates, sibilant, musical, and so on, to more descriptive terms have made breath sounds easier to describe and easier to explain **(Table 3.1)**.

Normal

Vesicular breath sounds are heard over most of the chest, except over the central airways.

TABLE 3.1: Terms used now and before 1982 for abnormal breath sounds.

Recommended terms	*Terms used before 1982*
Crackles	Rales or crepitation
Wheezes	Sibilant rales, musical rales, and Sibilant rhonchus
Rhonchi	Low-pitched wheeze and sonorous rales
Stridor	Stridor (no change).

The pitch is low in tone. The inspiratory-to-expiratory ratio is 1:2. The sound is said to be breezy.

Tracheal breath sounds are heard over the trachea. The pitch is very high in tone. The inspiratory-to-expiratory ratio is 5:6. The sound is said to be loud, harsh, and tubular.

Bronchial breath sounds are heard over the major central airways. They are high in pitch, with an inspiratory-to-expiratory ratio of 2:3 and are described as hollow or tubular in sound.

Bronchial-vesicular breath sounds are heard over the major central airways. The pitch is of medium tone, with an inspiratory-to-expiratory ratio of 1:1, and the sound is breezy.

CLASSIFICATION OF DYSPNEA

- *Class I:* Dyspnea only on severe exertion (appropriate).
- *Class II:* Can keep pace with persons of the same age and body size on a level surface without breathlessness, but not on hills or stairs.
- *Class III:* Can walk a mile at own pace without dyspnea but cannot keep pace on a level surface with a normal person.
- *Class IV:* Dyspnea present after walking about 100 yards on a level surface with a normal person or on climbing one flight of stairs.
- *Class V:* Dyspnea on even less activity or even at rest.

TABLE 3.2: Sputum observation.

Appearance	*Possible causes*
Mucoid, clear, thin, and frothy	Bronchial asthma, legionnaires disease, pulmonary tuberculosis, emphysema, and early chronic bronchitis
Mucopurulent and yellow-green	All of the above and infection, pneumonia, and cystic fibrosis
Purulent, yellow, thick, and viscid	Bronchiectasis, advanced chronic bronchitis, and *Pseudomonas* pneumonia
Apple green, thick pink, and thin blood-streaked	Haemophilus influenzae pneumonia *Streptococcus* pneumonia
Blood-rust	*Klebsiella* pneumonia, pneumococcal pneumonia, lung abscess, bronchiectasis, anaerobic infections, and aspiration
Black	Smoke and coal dust
Frothy pink	Pulmonary edema
Blood	Pulmonary emboli with infarction, tuberculosis, abscess, trauma, and mitral valve disease

EXAMINATION OF THE TRACHEA

With certain pulmonary conditions, the trachea may shift toward or away from the affected lung. Some causes that would pull the trachea toward the affected side are pulmonary atelectasis, pulmonary fibrosis, pneumonectomy, and diaphragmatic paralysis.

When the trachea is pushed to the unaffected side, the possible causes are neck tumors, thyroid enlargement, mediastinal mass, massive pleural effusion, tension pneumothorax, and hemothorax.

SPUTUM OBSERVATION

This is presented in **Table 3.2**.

CHEST X-RAYS (FIG. 3.3)

Reasons for Obtaining

There are five significant reasons for obtaining a chest X-ray:

1. Detects changes in the lung caused by pathologic processes.
2. Determines appropriate therapy.
3. Evaluates the effectiveness of treatment.
4. Determines proper tube and catheter placement.
5. Provides a way of trending the progression or decline of lung disease and tumors.

Standard View

The standard views of the chest are the postero-anterior (PA). The PA view is preferred because, since the heart is in the anterior half of the chest, there will be less cardiac magnification.

The left lateral view is also preferred because there is less cardiac magnification and a sharper view of the left lower lobe, which is partially obscured on the PA film.

Correlation between X-rays

Chest X-ray Interpretation

Always start with the name of the patient, date, and type of X-ray (AP, PA, and lateral). Comment on penetration, rotation, projection, and patient position.

A general rule is to start with bones, then fat, liquids/soft tissues, and finally air spaces. Other abnormalities, such as metallic opacities, lines, etc., should also be commented on.

- *Penetration:* Can individual vertebrae be identified?
- *Rotation:* Clavicle heads level? Trachea in line with vertebrae?
- *Projection:* Can the apices and lung bases be seen?
- *Position:* Patient lordotic/kyphotic?

Respiratory

- Trachea—should be central. Deviated toward the affected side in fibrosis, collapse, and pneumothorax, away from the affected side in tension pneumothorax and large effusion

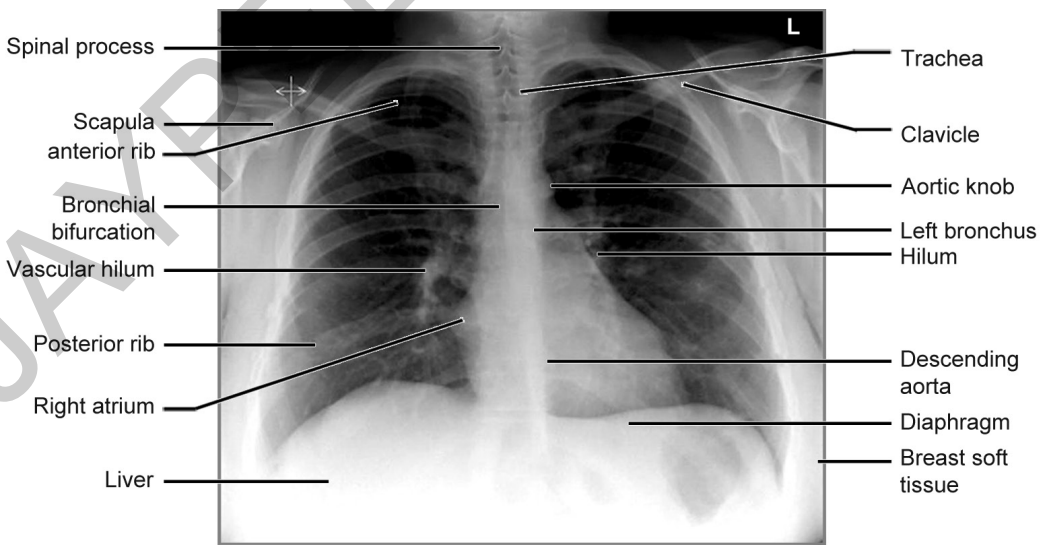

Fig. 3.3: Normal chest X-ray.

- Heart—enlarged (cor pulmonale), tall, and narrow (hyperinflation)
- Hila—left usually higher than right. Pulled down by fibrosis or collapse. Enlarged by infection, carcinoma, and pulmonary hypertension
- Diaphragm—right side usually higher than left. Raised in fibrosis, phrenic nerve palsy, and hepatomegaly. Low in hyperinflation

Abnormalities in Lung Fields

- *Nodular shadowing:* Well-defined
- May be due to neoplasia, granulomas, or pneumoconiosis

Reticular shadowing: "Net-like." Due to a disease of the interstitium.

Usually due to pulmonary interstitial edema. Other causes include fibrosis, sarcoidosis, and interstitial lung diseases.

Alveolar shadows: "Fluffy."

Usually due to pulmonary edema or pneumonia.

Ring shadows and tram lines: Bronchiectasis. Rings are also rarely due to a tumor or an abscess.

Typical Appearances

Asthma:

- X-ray is typical between episodes. Hyperinflated during attacks (tall, narrow heart, greater than six anterior ribs, low, flat diaphragm. May be nodular opacity if lobe collapse [mucus plugging, allergic bronchopulmonary aspergillosis (ABPA)]. Pigeon chest deformity may be seen on the lateral view.
- No abnormalities on CT.

Chronic obstructive pulmonary disease (COPD):

- X-ray often normal, may be signs of hyperinflation—tall, narrow heart, greater than six anterior ribs, low, flat diaphragm, and bullae.
- CT can be used to view emphysema, which appears as a fine shadow.

Bronchiectasis:

- X-ray shows no abnormalities unless there is severe disease.
- Ring shadows/tram lines may be seen. Complications such as collapse/infection may be apparent.
- CT is diagnostic. Shows dilated airways and thickened walls.

Pneumonia:

- X-ray—consolidation, often lobular or segmental.
- It may be a parapneumonic effusion. Maybe hilar lymphadenopathy.
- CT is not indicated unless something else is suspected (i.e., carcinoma).

Tuberculosis:

- X-ray—consolidation/collapse. Cavitation (granuloma).
- Pleural effusion/empyema, "miliary" shadowing; diffuse, speckled.
- CT is not indicated unless something else is suspected (i.e., carcinoma).

Bronchial carcinoma:

- X-ray signs differ depending on the site and size of the tumor.
- Common findings—unilateral hilar enlargement (central tumor).
- Peripheral opacity is usually irregular and can be very large.
- Collapse (distal to tumor); appears as a hazy shadowing. Pleural effusion (due to invasion of the pleural space or infection)
- Broadening of the mediastinum (paratracheal lymphadenopathy)
- Enlarged cardiac shadow (malignant pericardial effusion)
- Elevated hemidiaphragm (phrenic nerve palsy) and rib destruction (bone metastasis)
- Tumors that are too small to be picked up on X-ray may be detected on CT.

Mediastinal carcinoma:

- May present on X-ray as a broadening of the mediastinum, but CT and magnetic resonance imaging (MRI) are the investigations of choice.

Sarcoidosis:

- In early stages, X-rays show bilateral hilar lymphadenopathy (BHL), enlarged paratracheal nodes, which progress to include diffuse pulmonary opacities. BHL subsides in later stages. Frank fibrosis is seen in stage 4.
- Contrast-enhanced CT may show lymph nodes with "rim enhancement" and central necrosis, whatever that looks like.

Cryptogenic fibrosing alveolitis (CFA):

- *X-ray shows typical fibrotic changes:* Small lungs, high diaphragm, basal and peripheral opacities. Honeycombing in late-stage disease.
- HRCT shows characteristic changes (see Davidson's 552 old edition). Particularly useful in early disease, where an X-ray may be normal.

PE:

- X-ray may be normal. Otherwise, opacities that may be any size or shape can cavitate. Elevated hemidiaphragm. Bilateral horizontal opacities (usually in lower zones), pleural effusion. Rarely: Wedge-shaped opacity.
- Computed tomographic pulmonary angiography (CTPA) investigation of choice. Pick up emboli like nobody's business.

Pneumothorax:

- X-ray shows a reduced lung field with a sharply defined edge. Will show the extent of any mediastinal displacement (i.e., tension pneumothorax).
- CT indicates if doubt exists between a large emphysematous bulla and pneumothorax.

Cardiovascular

Best reviewed by PA in full inspiration.

Cardiothoracic ratio: Maximum width of cardiac silhouette/widest aspect of lung fields. It can only be accurately measured on a PA. >0.5 suggests cardiac hypertrophy/dilatation. *Important*: Cardiomyopathies, pericardial effusion, heart failure, and valvular disease.

Specific dilatations:

- Left atrium produces a straight left heart border, a double cardiac shadow to the right of the sternum, and a widened carina.
- The right atrium protrudes out into the right lung space.
- Left ventricle causes prominence of the left lower heart border and widening of the cardiac silhouette.
- Right ventricle displaces the apex upward and straightens the left heart border.

Pulmonary edema:

- "Ground glass" of alveolar edema
- Prominence of upper lobe vessels
- Enlarged hilar vessels
- Enlarged cardiac silhouette
- Kerley B lines (horizontal lines in lower zones)

Aortic dissection: Broadened upper mediastinum, distorted aortic knuckle (absent in 40% cases).

FINDINGS AND PHYSICAL EXAMINATION

There should be a correlation between what is seen on an X-ray and the physical examination. The following is a list of physical findings with related X-ray findings for certain disease states.

Atelectasis

Physical findings: Elevated diaphragm on the affected side on palpation of the lower chest. Decreased or absent breath sounds on the affected side by auscultation. Shift of the trachea to the affected side only if a large area of the lung is affected **(Fig. 3.4)**.

Consolidation

Physical findings: Dullness on percussion over affected areas. Auscultation reveals crackles over the affected area. Whispered voice sounds are usually louder than usual **(Fig. 3.5)**.

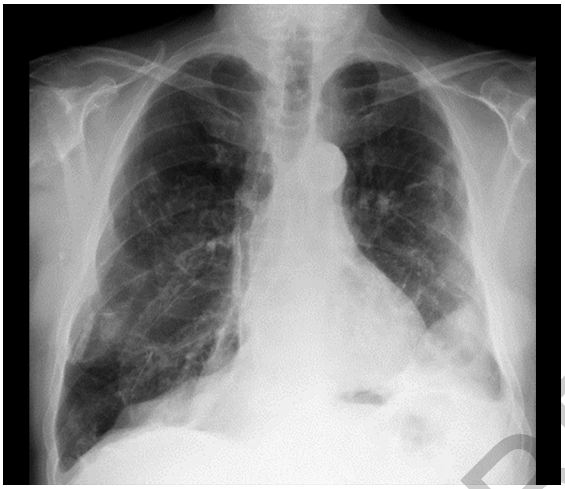

Fig. 3.4: X-ray findings—shift of the fissure lines and hilar structures toward the affected area and overall loss of volume with elevation of the hemidiaphragm.

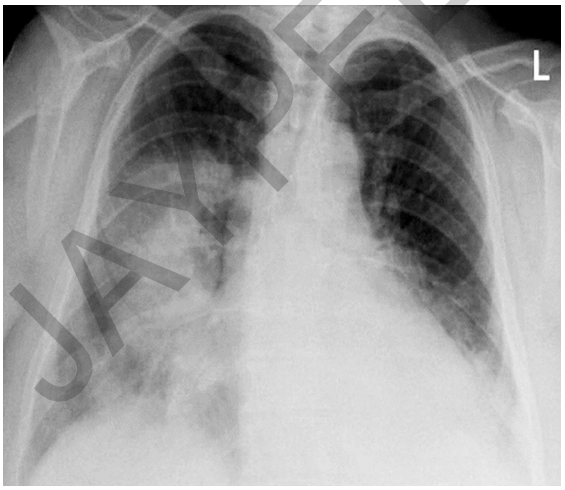

Fig. 3.5: X-ray findings—minimal loss of volume with lobar distribution and homogeneous density (whiter area) late in the consolidation process.

Congestive Heart Failure

Physical findings: Increased heart rate with either a regular or irregular rhythm. A third heard sound (S3) is a consistent finding. The peripheral pulse may be strong, alternating with a weak pulse every other beat. Pedal edema is usually present during the day but is somewhat relieved after a night of sleep. The patient usually complains of coughing, attacks of severe shortness of breath, fatigue, and weakness **(Fig. 3.6)**.

Hyperinflation (Chronic Obstructive Pulmonary Disease)

Physical findings: Barrel-chested with decreased breath sounds, wheezing, limited motion of the diaphragm, increased respiratory rate, and the use of accessory muscles to breathe **(Fig. 3.7)**.

Interstitial Fibrosis

Physical findings: May have a dry, nonproductive cough, dyspnea on exertion, and a history of exposure to inhaled agents **(Fig. 3.8)**.

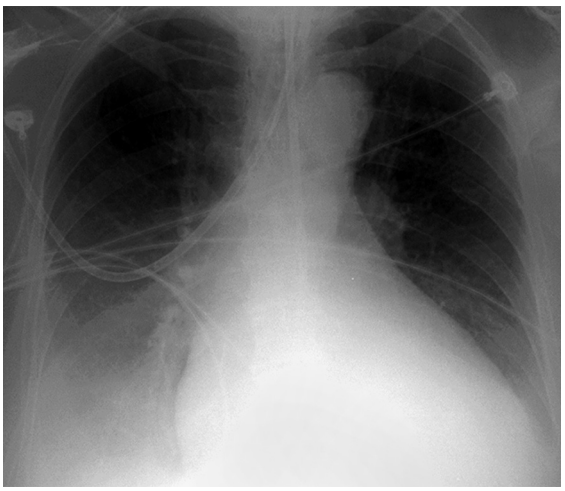

Fig. 3.6: X-ray findings—usually shows prominent pulmonary blood vessels. Development of Kerley B lines, which are seen in the right base. These Kerley B lines are horizontal and start at the periphery; they are usually 1 mm thick and 1 to 2 cm in length.

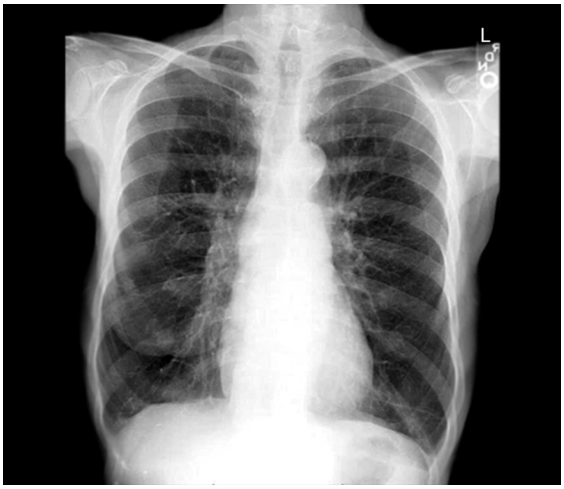

Fig. 3.7: X-ray findings—increased anteroposterior (AP) chest dimensions and anterior air space. Depressed diaphragms, marked hyperinflation with large lung volumes, and a small, narrow heart with enlarged intercostal spaces.

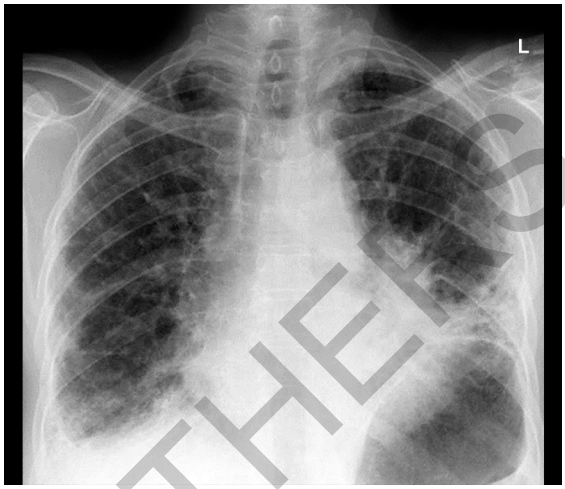

Fig. 3.8: X-ray findings—peripheral markings are enlarged and white in color. The air-filled airways are seen as clear with dark straight shadows. The peripheral white markings are said to have the appearance of ground glass, representing affected alveolar spaces.

Pleural Effusion

Physical findings: Decreased breath sounds over the affected side, pain on inspiration, coughing, and shortness of breath **(Fig. 3.9)**.

RESPIRATORY DISEASES

The following is the listing of possible causes, clinical findings, and treatment of common respiratory diseases.

Chronic Bronchitis

Structural Changes

Chronic inflammation and swelling of the peripheral airways were associated with excessive mucus production and accumulation in the bronchi.

In the early stages of chronic bronchitis, a cough usually occurs in the morning. As the disease progresses, coughing persists throughout the day. This chronic cough is commonly referred to as a "smoker's cough."

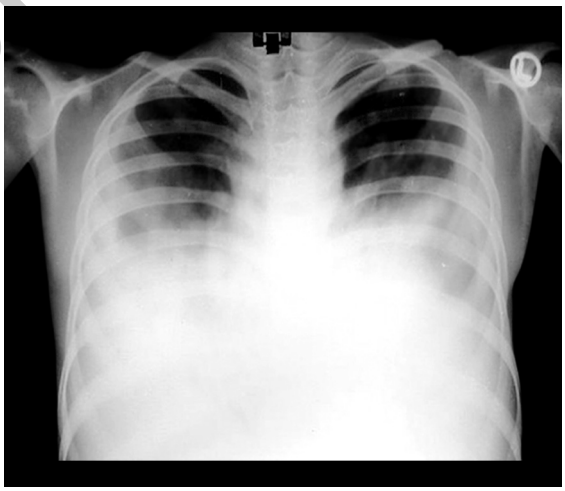

Fig. 3.9: X-ray findings—large volumes of complete "white out" on the affected side completely obscuring the hemi-diaphragm. With small-volume effusion, only partial "white-out" on the affected side is seen, with only partial obstruction of the hemi-diaphragm.

Also, in the early stages of chronic bronchitis, only the larger airways are affected, but eventually all airways are involved. Over time, the patient

experiences abnormal ventilation-perfusion, insufficient oxygenation of blood (hypoxemia), labored breathing (hypoventilation), and right-sided heart failure (cor pulmonale). Compared with acute bronchitis, which may respond quickly to medications, such as antibiotics, chronic bronchitis can be challenging to treat because many patients with chronic bronchitis are susceptible to recurring bacterial infections.

Excessive mucous production in the lungs provides a suitable environment for infection, which also causes inflammation and swelling of the bronchial tubes and a reduction in airflow in and out of the lungs. In the later stages of chronic bronchitis, the patient cannot clear this thick, tenacious mucus, which then causes damage to the hair-like structures (cilia) that help sweep away fluids and/or particles in the lungs. This, in turn, impairs the lung's defense against air-borne irritants.

Cigarette smoking is the most common cause of chronic bronchitis. People who have been exposed for a long time to irritants, such as chemical fumes, dust, and other noxious substances, can also get chronic bronchitis. As chronic bronchitis often coincides with emphysema, it is frequently difficult for a physician to distinguish between the two. Chronic bronchitis can also have an asthmatic component.

Possible Causes

Exact causes are not known; cigarette smoking, atmospheric pollutants, and repeated infection of the respiratory tract have been linked to the disorder.

Clinical Findings

Pulmonary function tests: Decreases in forced expiratory flow (FEF) 25–75, forced expiratory volume in 1 second (FEV1), maximum voluntary ventilation (MVV), peak expiratory flow rate (PEFR), vital capacity (VC), inspiratory reserve volume (IRV); increases in tidal volume (Vt), residual volume (RV), functional residual capacity (FRC), vital capacity (VC), and forced vital capacity (FVC) **(Fig. 3.10)**.

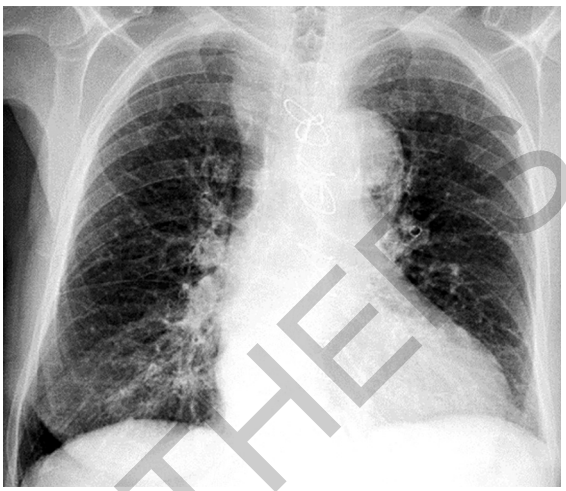

Fig. 3.10: Chest X-ray—translucent, depressed, or flattened diaphragm, spike-like projections on bronchogram, enlarged heart, pulmonary vascular engorgement, and increased anteroposterior chest diameter (barrel chest).

Respiratory Findings

Use of accessory muscles, diminished breath sounds with wheezing and/or rhonchi, chronic cough with excessive sputum production for three months per year for two or more successive years.

Arterial Blood Gases

Early: Decreased PaO_2, normal or decreased $PaCO_2$, and regular or reduced pH.

Advanced stage: Decreased PaO_2, increased $PaCO_2$, increased HCO_3, normal or decreased pH.

Treatment

Avoidance of smoking, inhaling irritants, and infections is recommended for people with contagious respiratory tract infections.

Mobilize bronchial secretions by nebulizer or aerosol therapy, increased fluid intake, chest

physical therapy, postural drainage, deep breathing aids (incentive spirometer), and suctioning. Sympathomimetics (Bronkosol, Alupent, etc.), methylxanthines (aminophylline, etc.), expectorants (Robitussin, etc.), antibiotics (ampicillin and tetracycline). Proper education and psychological and sociological support.

There is no cure for chronic bronchitis. Treatment is aimed at relieving symptoms and preventing complications. Lying down at night can worsen the condition, so some people with advanced chronic bronchitis must sleep sitting up. In late, severe stages, people who often have emphysema as well are called "blue bloaters" because lack of oxygen causes the skin to have a blue cast (cyanosis) and because the body is swollen from fluid accumulation caused by congestive heart failure.

Emphysema

Permanent enlargement and deterioration of air spaces distal to the terminal bronchioles. Destruction of pulmonary capillaries. Weakening of the distal airways, primarily the respiratory bronchioles.

Emphysema is a long-term, progressive disease of the lungs that primarily causes shortness of breath. In people with emphysema, the lung tissues necessary to support the physical shape and function of the lung are damaged. It is included in a group of diseases called chronic obstructive pulmonary disease or COPD (pulmonary refers to the lungs). Emphysema is called an obstructive lung disease because of the destruction of lung tissue around the smaller airways.

The airways, called bronchioles, make these airways unable to hold their shape properly when you exhale. This makes them inefficient at transferring oxygen into the blood and at removing carbon dioxide from the blood.

Emphysema changes the anatomy of the lung in several essential ways.

Usually, the lungs are very spongy and elastic. When a breath is taken, the chest wall expands, expanding the sponge. Just as a squeezed sponge will draw water into it when released, suction draws air into the lungs when the chest wall expands. Air is brought through the trachea (windpipe) and bronchi (the primary air tubes going to the right and left lungs). These tubes divide into smaller and smaller tubes, finally ending in alveoli. Alveoli, the tiniest structures in the lung, are tiny air sacs that are arranged like a bunch of grapes. The alveoli are at the ends of the smallest tubes called bronchioles. The alveoli and the bronchioles are critical structures for the lungs to function properly. It is these structures that are damaged by emphysema.

A sponge works to pick up water because all the tiny holes expand at once after being squeezed. If the holes were larger, the sponge would not pick up as much water. This is because a larger hole cannot expand enough by itself to equal the action of multiple smaller ones. Thinking of the lungs as a sponge in this way, it becomes easier to see how emphysema acts to cause impaired lung function. Lungs require an elastic quality so that they can expand and contract well. Also, as with the holes of the sponge, the lungs need many alveoli (hundreds of millions, in fact) to draw enough air into them. The fewer and the bigger the alveoli, the less effectively they perform.

Types

There are three types of emphysema:

1. *Centrilobular*: Changes mostly in the respiratory bronchioles.
2. *Panlobular*: Changes at the alveolar level.
3. *Bullous*: Changes at the alveolar and respiratory bronchioles.

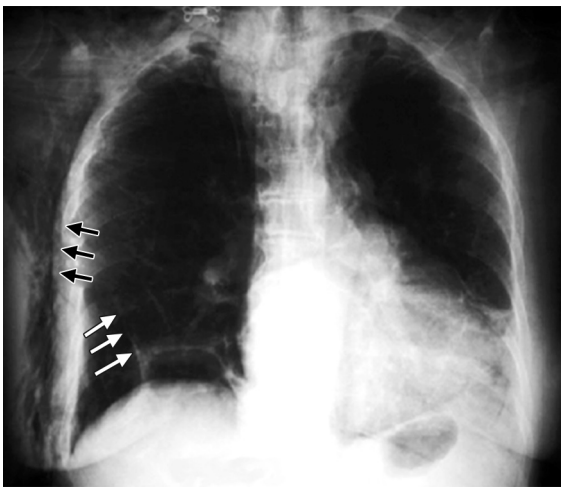

Fig. 3.11: Chest X-ray—increased anteroposterior chest diameter (barrel chest); translucent, depressed, or flattened diaphragm; elongated cardiac silhouette, and small heart.

Possible Causes

Cigarette smoking, alpha-antitrypsin deficiency, infections of the respiratory tract during childhood, and inhaled irritants (sulfur dioxide, nitrogen oxides, and ozone).

Clinical Findings

Pulmonary function tests: Increased Vt, RV, FRC, VC, decreased IRV, ERV, FVC, and FEF25-75, FEV1, MVV, PEFR **(Fig. 3.11)**.

Respiratory Findings

Increases in respiratory infections, respiratory rate, heart rate, cardiac output, and blood pressure. The patient will show an increase in the use of accessory muscles with pursed-lip breathing. Cyanosis and digital clubbing may also be seen.

Arterial Blood Gases

Early stages: Decreased PaO_2, normal or decreased $PaCO_2$, normal or decreased HCO_3, and standard or increased pH.

Advanced stages: Decreased PaO_2, increased $PaCO_2$, increased HCO_3, normal or decreased pH.

Treatment

Avoid smoking, people with contagious respiratory tract infections, and inhaling irritants. Proper nutrition; meals (possible high protein) should be given in small amounts but more often, 6–8 a day. Mobilization of bronchial secretions by aerosol or nebulizer therapy, increase in fluids, chest physical therapy, deep breathing aids (incentive spirometer), and suctioning. Supplemental oxygen at low flow. Possible medications: Sympathomimetic agents (Bronkosol, Alupent, etc.). Methylxanthines (aminophylline), expectorants (Nortussin, Robitussin, etc.), antibiotics, digitalis (Lanoxin, etc.) for patients with ventricular heart failure with emphysema. Proper education of the patient and family is crucial.

Complications of Emphysema

Recurrent chest infections, including pneumonia, the flu (influenza), cold, and the common cold.

Pulmonary hypertension: Unusually high blood pressure in the arteries of the lungs.

Cor pulmonale: Enlargement and strain on the right side of the heart. This can lead to heart failure.

Asthma

Smooth muscle constriction of bronchial airways. Excessive production of thick, tenacious tracheobronchial secretions. Hyperinflation of alveoli. Thickening of subepithelial membranes.

Four types of cells bring about the inflammatory reaction in bronchial asthma—namely, the dendritic cells and macrophages, T-helper lymphocytes, mast cells, and eosinophils. Dendritic cells and macrophages present antigens to T-helper cells and induce the switching of B

lymphocytes to produce immunoglobulin E (IgE). These cells are influenced by corticosteroids (e.g., beclomethasone, prednisolone), though beta receptor antagonists do not affect them.

T-helper lymphocyte is the key in the pathogenesis of bronchial asthma. They induce B cells to synthesize and secrete IgE through the production of interleukin 4 (IL-4) and induce eosinophilic inflammation via interleukin 5 (IL-5). T-helper cells are also influenced by corticosteroids but not by the beta receptor agonists.

Mast cells contribute to the inflammatory reaction by the production and release of histamine, tryptase, prostaglandin D_2 (PGD_2), and leukotriene C_4 (LTC_4). These cells are involved in the early phases of asthma, known as the early reaction. Unlike other types of cells, mast cell membranes are stabilized by beta receptor agonists (such as salbutamol and terbutaline) and chromones (such as sodium cromoglycate).

Eosinophils are involved in the late-phase reaction of bronchial asthma. They are attracted to the bronchial walls by interleukins 3 and 5 (IL-3, IL-5) and the granulocyte monocyte colony-stimulating factor (GM-CSF) secreted by the T-helper cells. Corticosteroids are effective in decreasing the entry of eosinophils as well as the number of eosinophils in circulation. In addition, corticosteroids prevent activation of eosinophils that have entered the bronchial walls.

Possible Causes

Extrinsic asthma (allergic reaction to pollen, house dust, feathers, etc.). Hypersensitivity to common environmental allergens. Intrinsic asthma (nonallergic or nonatopic), infection, cold air, vapor, drugs (aspirin), emotional stress, and exercise.

Clinical Findings

Pulmonary function tests: Decreases in FEF25-75, FEV1, MVV, PEFR, VC, IRV, ERV; increases in RV, FRC, RV/TLC ratio **(Fig. 3.12)**.

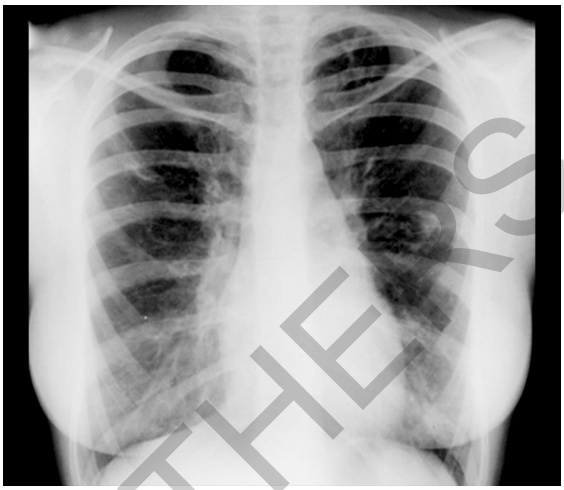

Fig. 3.12: Chest X-ray—increased anteroposterior chest diameter, translucent, depressed, or flattened diaphragm.

Respiratory Findings

Increased respiratory rate, heart rate, and blood pressure, use of accessory muscles, pursed-lip breathing, cough with expectoration, decreased breath sounds with wheezing and/or rhonchi often audible without a stethoscope, and a prolonged expiratory time.

Arterial Blood Gases

Acute: Decreased PaO_2, decreased $PaCO_2$, decreased HCO_3, increased pH. *Status asthmaticus*: Decreased PaO_2, increased $PaCO_2$, increased HCO_3, decreased.

Treatment

Medications: Sympathomimetic agents, methylxanthines, corticosteroids. Supplemental oxygen and possible mechanical ventilation in patients with status asthmaticus. Pneumothorax frequently develops in status asthmaticus, necessitating the need for chest tubes. Mobilization of bronchial secretions—suctioning, aerosol therapy, increased fluid intake—systemic, and possible chest physical therapy. Monitoring arterial blood gases, proper

education of the patient, and psychological and sociological support are essential.

Pneumonia

Inflammation of the alveoli. Alveolar consolidation.

Pathophysiology

There are different categories of pneumonia. Two of these types are hospital-acquired and community-acquired. Common types of community-acquired pneumonia are Pneumococcal pneumonia and Mycoplasma pneumonia. In some people, particularly the elderly and those who are debilitated, pneumonia may follow influenza. Hospital-acquired pneumonia tends to be more serious because defense mechanisms against infection are often impaired. Some of the specific pneumonia-related disorders include aspiration pneumonia, pneumonia in an immunocompromised host, and viral pneumonia.

Possible Causes

Gram-positive, gram-negative organisms, viral, rickettsial infection, psittacosis, varicella, rubella, and aspiration.

Clinical Findings

Pulmonary function tests: Decreases in VC, RV, FRC, TLC, and Vt **(Fig. 3.13)**.

Respiratory Findings

Increases in respiratory rate, heart rate, blood pressure, cough with expectoration, chest pain with decreased chest expansion, bronchial breath sounds with rales/rhonchi/wheezing, and possible pleural friction rub.

Arterial Blood Gases

Early stages: Decreased PaO_2, normal to decreased $PaCO_2$, normal to decreased HCO_3, and increased pH.

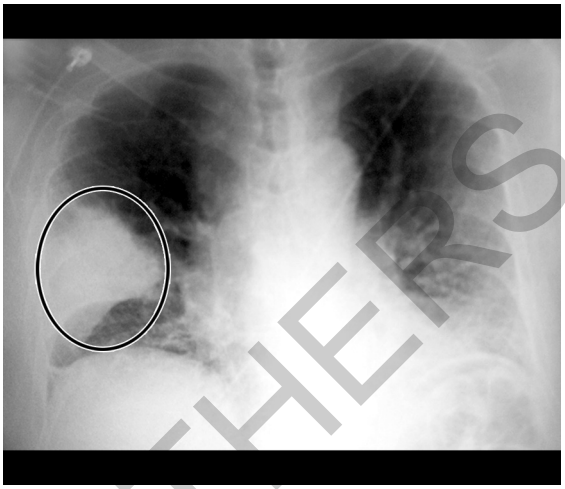

Fig. 3.13: Chest X-ray—increased opacity (whiter in appearance), increased lung density.

Advanced stages: Decreased PaO_2, increased $PaCO_2$, increased HCO_3, and decreased pH.

Treatment

Medications: Antibiotic agents, sympathomimetic, and non-sedative analgesic agents. Oxygen therapy (keeping the PaO_2 60–80 mm Hg), possible mechanical ventilation for patients with chronic lung problems and pneumonia, increases in fluid intake, deep breathing aids [intermittent positive pressure breathing (IPPB), incentive spirometer], chest physical therapy, nebulizer therapy, possible thoracentesis, bronchoscope, control of fever, and excess cough. Keep a close watch for complications.

Pulmonary Edema

Interstitial edema, including fluid engorgement of the perivascular and peribronchial spaces and the alveolar wall interstitium, increased surface tension, alveolar shrinkage, and frothy secretions throughout the tracheobronchial tree.

Pathophysiology

Pulmonary vessels create an imbalance in the Starling forces, leading to an increase in the fluid

filtration into the interstitial spaces of the lungs that exceed the lymphatic capacity to drain the fluids away, increasing volumes of fluid leak into the alveolar space.

The lymphatic system drains excess interstitial fluid volume. Additional fluid in the pleural drains into the titer lymph nodes. In this, the pathway becomes overwhelmed; however, fluid moves from the interstitial space in the alveolar walls. If the alveolar epithelium is damaged, the fluid accumulates in the alveoli. Alveolar edema is a series of late signs in the progression of fluid imbalance. Hypoxemia develops when the alveolar membrane is thickened by fluid that impairs the gas exchange of oxygen and CO_2. As fluid fills the interstitial and alveolar space, lung compliance decreases, and oxygen diffusion decreases.

Possible Causes

Cardiogenic causes: Increases in hydrostatic pressure, arrhythmias, excessive fluid administration, left ventricular failure, mitral valve disease, myocardial infarction, pulmonary embolus, renal failure, and systemic hypertension.

Noncardiogenic causes: Pulmonary edema, alveolar hypoxia, ARDS, inhalation of toxic gas (chlorine, sulfur dioxide, and ammonia), therapeutic radiation of the lungs, lymphatic insufficiency, overtransfusion or rapid transfusion, uremia, hypoproteinemia, acute nephritis, allergic reaction to drugs, aspiration, central nervous system stimulation, encephalitis, and head trauma.

Clinical Findings

Pulmonary function tests: Decreases in VC, RV, FRC, TLC, and Vt **(Fig. 3.14)**.

Respiratory Findings

Increased respiratory rate, nocturnal dyspnea, cough with expectoration, cyanosis, breath sounds, rales/rhonchi/wheezing, and an increase in pulmonary wedge pressure—cardiogenic only.

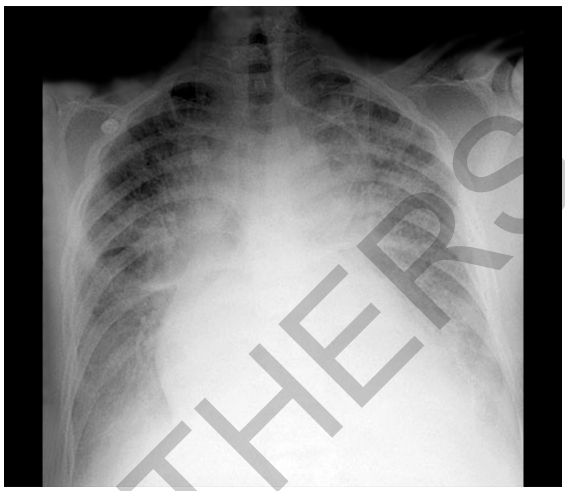

Fig. 3.14: Chest X-ray—increased opacity, enlarged heart, prominent pulmonary vessels, and Kerley B lines.

Arterial Blood Gases

Early: Decreased PaO_2, normal or decreased $PaCO_2$, normal or decreased HCO_3, and normal to reduced pH.

Advanced stage: Decreased PaO_2, increased $PaCO_2$, increased HCO_3, and decreased pH.

Pulmonary wedge pressure for cardiogenic pulmonary edema >12 mm Hg.

Treatment

Medications—morphine sulfate, diuretic agents, digitalis, sympathomimetic agents, albumin, possible inhaled ethanol; positioning patients in Fowler's position; hyperinflation and oxygen (keeping PaO_2 60–80 mm Hg), possible intubation with volume-cycled ventilator with positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP). Hemoglobin <10 g should be corrected by blood transfusion and fluid management.

Pulmonary Embolism

Blockage of the pulmonary vascular system. Pulmonary infarction. Pulmonary tissue necrosis.

Thrombotic pulmonary embolism is not an isolated disease of the chest but a complication of venous thrombosis. Deep venous thrombosis (DVT) and pulmonary embolism are therefore parts of the same process, venous thromboembolism. Evidence of leg DVT is found in about 70% of patients who have sustained a pulmonary embolism; in most of the remainder, it is assumed that the whole thrombus has already become detached and embolized. Conversely, pulmonary embolism occurs in up to 50% of patients with proximal DVT of the legs (involving the popliteal and/or more proximal veins). It is less likely when the thrombus is confined to the calf veins. Rarely, the source of emboli is the iliac veins, renal veins, right heart, or upper extremity veins; the clinical circumstances usually point to these unusual sites.

Pathophysiology

The effects of an embolus depend on the extent to which it obstructs the pulmonary circulation, the duration over which that obstruction accumulates, and the pre-existing state of the patient, which has been defined only imprecisely. Some humoral mediators (for example, serotonin or thromboxane from activated platelets) can probably produce vasospasm in non-embolized segments of the lung. As a result, a degree of pulmonary hypertension may develop disproportionate to the amount of vasculature that is mechanically occluded. In general, a patient who has pre-existing cardiopulmonary disease or who is old, frail, or debilitated will be more sensitive to the effects of pulmonary embolism than a patient who was well until the embolic event occurred. Most emboli are multiple. As both the extent and chronicity of obstruction vary so widely, pulmonary embolism can produce widely differing clinical pictures, disregarding chronic thromboembolic pulmonary hypertension. The first and most common presentation is dyspnea with or without pleuritic pain and hemoptysis (acute minor pulmonary embolism). The second presentation is hemodynamic instability, which is associated with acute massive pulmonary embolism. The third and least common presentation mimics heart failure or indolent pneumonia, especially in the elderly (subacute massive pulmonary embolism).

Possible Causes

Prolonged bed rest and/or immobilization; prolonged sitting (car, plane, bus, etc.); congestive heart failure; varicose veins; long bone fractures—trauma; injury of the soft tissues, vessels; extensive hip, abdominal, or chest surgery; oral contraceptives; obesity; pregnancy; and burns.

Clinical Findings

Physical findings: Chest pain, increased respiratory rate, cough with hemoptysis, syncope, lightheadedness, confusion, cyanosis, breath sounds—rales/rhonchi/wheezing/pleural friction rub, abnormal perfusion lung scan, pulmonary hypertension, systemic hypotension. ECG—sinus tachycardia or atrial arrhythmia or possible incomplete right bundle-branch block.

Arterial Blood Gases

Decreased PaO_2, normal to decreased $PaCO_2$, normal to decreased HCO_3, and normal to increased pH.

Treatment

Heparin dicumarol, supplemental oxygen, possible embolectomy, and treat any cardiac problems that might arise.

Anticoagulants or blood thinners decrease your blood's ability to clot. They are used to stop blood clots from getting larger and prevent clots

from forming. Blood thinners do not break up blood clots that have already formed (the body dissolves most clots with time).

Blood thinners are available as either a pill, an injection, or through a needle or tube inserted into a vein (called intravenous, or IV, injection). Warfarin is given as a pill. Heparin is given as an injection or through an IV tube.

Adult Respiratory Distress Syndrome

Interstitial and intra-alveolar edema and hemorrhage. Alveolar consolidation. Intra-alveolar hyaline membrane. Pulmonary surfactant deficiency or abnormality. Atelectasis.

Pathophysiology

- Exudative phase with alveolar edema, capillary congestion, and hyaline membrane formation.
- Proliferative phase with proliferation of type II alveolar cells and cellular infiltration.
- Fibrotic phase with fibrosis of membranes, septae, and ducts.

Two defining characteristics of ARDS are edema/infiltration and fibrosis/low compliance. Edema occurs because the membranes become permeable to protein. Oncotic gradient is lost, and protein leaks into the interstitium and alveoli. The filtration coefficient (K_f) goes way up. Capillaries also vasoconstrict due to hypoperfusion. This protein permeability and increased pressure serve to fill the interstitium and alveoli with protein and water. This edema distributes with gravity because of the weight of the lungs pressing down and closing alveoli. So, if they lie on their back, posterior regions close off, and vice versa. This is in contrast to congestive heart failure (CHF) edema, which is due solely to high capillary pressure and is, therefore, a protein-poor edema. There is also fibrosis and loss of surfactant, both of which give the lungs reduced compliance **(Fig. 3.15)**.

The following criteria characterize adult respiratory distress syndrome:

- Lung injury of acute onset, within 1 week of an apparent clinical insult, and with progression of respiratory symptoms
- Bilateral opacities on chest imaging not explained by other pulmonary pathology (e.g., pleural effusion, pneumothorax, or nodules)
- Respiratory failure not explained by heart failure or volume overload.

Decreased arterial PaO_2/FiO_2 ratio:

- *Mild ARDS:* Ratio is 201–300 mm Hg (≤39.9 kPa)
- *Moderate ARDS:* 101–200 mm Hg (≤26.6 kPa)
- *Severe ARDS:* ≤100 mm Hg (≤13.3 kPa)

The above characteristics are the "Berlin criteria" of 2012 by the European Society of Intensive Care Medicine, endorsed by the American Thoracic Society and the Society of Critical Care Medicine. They are a modification of the previously used criteria:

Acute onset: Bilateral infiltrates on chest radiograph, sparing the costophrenic angles.

Pulmonary artery wedge pressure <18 mm Hg (obtained by pulmonary artery catheterization), if this information is available; if unavailable, then lack of clinical evidence of left atrial hypertension.

- *If PaO_2:* FiO_2 <300 mm Hg (40 kPa), acute lung injury (ALI) is considered to be present.
- *If PaO_2:* FiO_2 <200 mm Hg (26.7 kPa), ARDS is considered to be present.

Possible Causes

Aspiration; central nervous system disease; cardiopulmonary bypass; congestive heart failure; disseminated intravascular coagulation (DIC); drug overdose; fat or air emboli; fluid overload; infections; inhalation of chlorine, nitrogen dioxide, smoke, or ozone; immunologic reaction.

Clinical Findings

Pulmonary function tests: Decreases in VC, RV, FRC, TLC, and Vt **(Fig. 3.16)**.

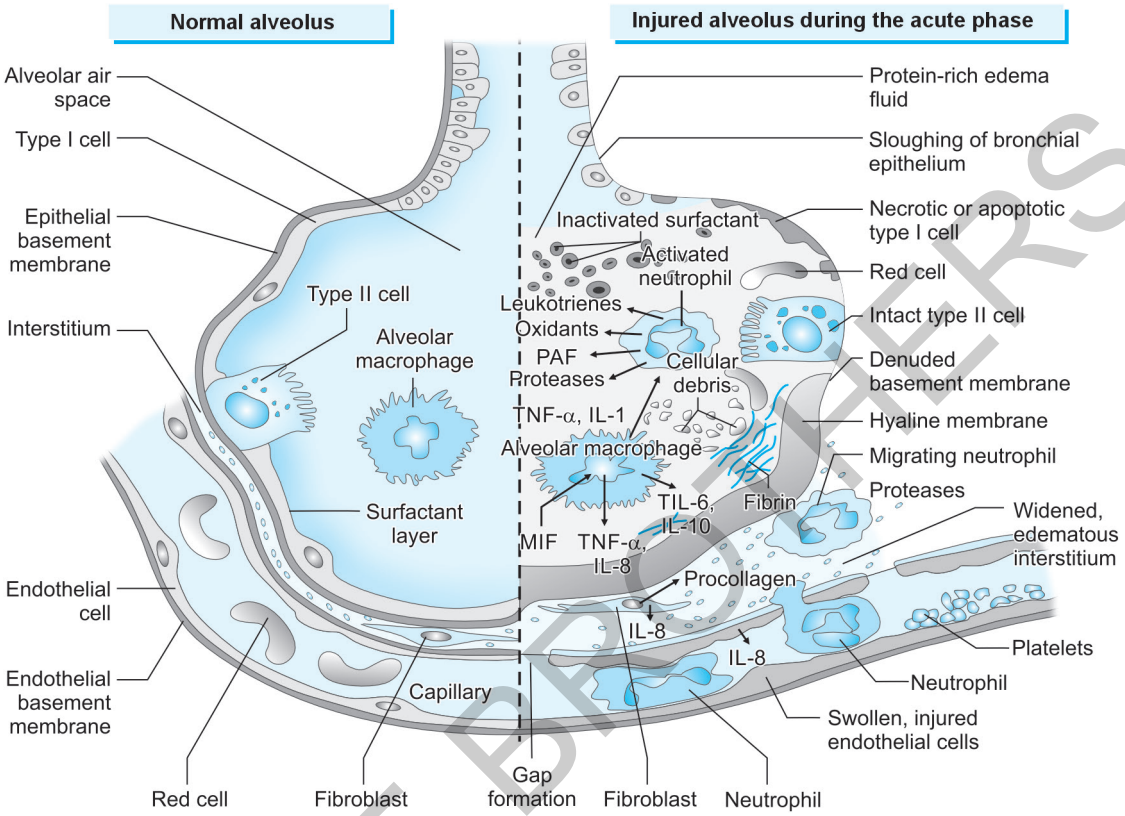

Fig. 3.15: Normal alveolus vs injured alveolus.

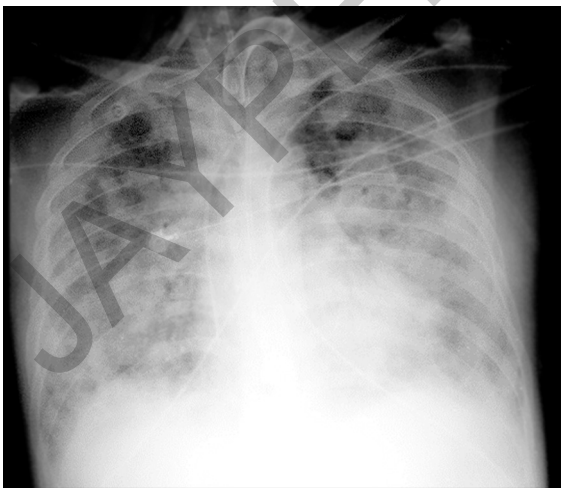

Fig. 3.16: Chest X-ray—increased opacity, diffused alveolar infiltrates (honeycomb effect).

Respiratory Findings

Increased respiratory rate and blood pressure, intercostal retractions, cough, nausea, vomiting, fever, decreased compliance, breath sounds, rales/rhonchi, and mental disorientation.

Arterial Blood Gases

Decreased PaO_2, decreased $PaCO_2$, and increased pH. These changes occur in the early stages.

Treatment

Medications: Diuretic agents, corticosteroids, and antibiotics, hyperinflation, and oxygenation. Possible intubation with ventilation and PEEP therapy, maintaining PaO_2 >60 mm Hg, possible fluid therapy, watching input and output.

Flail Chest

Structural Changes

Double fracture of multiple adjacent ribs. Rib instability. Lung restriction. Atelectasis. Lung collapse.

A flail chest occurs when a segment of the thoracic cage is separated from the rest of the chest wall. This is usually defined as at least two fractures per rib (producing a free segment), in at least two ribs. A segment of the chest wall that is flail is unable to contribute to lung expansion. Large flail segments will involve a much greater proportion of the chest wall and may extend bilaterally or involve the sternum. In these cases, the disruption of standard pulmonary mechanics may be significant enough to require mechanical ventilation.

The primary significance of a flail chest, however, is that it indicates the presence of an underlying pulmonary contusion. In most cases, it is the severity and extent of the lung injury that determines the clinical course and requirement for mechanical ventilation. Thus, the management of flail chest consists of standard management of the rib fractures and the pulmonary contusions underneath.

Possible Causes

Direct compression by a heavy object (automobile or industrial accident).

Clinical Findings

Pulmonary function tests: Decreases in VC, RV, FRC, TLC, and Vt **(Fig. 3.17)**.

Respiratory Findings

Increased respiratory rate, heart rate, blood pressure, and paradoxical chest movement (uneven movement).

Arterial Blood Gases

Early stages: Decreased PaO_2, normal or decreased $PaCO_2$, regular or reduced HCO_3, normal or increased pH.

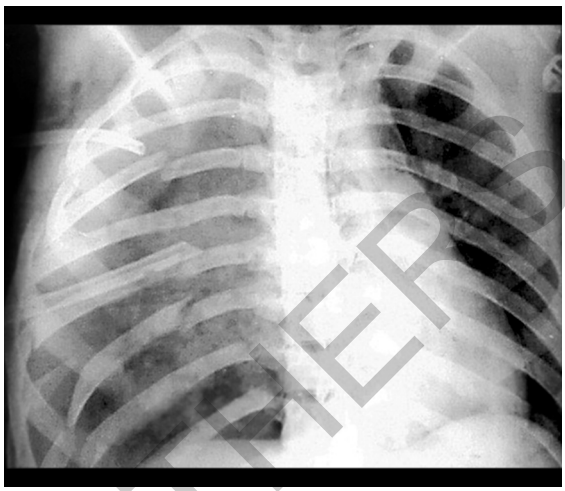

Fig. 3.17: Chest X-ray—rib fractures involving three or more adjacent ribs, or multiple fractures of two or more ribs will increase opacity.

Advanced stages: Decreased PaO_2, increased $PaCO_2$, increased HCO_3, and decreased pH.

Treatment

Deep-breathing aids (incentive spirometer), medication for pain, bronchial hygiene, stabilization of fractures, and possible mechanical ventilation with PEEP. Look for possible pneumothorax.

Pneumothorax

Structural Changes

Lung collapse. Atelectasis. Compression of the great veins and decreased venous return.

In normal subjects, the pressure in the pleural space is negative concerning the alveolar pressure during the entire respiratory cycle. The pressure gradient between the alveoli and the pleural space is the transpulmonary pressure and is the result of the inherent elastic recoil of the lung. During spontaneous breathing, the pleural pressure is also negative concerning the atmospheric pressure. The FRC, or resting end-expiratory volume of the lung, is the volume at

which the inherent outward pull of the chest wall is equal to, but opposite in direction to, the inward pull (recoil) of the lung.

A primary pneumothorax occurs without an apparent cause and in the absence of significant lung disease, while a secondary pneumothorax occurs in the presence of existing lung pathology. In a minority of cases, the amount of air in the chest increases markedly when a one-way valve is formed by an area of damaged tissue, leading to a tension pneumothorax. This condition is a medical emergency that can cause steadily worsening oxygen shortage and low blood pressure. Unless reversed by effective treatment, these sequelae can progress and cause death.

A traumatic pneumothorax may result from either blunt trauma or penetrating injury to the chest wall. The most common mechanism is due to the penetration of sharp bony points at a new rib fracture, which can cause damage lung tissue. Traumatic pneumothorax may also be observed in those exposed to blasts, even though there is no apparent injury to the chest.

Possible Causes

Traumatic pneumothorax (penetrating wounds, also called sucking chest wound). Spontaneous pneumothorax (suddenly without any apparent underlying cause). Iatrogenic pneumothorax (occurs during specific diagnostic or therapeutic procedures).

Clinical Findings

Pulmonary function tests: Decreases in VC, RV, FRC, TLC, and Vt **(Fig. 3.18)**.

Respiratory Findings

Increased difficulty in ventilating if on a mechanical ventilator, vital signs will deteriorate, breath sounds will be absent on the affected side, trachea and mediastinum may shift toward the unaffected side, possible pleuritic pain, and a dry hacking cough.

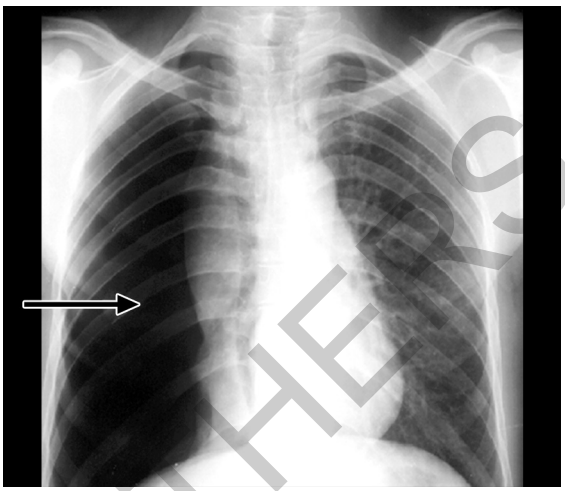

Fig. 3.18: X-ray findings—show a pleural line that runs down the chest wall. The trachea is shifted toward the unaffected side.

Treatment

Decompression of the thorax by chest tube insertion or needle aspiration for pneumothorax >20%, bed rest for pneumothorax <20%, oxygen therapy, watch for atelectasis, if present, deep-breathing aids (incentive spirometer). IPPB should be given only if the pneumothorax has been corrected or treated.

Respiratory Care and Surgery—Preoperative Evaluation

History: Chronic cough, shortness of breath, chest pain, smoking, edema in hands and feet, age, previous or existing heart disease, obesity, any chronic disease, or muscle weakness noted.

Physical examination: State of nutrition, movement of the chest wall and abdomen, pursed-lip breathing, rib cage deformities, and auscultation of the lungs.

HANDBOOK OF MECHANICAL VENTILATION

Salient Features

Handbook of Mechanical Ventilation, Third Edition is a concise, and practical resource designed primarily for bedside use by healthcare professionals.

Key objectives and audience:

- *Comprehensive audience:* The book is designed for a broad range of medical and paramedical professionals, including doctors, respiratory therapists, physiotherapists, nurses, and other allied healthcare staff.
- *Practical resource:* It serves as a practical and accessible resource for those involved in the care of patients requiring ventilatory support.
- *Goal:* The primary goal is to provide a clear, concise, yet comprehensive understanding of the principles and practices of mechanical ventilation and airway management.

Content and approach:

- *Focus on fundamentals:* The book presents the fundamentals of mechanical ventilation in an accessible and engaging manner, avoiding unnecessary technical minutiae that might overwhelm readers.
- *Bridging theory and practice:* It is designed to bridge the gap between theoretical concepts and clinical application.
- *Bedside utility:* It aims to be a concise handbook containing essential information that can be easily carried to the bedside.
- *Clinical relevance:* Special attention is given to:
  - Commonly encountered clinical scenarios
  - Ventilator settings
  - Patient–ventilator asynchronies
  - Troubleshooting methods

Value proposition:

- *Strong foundation*: Readers will undoubtedly gain a strong foundation in ventilator management.
- *Equipping professionals:* The book aims to equip healthcare professionals with the confidence and clarity needed to make informed decisions.
- *Examination preparation:* It will be of great benefit to respiratory therapists preparing for their examinations, as it encompasses much of the core syllabus.

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