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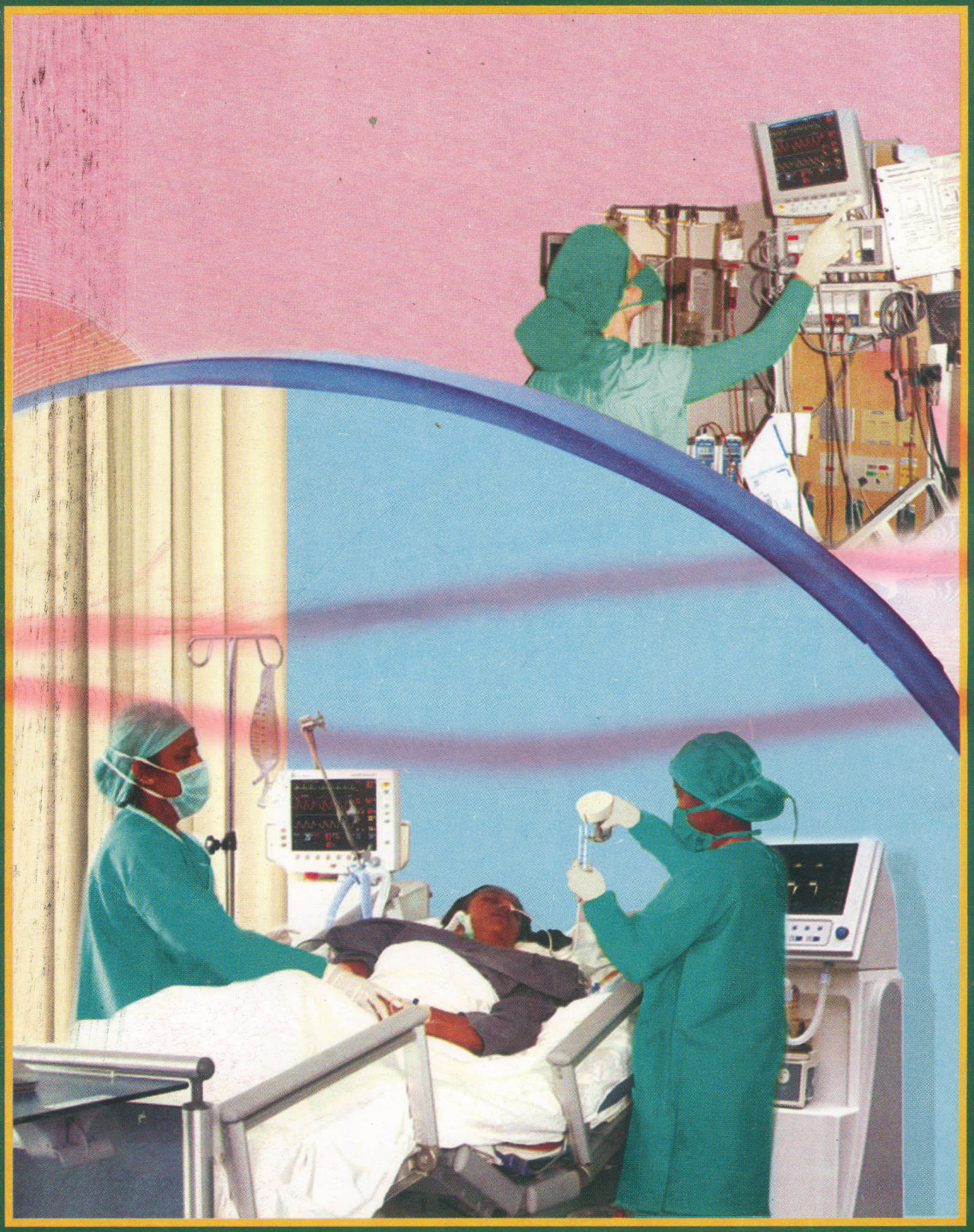
Indira Gandhi National Open University
School of Health Sciences

BNS-032
Nursing Management in Critical Care Conditions



CARE OF CRITICALLY ILL PATIENTS WITH PROBLEMS OF RESPIRATORY SYSTEM

2

"शिक्षा मानव को बन्धनों से मुक्त करती है और आज के युग में तो यह लोकतंत्र की भावना का आधार भी है। जन्म तथा अन्य कारणों से उत्पन्न जाति एवं वर्गगत विषमताओं को दूर करते हुए मनुष्य को इन सबसे ऊपर उठाती है।"

— इन्दिरा गांधी

"Education is a liberating force, and in our age it is also a democratising force, cutting across the barriers of caste and class, smoothing out inequalities imposed by birth and other circumstances."

– Indira Gandhi

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Block

2

CARE OF CRITICALLY ILL PATIENTS WITH PROBLEMS OF RESPIRATORY SYSTEM

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BLOCK INTRODUCTION

In block 1 you have studied about nursing care of critically ill patients clients with the problem of gastrointestinal system ill patients. In this block you will learn respiratory problems commonly seen among critically ill patients. This block will focus on the concepts, definition and meaning of the critical conditions, current assessment technologies and evidence based approaches of therapeutic management of these problems. Emphasis has been given on nursing assessment, nursing diagnosis and intervention that will guide you to provide competent care to critically ill clients with respiratory problems.

The block has been organised into 5 units as follows:

Unit 1 deals with acute respiratory distress syndrome

Unit 2 covers chest trauma hemothorax, pneumothorax, hydrothorax, pyothorax.

Unit 3 explain Pulmonary odema, Pulmonary, Embolism and atelectasis

Unit 4 focuses on acute exacerbation of chronic obstructive pulmonary disease and status asthmaticus.

Unit 5 includes pneumonia, interstitial lung disease and pleural effusion.

UNIT 1 ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

Structure

1.0 INTRODUCTION

Acute Respiratory Distress Syndrome (ARDS) is a progressive and severe form of acute respiratory failure. Various direct and indirect predisposing factors lead to alveolar capillary inflammation and damage. Interstitial edema prevents normal gas exchange. With increased understanding of the disease process, and current improved management the mortality has been reduced to 40% from a devastating rate of 70% **(American Lung Association, 2006).** In this unit we shall discuss about meaning, causes, predisposing factors, pathophysiology, clinical manifestation, nursing diagnoses. It also focuses on complications and the nursing management of complications of Acute Respiratory Distress Syndrome (ARDS).

1.1 OBJECTIVES

After completing this unit, you will be able to:

- explain the meaning of acute respiratory distress syndrome;
- state the etiology of Acute Respiratory Distress Syndrome;
- relate the clinical features and diagnostic findings to the pathophysiological process;
- list the possible nursing diagnoses of clients with Acute Respiratory Distress Syndrome;
- explain the management of patients with Acute Respiratory Distress Syndrome with rationale for the interventions; and
- discuss potential complications of Acute Respiratory Distress Syndrome (ARDS) and the related interventions.

1.2 MEANING AND PREDISPOSING FACTORS OF ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

We shall discuss the meaning, causes, predisposing factors and path physiology as given below:

1.2.1 Meaning of Acute Respiratory Distress Syndrome

Acute Respiratory Distress Syndrome (ARDS) is a pulmonary disorder of critically ill patients of any age following any injury to the body. The insult may be direct and indirect causing injury to lung and surrounding pulmonary tissue. This leads to alveolar capillary membrane damage, lung inflammation, pulmonary edema, shunting and hypoxemia. Shunt refers to the amount of blood that goes from right side to left side of the heart without getting oxygenated. This happens when blood passes through alveoli but does not pick up oxygen.

1.2.2 Predisposing Factors

Predisposing factors associated with Acute Respiratory Distress Syndrome (ARDS) are:

Direct injury

- Aspiration (gastric fluid, near drowning)
- Infectious pneumonia
- Lung contusions with trauma
- Toxic inhalation
- Upper airway obstruction (relieved)
- Severe acute respiratory syndrome (SARS)
- Neurogenic pulmonary edema
- Milliary tuberculosis.

Indirect Injury

- Sepsis
- Trauma
- Burns
- Massive Blood Transfusion
- Lung or bone marrow transplantation
- Drug or alcohol overdose
- Drug reaction
- Cardio pulmonary bypass surgery
- Acute Pancreatitis
- Multiple fractures
- Venous air embolism

- Amniotic fluid embolism
- High concentration of supplemented oxygen
- Near drowning
- Radiation therapy
- Disseminated intravascular coagulation.

1.3 PATHOPHYSIOLOGY CLINICAL MANIFESTATIONS AND DIAGNOSIS

You are now aware of the causes of Acute Respiratory Distress Syndrome (ARDS). Whatever be the cause, you will find the following pathological changes taking place.

- The basic pathology of ARDS is a diffuse inflammatory process that involves both lungs.
- The lung consolidation originates from a systemic activation of circulating neutrophils.
- The activated neutrophils become sticky and adhere to the vascular endothelium in the pulmonary capillaries.
- The neutrophils then release inflammatory mediators which cause cell damage, inflammation, fibrosis and increase membrane permeability to the alveolocapillary bed. The mediators are: (see box 1.1)
    - proteolytic enzymes
    - toxic oxygen products
    - arachidonic acid metabolites (prostaglandin, thromboxanes and leukotrienes)
    - other mediators (platelet activating factor, tumor necrosis factor, interleukins).
- The increased permeability of the alveolocapillary membrane causes fluid, proteins, plasma, blood to leak into the interstitial space and intra-alveolar spaces leading to gross pulmonary odema and hemorrhage that quickly leads to alveolar flooding and atelectasis.
- Atelectasis is due to reduced surfactant activity because the alveoli and respiratory bronchioles have become saturated with fluid or collapsed.
- This causes reduced lung compliance, decreased functional residual capacity, V/Q (Ventilation / perfusion) mismatch, increased dead space, severe hypoxemia, laboured breathing and pulmonary hypertension.
- Within 48 to 72 hours, interstitial and bronchoalveolar inflammation begins and epithelial and interstitial cells multiply.
- Hyaline membrane forms and collagen accumulates rapidly causing interstitial fibrosis and lung damage. See box 1.2 phases of Acute Respiratory Distress Syndrome (ARDS).

- Eventually inflammation, endothelial damage, capillary permeability leads to systemic inflammatory syndrome, multi organ failure and death.

Box 1.1: Examples of Pathological Responses to Biological Mediators

Response	Biological Mediators
Persistent inflammatory response	Cytokines: Interleukins(IIL-III-6) interferon-y (INF-y) tumor necrosis factor-á (TNF-á) complement, thromboxane
Endothelial membrane disruption	Complement, thromboxane, kinins, TNF- á, toxia oxygen metabolites, leukotrienes, prostaglandins(PGE and PGE2)
Selective vasoconstriction	Thromboxane, TNF- á, platelete-activating factor (PAF toxic oxygen metabolites)
Systemic vasodilatation	Complement, prostaglandins TNF- á, IL-1, IL-6
Myocardial depression	Complement, leukotrienes, TNF- á, myocardial depressant factor
Bronchoconstriction	Complement, thromboxane, leukotrienes, PAF

Box 1.2: Phases of Acute Respiratory Distress Syndrome (ARDS)

Phase	Characteristic
Exudative phase	Injury to endothelium and epithelium result in inflammation and leakage of fluid into the alveoli.
Fibroproliferative phase	Fibroblasts reproduce and flow into the lung tissue. Injury can improve or persist.
Fibrosis phase	Pulmonary fibrosis develops at varying levels depending on the resolution of inflammation.

1.3.2 Clinical Manifestations

Clinical manifestations include following:

- Marked dyspnea, early
- Refractory hypoxemia (unresponsive to oxygen therapy)
- Diffusive alveolar infiltrates evident on X-Ray of chest
- Pulmonary artery occlusive pressure < 18mm Hg
- Rapid shallow respiration with intercostal and suprasternal retractions observed on early inspiration
- Crackles, rhonchi, wheezes on auscultation

- Respiratory alkalosis (acidosis in advanced stage)
- Decreased lung compliance
- Cyanosis or mottled skin
- Hypotension (late)
- Decreased cardiac output (late).

1.3.3 Diagnosis

The actual diagnosis of the condition is based on physical examination, arterial blood gas analysis (ABG), radiographic findings.

- ABG analysis initially reveals respiratory alkalosis: elevated pH, low PaO2, normal or low PaCO2. This is because O2 cannot travel through collapsed and consolidated lung tissue.
- Chest X-Ray may look like pulmonary edema of cardiac origin, because of alveolar infiltration. But the cardiac silhouette is not enlarged.
- The distinctive diagnostic factor therefore is the Pulmonary arterial occlusive pressure which is low, <18mmHg and high in cardiac failure, >20mmHg. The PAOP is the pressure of left atrium. So if the left arterial pressure is low it indicates that the left heart failure is absent and the clinical features are due to non cardiac pulmonary edema.

Check Your Progress 1

i) The diagnosis of ARDS is made by following:

a) careful assessment of radiographic changes

b) tachycardia

c) tachypnoea

ii) **List the clinical manifestation of** Acute Respiratory Distress Syndrome

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1.4 NURSING DIAGNOSIS AND MANAGEMENT

We shall focus on nursing dignosis and collaborative management as given below:

1.4.1 Nursing Diagnosis

Based on your understanding of the pathology and the resulting clinical features of the disease process you can list out the following Nursing Diagnosis for your client.

- Impaired gas exchange related to hypoxemia and alveolar leaks found in alveolar capillary injury states.

- Ineffective airway clearance related to increased secretion, production and decreased ciliary motion. .
- Ineffective breathing pattern related inadequate gas exchange, increased secretion, fear and exhaustion.
- Risk for aspiration
- Altered nutrition: less than body requirement
- Risk for infection related to invasive monitoring devices and endotracheal intubations Altered tissue perfusion
- Ineffective coping with illness (patient and family) related to critical illness anxiety, fear of death, role changes and permanent disabilities.

***The optimal expected outcomes of management*:**

- Oxygen saturation within expected range of PaO2 55-80 mm Hg or SaO_2 **88-95 mm** Hg
- Absence of complications related to PEEP therapy
- Cardiac output within expected range
- Chest radiographs return to baseline
- Hemodynamic stability, normal Blood pressure, Heart Rate, Central Venous Pressure
- Activity tolerance in expected range
- Family coping with acute illness.

1.4.2 Collaborative Management

This is a condition where you will require a multidisciplinary approach.

- Antibiotics may be administered for preventing infection.
- **Mechanical Ventilation** to improve gas exchange.
- The main aim of ventilator support is to give a low tidal volume and a Positive end Expiratory pressure (PEEP) of up to 24cm of H_2O, PEEP helps to prevent the alveoli from continuously opening and closing which results in more inflammation.
- PEEP may cause reduced venous return which in turn may result in low cardiac output which is a side effect of high PEEP.
- Inverting the (you may know that normal inspiratory: expiratory (I:E ratio is 1:2) ratio helps better oxygenation with prolonged inspiration. Since this is opposite of normal respiration, patients need to be sedated.
- **Kinetic therapy** is the continuous side-to-side turning of patients in these cases at least every 2 hours to improve mobilization of static fluid and improve V/Q balance, and oxygenation. Prone position is also very effective but difficult without special kinetic beds. Fowler's position facilitates lung expansion and reduces breathing effort, when patients are in weaning mode of ventilation.

- **Fluid Balance** is very important to prevent hypotension. Also PEEP mode ventilation may cause reduced venous return. Once fluid balance is restored, diuretics may be given to reduce pulmonary congestion. It is important to check the intake and output balance, vital signs, daily weight if possible, hemodynamic values to help maintain fluid balance.
- **Nutrition** must be provided either by external or parenteral route within 24 hours. Calories should be calculated with the help of dietitian.

1.4.3 Complications and Nursing Management

1) **Ventilator Associated Pneumonia (VAP):** VAP is a term you will hear in all CCU / VAP may be induced by prolonged ventilation, weak immune system, aspiration of gastric contents or oral mucus, traumatic intubation and contaminated equipment. Nursing patients in 30 degrees elevation of head end, use of standard precautions to prevent infection, closed system endotracheal suctioning and oral hygiene 4th hourly can prevent VAP.

2) **Multi-organ Failure :** Older patients >65 year are at risk.

3) **Stress Ulcers:** Bleeding from ulcers in critically ill patients is common and it can be prevented by starting enteral nutrition early, administering H2 receptor blockers or proton pump inhibitors.

4) **Skin Breakdown:** Immobility, medical equipment used, malnutrition, catabolic states, incontinence, shock, high doses of vasopressors can all contribute to skin breakdown. Interventions include 2 hourly turning the patient, care of pressure points, using pressure relieving mattress, changing angle of ET tube 8 hourly and using incontinence collection devices.

5) **Barotraumas:** Caused by over distention and rupture of alveoli during mechanical ventilation. Air is found where it should not be causing pneumothorax, subcutaneous emphysema. Use of low tidal volume can prevent this.

6) **Deep Vein Thrombosis (DVT):** You know that long term inactivity due to imposed and critical illness causes sluggish venous return. DVT may lead to pulmonary embolus a life threatening complication. Low dose heparin administration initiated within 48 hours of admission, graded elastic stocking frequent mobilization and ambulation reduce the risk of DVT.

Check Your Progress 2

i) The strategies to prevent ventilator associated lung injury include:

   a) Allow tidal volume, low PEEP.

   b) Low tidal volume, high PEEP

   c) Volume controlled ventilation

ii) Complication associated with ARDS include all of the following EXCEPT

   a) DVT

   b) Multiple Organ Failure

c) Pneumothorax

d) Intracranial Haemochage

iii) Complications associated with ARDS may be limited by the following nursing interventions:

a) Raising the head of the bed at least 30 degrees

b) Continuous administration of sedation.

c) Bed rest throughout the ventilatory period.

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1.5 LET US SUM UP

In this unit you have studied the meaning of ARDS, its direct and indirect causes and precipitating factors. You have also learnt abut the –

- ❑ The pathological changes and the phases of ARDS,
- ❑ The clinical manifestations along with the diagnostic findings.
- ❑ You have also learned the nursing diagnosis and possible expected outcomes.
- ❑ Since this is a condition that has to have a multidisciplinary approach, the collaborative management has been discussed, along with the nursing interventions with rationale.
- ❑ You have been explained the possible complications of a patient with ARDS and the nursing interventions that you must take to prevent the same.

1.6 GLOSSARY

ARDS	:	Acute Respiratory Distress Syndrome
V/Q	:	Ventilation Perfusion ratio that is normally 0.8. When blood passing through alveoli does not get oxygenated this balance is altered.
PaO2	:	The partial oxygen pressure of blood when arterial blood is put through blood gas analysis. The normal value is 80 - 100mm Hg
PaCO2	:	The partial pressure of carbon dioxide in arterial blood. Normal value is 35 - 45mmHg
PEEP	:	Positive End Expiratory Pressure is a mode of ventilation used to keep the alveoli open.
Respiratory alkalosis		It is the term used when Ph is >7.45 and the PaCO2 is normal or low.
Respiratory acidosis		It is the term used when the Ph is <7.35 and the Pa CO2 is high or more than 45mmHg

1.7 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

i) a

ii) Clinical manifestation includes following:

- Marked dyspnea, early
- Refractory hypoxemia (unresponsive to oxygen therapy)
- Diffusive alveolar infiltrates evident on X-Ray of chest
- Pulmonary artery occlusive pressure < 18mm Hg
- Rapid shallow respiration with intercostal and suprasternal retractions observed on early inspiration
- Crackles, rhonchi, wheezes on auscultation
- Respiratory alkalosis (acidosis in advanced stage)
- Decreased lung compliance
- Cyanosis or mottled skin
- Hypo tension (late)
- Decreased cardiac output (late).

Check Your Progress 2

i) b)

ii) d)

iii) a)

iv) a

1.8 REFERENCES

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UNIT 2 CHEST. TRAUMA HEMOTHORAX, PNEUMOTHORAX, HYDROTHORAX, PYOTHORAX

Structure

2.0 INTRODUCTION

Thoracic injuries may range from simple abrasions to life threatening insults to the thoracic viscera. Although these injuries are associated with high mortality rate usually with chest tube insertion, mechanical ventilation, pain control measures and supportive care they can be managed. In this unit you will learn about chest and pleural space injuries caused by chest trauma which include, conditions like flail chest, pneumothorax, hemothorax. Pyothorax and Hydrothorax.

2.1 OBJECTIVES

After completing this unit you will be able to:

- explain the meaning and causes of chest trauma;
- differentiate among pneumothorax, hemothorax, chylothorax, pyothorax, and hydrothorax; and
- describe the pathophysiology, and assessment Nursing management of various types of chest trauma.

2.2 CHEST AND PLEURAL SPACE INJURIES

Let us begin with meaning assessment and diagnosis of chest and pleural space injuries.

Meaning of chest and pleural space injuries

Chest injury is the leading cause of death from trauma. You must be aware that Motor vehicle crashes, crimes and falls are on the rise very day. Chest injuries

can be mild to fatal. Pleural space injuries are caused by disruption of the intrathoracic structure that allows air or blood to build up in the pleural layers, thereby leading to a decrease in the negative pressure in the intrathoracic cavity. This in turn produces tension pneumothorax or tension hemothorax. Pleural space injuries are caused due to blunt (unrestrained driver whose chest hits the steering wheel) or penetrating (stab or bullet) trauma.

Assessment

Rapid and continuous assessment of airway, breathing, ABC is vital to patient survival.

On assessment of clients with chest injury you will find respiratory distress with altered ventilation. This leads to impaired gas exchange that you will see is evidenced by **restlessness, anxiety, tachypnea, decreased oxygenation, poor color and diaphoresis**. It is important for you to assess continuously because the condition can deteriorate. A small injury can expand and lead to life threatening situation.

Diagnosing Chest and Pleural Space Injury

Chest and pleural space injury can be diagnosed by chest radiography. If you cannot see smaller injuries (less than 20%) of the thoracic cavity with the help of X-ray chest you can see it through a CT scan. You need to monitor Oxygen saturation and do Arterial blood gas (ABG) analysis to assess the severity of the problem. The various types of traumas are discussed in following sub-sections:

2.2.1 Flail Chest

Multiple rib fractures may impair the chest wall stability and normal chest wall function. When two or more consecutive ribs are fractured in multiple places, a free floating segment of the chest wall or flail chest results.

You can see in figure 2.1 how the flail segment of the chest wall moves in, or rather gets sucked in during inhalation and moves outward during expiration. This is called **paradoxical movement.**

This causes impaired ventilation and gas exchange. If associated with underlying pulmonary contusion it may lead to respiratory failure.

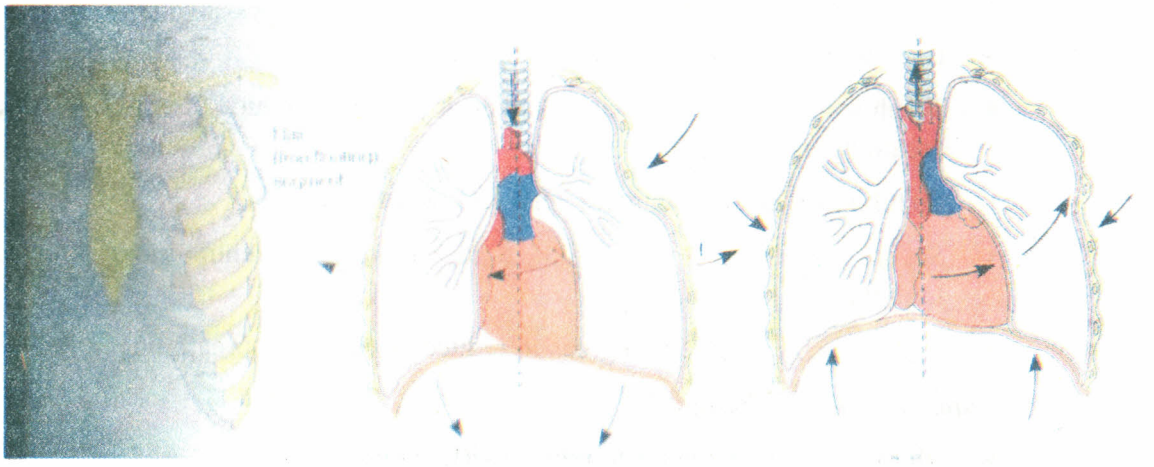

Fig. 2.1: Flail chest with paradoxical movement

Nursing Assessment

- First you must assess for circulation, airway, breathing (CAB) (now the terms CAB is used in place of ABC as per recent guidelines). Specifically observe the patient's breathing and chest wall movement. If breathing is rapid and labored, you will see assymetrical and uncoordinated chest wall movement.
- Continuously monitor the level of consciousness.
- Assess for pain using a pain scale and subjective data.
- You will find on assessment that the patient has dyspnea and pain on inspiration specially on inspection. Chest wall movement will be unequal.
- On palpation you will feel crepitus from bony fragments.
- You will feel on percussion Hyper resonance.
- On auscultation you will hear diminished breath sounds and may hear crackles.
- Chest X-Ray will show rib fractures.
- Arterial blood gas (ABG) analysis and oxygen saturation will alter depending on the degree to which ventilation and gas exchange have been impaired.

Nursing Diagnosis and Management

Acute Pain :

- Pain interferes with coughing and poor lung expansion that may lead to pneumonia or atelectasis.
- The physician may prescribe intercostal nerve block or epidural analgesia.
- Be alert that Narcotic analgesia may cause respiratory depression.
- Don't wait for pain to be intense.
- Increasing rate and shallow respiration and reluctance to move or cough indicate inadequate pain control.

Ineffective airway clearance :

- Practice aggressive respiratory hygiene.
- Assess and maintain a patent airway.
- Turn patient 2 hourly.
- Encourage deep breathing and coughing.
- Teach splinting of chest with blanket or pillow to reduce discomfort while breathing and coughing.
- Suction airway and maintain adequate ventilation.
- Secure Endotracheal tube. If tube is in one bronchus ventilation of affected lung may be prevented.
- Elevate head end to 30 degrees to reduce workload of breathing.

Impaired Gas Exchanges:

- Monitor vitals, oxygen saturation, ABG color.
- Assess for signs of anxiety, restlessness, confusion, lethargy, headache. These indicate hypoxemia or hypercapnea.

- Maintain oxygen therapy and mechanical ventilation as ordered.
- Before suctioning, hyper oxygenate to reduce hypoxia during suctioning.
- Monitor intake output balance, weight, central venous pressure, pulmonary artery pressure.
- Follow fluid restriction if ordered.
- These help to reduce risk of pulmonary edema.
- Space out activities to allow uninterrupted periods of sleep. Rest reduces metabolic rate and oxygen consumption.

Check Your Progress 1

Mark T for True and F for False

a) In flail chest during inspiration chest wall will move out. ❑

b) Pain control medication is given only when increases. ❑

c) Patients should be kept with head end elevated. ❑

d) Elevate head end to 30 degrees to reduce ❑

e) Rest increases metabolic rate and oxygen consumption breathing effort ❑

2.2.2 Pneumothorax and Hemothorax

Let us begin with –

Pneumothorax

Definition: Pneumothorax or collapsed lung is a potential medical emergency caused by accumulation of air within the pleural space. It may compromise respiration by interfering with the expansion of the lung.

A pneumothorax is air entry into the pleural space between the visceral and parietal layers, producing partial or complete lung collapse. You will find these patients usually have a fractured rib or penetrating wound.

Etiology

Pneumothorax is caused due to blunt rupture of the lung surface, rather than laceration by broken ribs. It most commonly arises:

a) Spontaneously (most commonly in tall slim young males and in Marfan syndrome)

b) Following a penetrating chest wound

c) Following Barotrauma to the lungs

It may also be due to:

- Chronic lung pathologies including emphysema, asthma
- Acute infections and Chronic infections, such as tuberculosis
- Cancer
- Rare diseases that are unique to women such as Catamenial pneumothorax (due to endometriosis in the chest cavity) and lymphangioleiomyomatosis (LAM).

Clinical Findings and Diagnostic Measures

Breath sounds will be decreased on the affected side. Pleuritic pain may not develop for hours. If the pneumothorax is large, hyper resonance to percussion may be present. Respiratory distress, shock, unilateral absence of breath sounds, and hyper resonance to percussion indicate tension pneumothorax.

Diagnostic Measures

Absence of audible breath sounds through a stethoscope

- Chest x-ray by observing a pleural stripe that has fallen away from the chest wall, with absence of lung markings beyond the stripe. A very small pneumothorax may not be visible on x-ray. Tension pneumothorax must be diagnosed on clinical grounds, as the physician must intervene before an x-ray can be taken.

- On finding the triad of respiratory distress, shock, and unilateral absence of breath sounds, a needle should be inserted in the second intercostal space anteriorly. The hiss of escaping air establishes the diagnosis.

Pathophysiology

The lungs are located inside the chest cavity, which is a hollow space. Air is drawn into the lungs by the diaphragm (a powerful abdominal muscle). The pleural cavity is the region between the chest wall and the lungs. If air enters the pleural cavity, either from the outside (open pneumothorax) or from the lung (closed pneumothorax), the lung collapses and it becomes mechanically impossible for the injured person to breathe, even with an open airway. If a piece of tissue forms a one-way valve that allows air to enter the pleural cavity from the lung but not to escape, overpressure can build up with every breath; this is known as **tension pneumothorax**. It may lead to severe shortness of breath as well as circulatory collapse, both life-threatening conditions. This condition requires urgent intervention.

Spontaneous Pneumothorax

This condition occurs as a result of rupture of air into the pleural space without obvious cause.

Manifestations

- Abrupt onset
- Pleuritic chest pain
- Dypnea, shortness of breath.

Spontaneous pneumothorax may be primary or secondary.

Primary spontaneous pneumothorax: It occurs in the absence of lung disease. It is commonly found in young adolescents or adults up to 30 years of age and with family history of smoking.

Manifestations include following:

- Tachypnea
- Tachycardia
- Unequal lung expansion

Secondary pneumothorax

It occurs as a complication of lung disorder. This largely occurs in chronic obstructive pulmonary disease (COPD) patients. Other causes include asthma, cystic fibrosis, pneumonia, sarcoidosis, necrotizing pneumonia and histiocystosis X.

Manifestations

Decreased breath sounds and hyper resonant tone on percussion on affected side.

Traumatic pneumothorax

It occurs in critically ill patients due to invasive procedures and barotraumas. Accidental air entry into the pleural space causes iatrogenic pneumothorax. It may occur in patients with central line catheters and barotraumas in positive pressure mechanically ventilated patients.

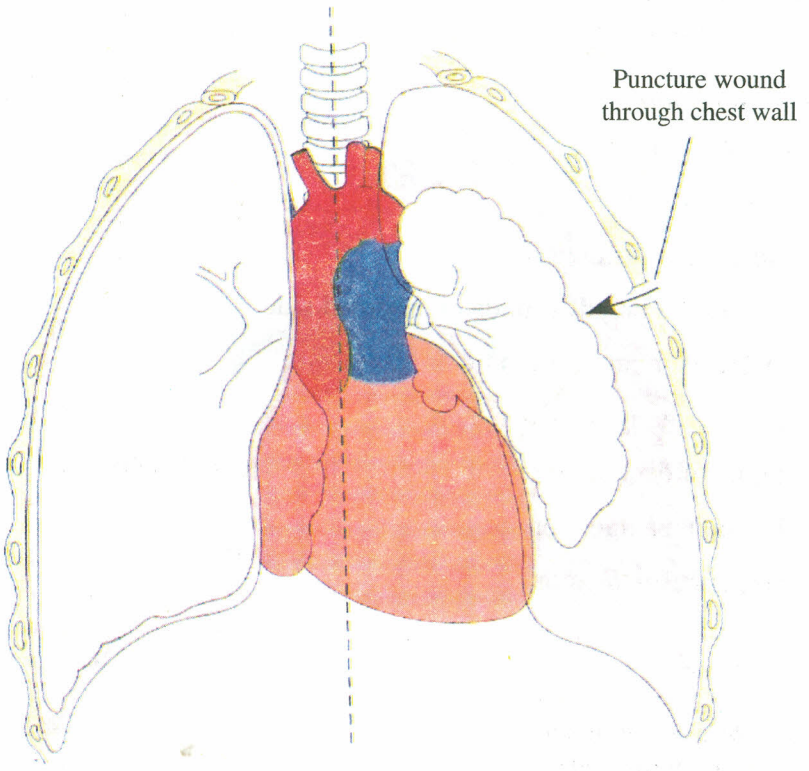

Fig. 2.2: Traumatic pneumothorax

Symptoms

- Hypoxemia
- Apprehension
- Respiratory distress (severe tachypnea)
- Increasing peak and mean airway pressures, decreasing lung compliance, and auto-positive end expiratory pressure in patients receiving mechanical ventilation.
- Cardiovascular collapse (heart rate > 140 / minute) and any of the following: pulse less electrical activity, peripheral cyanosis, hypotension.

- Respiratory distress usually is not seen until the pneumothorax exceeds 40% of one lung's volume, unless the patient has pre-existing lung disease or parenchymal lung injury. If pressure increases within the pleural space (tension pneumothorax), venous return to the chest slows, and shock develops.
- Sudden shortness of breath, dry coughs, cyanosis (turning blue) and pain felt in the chest, back and/or arms are the main symptoms.

Tension pneumothorax

It is a life threatening situation. You may get this condition as a late complication of tracheobronchial injuries or mechanical ventilation. Air enters pleural space and is trapped without exit. The pressure of air in the pleural space exceeds that of atmospheric pressure. The mediastinum shifts where placing torsion on the inferior vena cava and reducing venous return to the right side of the heart. This causes compression of trachea, heart, lungs, and great vessels and prohibits them from adequate functioning. Result is ventilation failure, reduced venous return and low cardiac output.

Signs and Symptoms

- Chest asymmetry
- Tracheal shift
- Neck vein distention
- Decreased breath sounds on affected side
- Decreased blood pressure
- Poor tissue perfusion-cyanosis
- Signs of decreased cardiac output- hypoxia, pale cool, skin
- Dysppnoea, tachypnooa
- Hyper-resonance or percussion

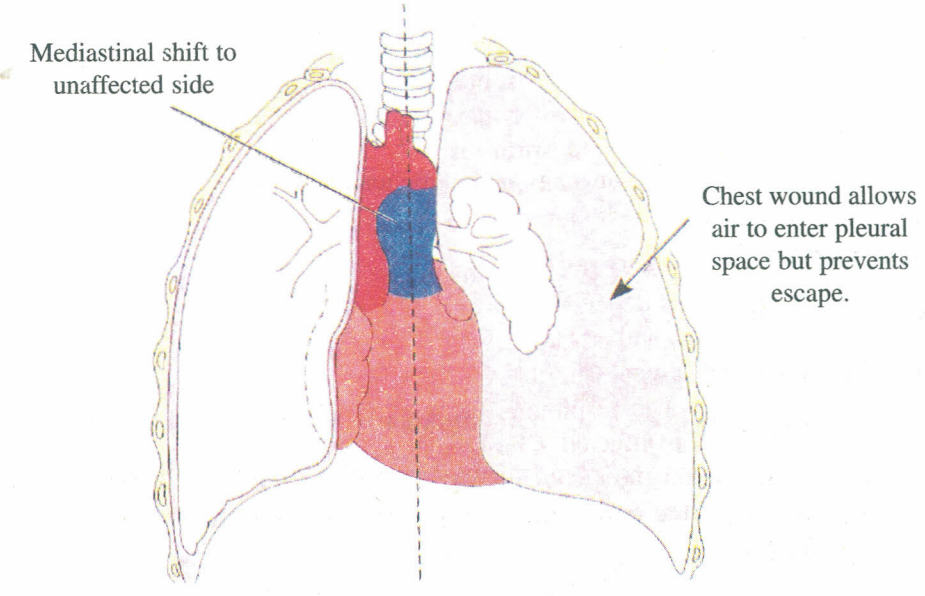

Fig. 2.3: Tension pneumothorax

Treatment of pneumothorax

A very small (less than 1-2 cm) pneumothorax in an otherwise healthy individual can be observed without treatment if it remains stable on subsequent x-rays (6-8 hours later). Otherwise, evacuating the pneumothorax by chest tube insertion should be done.

Immediate decompression by inserting a 14 – 16 gauge needle into the pleural space between the 2nd to 4th anterior intercostals space can relive it.

- Immediate rush of air followed by improved patient ventilation should follow.
- Oxygen supplement is provided throughout.
- After the emergent decompression, chest drains are introduced to allow lungs to expand and prevent recurrence.
- Continued assessment and reassessment by nurse is required.
- Pleurodesis – creation of adhesions between the parietal and visceral layers, to prevent the occurrence of recurrent pneumothorax. It involves introducing a chemical agent like doxycycline into the pleural space. Subsequent inflammatory response produces scar tissue and adhesions.
- Surgery through video assisted thoracoscopy for those patients at high risk of recurrence of the condition. A thoracotomy is done to excise or sew the blebs at the apices of lungs. The overlying pleura are then roughened to initiate scarring and adhesions to the lung surface to prevent recurrence.

Hemothorax

Hemothorax is a life threatening condition that results from accumulation of blood in pleural cavity. Hemothorax is defined as intrathoracic blood loss of 1.5 to 4 liters. When blood collects in the pleural space pressure on the affected lung impairs ventilation and gas exchange.

Causes

It may be caused by chest trauma, surgery or diagnostic procedures.

A left massive hemothorax is more common than a right one. It may be caused by aortic rupture.

The source of bleeding may be a laceration of a large systemic blood vessel or internal mammary artery or mediastinal structure. The serous membrane lining the thorax ruptures allowing blood to spill into pleural space.

Signs and Symptoms

You may receive such a patient in the emergency unit in a state of cardiopulmonary arrest.

In those patients not in arrest, you will recognize manifestations of hypovolemic shock, like dyspnea, tachypnea, and cyanosis.

Treatment

You will hear diminished lung sounds on auscultation, dull tone on percussion over the area of collected blood typically at the base of the lung and unequal chest wall movement.

Diagnosis Chest X-Ray is the method for confirming diagnosis. A tracheal deviation occurs.

You will need to correct shock by infusing fluids through large bore intravenous lines as prescribed.

Bleeding will stop only when the pressure in the pleural cavity is equal to or greater than the pressure in the blood vessel damaged.

Insertion of a chest tube to drain out the blood is one to relieve the symptoms. Thoracentesis or thoracotomy may be done.

The priority nursing diagnosis are Impaired gas exchange, **Ineffective breathing pattern, Decreased cardiac output, Risk for fluid volume deficit.**

You are now familiar with the interventions for these problems as discussed for pneumothorax.

Check Your Progress 2

State True or False

a) The affected side of the chest in pneumothorax will have reduced chest expansion. ❑

b) Tension pneumothorax is life threatening. ❑

c) The needle to enter the pleural space is inserted at the 6^{th} and 7^{th} intercostals space. ❑

d) Tracheal shift to one side occurs in spontaneous pneumothorax. ❑

e) Pleurodesis is creation of adhesions ❑

2.2.3 Pyothorax/Pleural Empyema

Pyothorax

Definition Pleural empyema (also known as a pyothorax or purulent pleuritis) is an accumulation of pus in the pleural cavity.

Etiology

Most pleural empyemas arise from an infection within the lung (pneumonia), often associated with parapneumonic effusions.

Stages

There are three stages of pyothorax which include exudative, fibrinopurulent and organizing.

In the **exudative stage**, the pus accumulates. This is followed by the fibrinopurulent stage in which there is loculation of the pleural fluid (the creation of grapelike pus pockets). In the final organizing stage, scarring of the pleural space may lead to lung entrapment.

Signs and Symptoms

Symptoms of empyema may vary in severity. Typical symptoms include:

- Fever
- Chest pain or discomfort
- Cough
- Sweating and shortness of breath.
- Clubbing of the fingernails may be present.
- There is a dull percussion note and reduced breath sounds on the affected side of the chest.

Diagnosis

- Chest x-ray
- CT scan
- Ultrasonography.
- Diagnosis is confirmed by thoracentesis; frank pus may be aspirated from the pleural space. The pleural fluid typically has a low pH (< 7.20), low glucose (<60 mg/dL), and contains infectious organisms.

Treatment

Definitive treatment for empyema entails drainage of the infected pleural fluid. A chest tube may be inserted, often using ultrasound guidance. Intravenous antibiotics are given. If this is insufficient, surgical debridement of the pleural space may be required.

2.2.4 Hydrothorax

A hydrothorax is a condition that results from serous fluid accumulating in the pleural cavity. This specific condition can be related to cirrhosis with ascitis in which ascitic fluid leaks into the pleural cavity. Hepatic hydrothorax is often difficult to manage in end stage liver failure and often fails to respond to therapy.

In similar conditions of such as pleural effusion, hemothorax the fluid is blood (as in case of major chest injuries), in pyothorax the fluid is pus (resulting from chest infections), and in chylothorax it is lymph fluid (resulting from rupture of the thoracic duct).

Treatment

Treatment of hydrothorax is difficult for several reasons. The underlying condition needs to be corrected, however, often the source of the hydrothorax is end stage liver disease and corrected only by transplant. Chest tube placement should not occur.

2.2.5 Hemopneumothorax

Hemopneumothorax, is a medical term describing the combination of two conditions: pneumothorax, or air in the chest cavity and hemothorax, or blood in the chest cavity.

The resulting condition is a serious state which may interfere with breathing. The blood and air fill the pleural space, the space between the lung and the chest wall, putting pressure on the lung. This can cause the lung to collapse and unable to be fill with air (unilateral hemopneumothorax). In the most of serious cases,

both lungs may collapse (bilateral hemopneumothorax). Death may follows if respiration is inhibited seriously enough.

Treatment

Treatment for this condition is the same as for hemothorax and pneumothorax tube thoracostomy is indicated in which, the insertion of a chest drain through an incision made between the ribs, into the intercostal space is done. Commonly, surgery is needed to close off whatever injuries caused the blood and air to enter the cavity (e.g., stabbing, broken ribs).

2.3 LET US SUM UP

Thoracic injuries range from simple abrasions to life threatening insults to thoracic viscera. These injuries lead to morbidity and mortality. As a critical care nurse you have to prevent life threatening situation and organ functions a rising from severe injuries. In this unit we have discussed various injuries such as Chest and Pleural Space Injuries, Flail chest, Pneumothorax and Hemothorax, Pyothorax, Hydrothorax, Hemopneumothorax.

2.4 GLOSSARY

Pulmonary contusion : Abrupt chest compression resulting from sudden compression this type of lung tissue injury is frequently associated with flail chest.

Sarcoidosis : Sarcoidosis (from *sarc* meaning *flesh*, *-oid*, *like*, and *-osis*, *process*), is a disease in which abnormal collections of chronic inflammatory cells (granulomas) form as nodules in multiple organs.

Hystiocystosis X : It is characterized by an abnormal proliferation of histiocytes (an archaic term to activated dendritic cells andmacrophages). These diseases are related to other forms of abnormal proliferation of white blood cells, such as leukemias and lymphomas.

2.5 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

a) False

b) False

c) True

d) True

e) False

Check Your Progress 2

a) True

b) True

c) False

d) False

e) True

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UNIT 3 PULMONARY ODEMA, EMBOLISM AND ATELECTASIS

Structure

3.0 INTRODUCTION

In unit 1 and 2, you have learnt about Acute Respiratory Distress Syndrome (ARDS) and Chest Trauma. In this unit you will learn about pulmonary edema, pulmonary embolism and atelectasis which are not diseases in themselves but rather condition arising due to pathological changes occurring as a sequelae to various disease conditions. We shall focus on definition, concepts, causes, signs and symptoms, pathophysiology and medical and nursing management.

3.1 OBJECTIVES

After completing this unit, you will be able to:

- explain the meaning of Pulmonary Edema, Pulmonary Embolism and Atelactasis;
- describe the etiology and patho physiology of these conditions;
- state the classification on the basis of Pathophysiology;

- explain signs, symptoms and diagnostic measures;
- discuss the therapeutic / medical and surgical management; and
- describe the nursing management.

3.2 PULMONARY EDEMA

In this section we shall discuss about the concept and definition, etiology pathophysiology and medical, surgical and nursing management.

3.2.1 Concept and Definition

Pulmonary edema is an abnormal fluid accumulation in the alveoli and interstitial spaces of the lungs (Due to extravasations of fluid from the pulmonary vasculature into the interstitium and alveoli of the lung). It leads to impaired gas exchange and may cause respiratory failure. It is due to either failure of the heart to remove fluid from the lung circulation ("cardiogenic pulmonary edema") or a direct injury to the lung parenchyma ("non-cardiogenic pulmonary edema"). Treatment depends on the cause, but focuses on maximizing respiratory function and removing the cause.

3.2.2 Etiology and Pathophysiology

We shall discuss about the etiology of non-Cardiogenic and cardiogenic pulmonary edema separately. Let us begin with cause of non cardiogenic pulmonary.

Causes of non-cardiogenic pulmonary edema:

Direct Injury to Lung are mediated via the airways (e.g., aspiration) or as the consequence of blunt chest trauma. They include following:

- Chest trauma, pulmonary contusion
- Aspiration
- Smoke inhalation
- Pneumonia
- Oxygen toxicity
- Pulmonary embolism, re-perfusion.

Indirect injury is the consequence of mediators that reach the lung via the blood stream. Hematogenous Injury to Lung and is caused by following:

- Sepsis
- Non-thoracic trauma
- Leuko-agglutination reactions
- Multiple transfusions
- Intravenous drug use, e.g., heroin
- Cardiopulmonary bypass.

Possible Lung Injury and Elevated Hydrostatic Pressures

- **High altitude pulmonary edema and Neurogenic pulmonary edema:** These result as a consequence of acute changes in pulmonary vascular pressures, possibly the result of sudden autonomic discharge such as, Severe brain insult, e.g., subarachnoid haemorrhage. A study was done to describe a case report where a 45 year old woman who presented with neurogenic pulmonary edema. The neurogenic pulmonary edema developed after acute injuries to pulmonary central nervous system that this patient developed after a subarachnoid haemorrhage. The pathophysiological mechanism explained that it could have been due to alterations in capillary permeability or elevations in pulmonary venous hydrostatic pressure due to left ventricular dysfunction or both.
- **Re-expansion pulmonary edema:** This condition result from a sudden swing of pleural pressure, as well as due to transient damage to the pulmonary capillaries.

Causes of non-cardiogenic edema:

Raised pulmonary capillary pressure:

The associated condition related to various body organs include following:

Heart

- Myocardial infarction, ventricular septal rupture following infarction:
- High output heart failure due to septicaemia, thyrotoxic crisis, anaemia
- Valvular disorders : Acute aortic regurgitation, mitral stenosis, severe aortic stenosis
- Severe hypertension
- Acute arrhythmia
- Acute myocarditis
- Left atrial myxoma
- Cardiac tamponade
- Aortic dissection
- Cardiomyopathy

Renal

- Acute renal failure, chronic renal failure
- Renal artery stenosis.

Iatrogenic fluid overload

Increased pulmonary capillary permeability which includes the following:

- Acute respiratory distress syndrome (ARDS)
- Inhaled or aspirated toxic substances
- Liver failure
- Fat embolism or amniotic fluid embolism

Lymphatic obstruction:

- Mediastinal carcinomatosis
- Silicosis.

Pathophysiology

The lungs essentially provide the interface between air and blood. The lungs consist of a series of folded membranes (the alveoli), which are located at the ends of very fine branching air passages (bronchioles). Blood which arrives into the lungs from the pulmonary artery gets into smaller and smaller blood vessels until it ends up in the capillaries located within the walls of the alveoli, which is a very thin membrane. In this moist environment, oxygen diffuses from within the alveoli into the blood stream, while carbon dioxide moves out of the blood stream into the alveoli and is expelled out of the air passages. This is the normal mechanism of gas exchange.

Pulmonary edema refers to extravasations of fluid from the pulmonary vasculature into the interstitium and alveoli of the lung. The formation of pulmonary edema may be caused by 4 major pathophysiologic mechanisms:

- Imbalance of Starling forces (i.e., increased pulmonary capillary pressure, decreased plasma oncotic pressure, increased negative interstitial pressure)
- Damage to the alveolar-capillary barrier
- Lymphatic obstruction, and
- Idiopathic or unknown mechanism.

The three possible cause of leakage of intravascular fluid into the interstitial spaces of the lung and into the alveoli are the following :

i) **Volume:** Too much fluid is present in the delicate pulmonary capillaries.

ii) **Pressure:** Pulmonary capillaries constrict to such an extent that fluid is forced across the capillaries into the alveoli.

iii) **Capillary injury:** The pulmonary capillary membrane itself leaks even with a normal amount of blood flow under normal pressure.

Normally, there is a balance between the hydrostatic and colloidal oncotic pressures in the pulmonary capillaries.

The extent to which fluid accumulates in the interstitium of the lung depends on the balance of hydrostatic and oncotic forces within the pulmonary capillaries and in the surrounding tissue. Normally, there is a balance between the hydrostatic and colloidal oncotic pressures in the pulmonary capillaries.

- If the hydrostatic pressure increases or the colloidal oncotic pressure decreases, the net effect will be fluid leaving the pulmonary capillaries and entering the interstitial space. This stage is referred to as interstitial edema. At this stage, the lymphatic can usually drain away the excess fluid. Hydrostatic pressure favours movement of fluid from the capillary into the interstitium.
- The oncotic pressure, which is determined by the protein concentration in the blood, favours movement of fluid into the vessel. Albumin, the primary

protein in the plasma, may be low in certain conditions such as cirrhosis and nephrotic syndrome. Hypoalbuminemia favours movement of fluid into the tissue for any given hydrostatic pressure in the capillary. If the fluid leak from the pulmonary capillaries, it will enter the alveoli. This stage is referred to as alveolar edema.

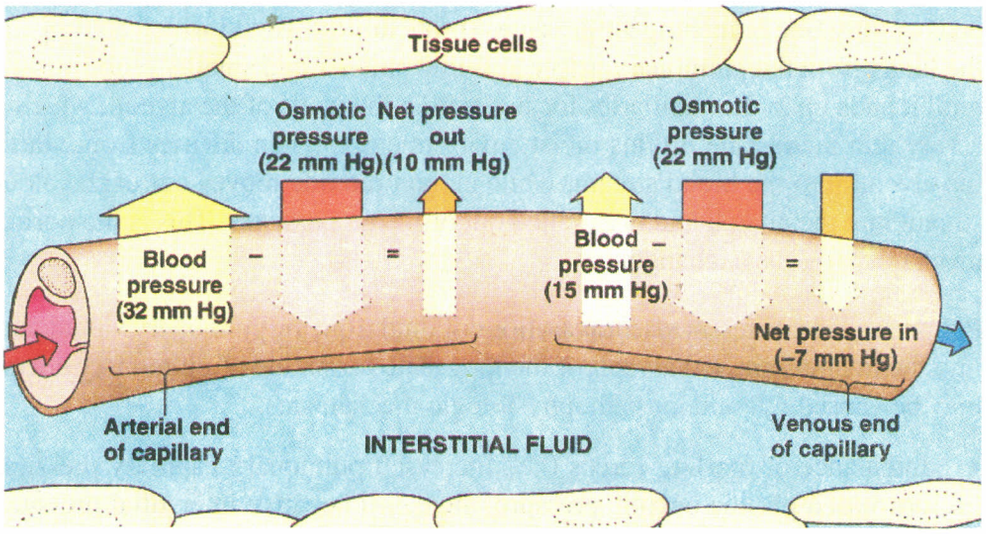


Fig. 3.1: Mechanism of fluid accumulation

Stages of cardiogenic pulmonary edema (CPE)

The progression of fluid accumulation in CPE can be identified as 3 distinct physiologic stages.

Stage 1

- Elevated left atrial pressure
- Distention and opening of small pulmonary vessels
- At this stage, blood gas exchange does not deteriorate, or it may even be slightly improved.

Stage 2

- Fluid and colloid shift into the lung interstitium from the pulmonary capillaries
- But an initial increase in lymphatic outflow efficiently removes the fluid
- The continuing filtration of liquid and solutes may overpower the drainage capacity of the lymphatics. (In this case, the fluid initially collects in the relatively compliant interstitial compartment, which is generally the perivascular tissue of the large vessels, especially in the dependent zones)

The accumulation of liquid in the interstitium may compromise the small airways

Hypoxemia at this stage is rarely of sufficient magnitude to stimulate tachypnea (Tachypnea at this stage is mainly the result of the stimulation of juxtapulmonary capillary (J-type) receptors, which are non-myelinated nerve endings located near the alveoli.

Leading to mild hypoxemia

Stage 3

As fluid filtration continues to increase, filling of loose interstitial space occurs

Fluid then accumulates in the relatively noncompliant interstitial space

Interstitial space can contain up to 500 ml of fluid

With further accumulations, the fluid crosses the alveolar epithelium in to the alveoli

Alveolar flooding

At this stage, abnormalities in gas exchange are noticeable, vital capacity and other respiratory volumes are substantially reduced, and hypoxemia becomes more severe

Non-cardiogenic Pulmonary Edema

In non-cardiogenic pulmonary edema (hydrostatic pressures are normal)

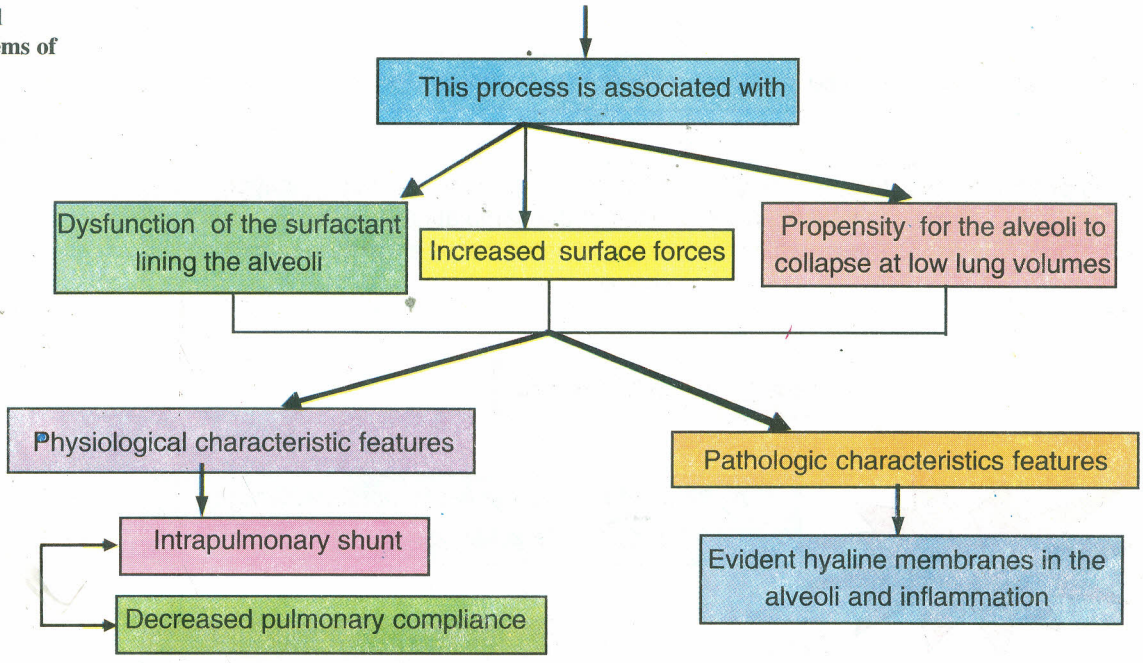
Damage of the pulmonary capillary lining

Fluid follows the protein

Oncotic forces are shifted from the vessel to the surrounding lung tissue

Leakage of proteins and other macromolecules move into the tissue

Lung water increases



3.2.3 Signs, Symptoms and Diagnostic Measures

In a typical case of acute pulmonary edema you will find rapid onset of dyspnea at rest, tachypnea, tachycardia, and severe hypoxemia. You may find Hypertension is usually present due to release of endogenous catecholamines. Patients may be sitting upright, they may demonstrate air hunger, and they may become agitated and confused.

- Patients usually appear anxious and diaphoretic.
- Hypertension is often present because of the hyperadrenergic state. Hypotension indicates severe Left ventricular systolic dysfunction and the possibility of cardiogenic shock. Cool extremities may indicate low cardiac output and poor perfusion.
- On Auscultation of the lungs you will hear fine crepitant rales, but rhonchi or wheezes may also be present. Rales are usually heard at the bases first; as the condition worsens, they progress to the apices.
- Cardiovascular findings are usually notable for S_3, accentuation of pulmonic component of S_2 and jugular venous distension. Auscultation of murmurs can help in the diagnosis of acute valvular disorders manifesting with pulmonary edema.
- Aortic stenosis is associated with a harsh crescendo-decrescendo systolic murmur, which is heard best at the upper sternal border and radiating to the carotid arteries.
- In contrast, acute aortic regurgitation is associated with a short, soft diastolic murmur.
- Acute mitral regurgitation produces a loud systolic murmur heard best at the apex or lower sternal border. In the setting of ischemic heart disease, this may be a sign of acute myocardial infarction (MI) with rupture of mitral valve chordae.

- Mitral stenosis typically produces a loud S_1, opening snap, and diastolic rumble at the cardiac apex.

- Another notable physical finding you will come across is skin pallor or mottling resulting from peripheral vasoconstriction, low cardiac output, and shunting of blood to the central circulation in patients with poor left ventricular (LV) function and substantially increased sympathetic tone. Skin mottling at presentation is an independent predictor of an increased risk of in-hospital mortality.

- Patients with concurrent right ventricular (RV) failure may present with hepatomegaly, hepatojugular reflux, and peripheral edema.

- Severe CPE may be associated with a change in mental status, which may be the result of hypoxia or hypercapnia. Although CPE is usually associated with hypocapnia, hypercapnia with respiratory acidosis may be seen in patients with severe CPE or underlying COPD.

General Symptoms of pulmonary edema: It **is** due to its main complication of hypoxia. The signs and symptoms includes the following:

- Difficulty in breathing
- Coughing up blood : A classical sign of pulmonary edema is the production of pink frothy sputum
- Occasionally, hoarseness may be present as a result of recurrent laryngeal nerve palsy from mitral stenosis or pulmonary hypertension (Ortner sign).
- Excessive sweating,
- Anxiety, and pale skin.
- If left untreated, it can lead to coma and even death.

If pulmonary edema has been developing gradually, **symptoms of fluid overload** may be elicited. They are:

- Nocturia
- Ankle edema (swelling of the legs, in general, of the "pitting" variety, wherein the skin is slow to return to normal when pressed upon)
- Orthopnea (inability to lie down flat due to breathlessness), and
- Paroxysmal nocturnal dyspnea (episodes of severe sudden breathlessness at night).

Distinguishing Cardiogenic (CPE) and Noncardiogenic Pulmonary Edema (NCPE) in terms of clinical features and diagnostic evaluation

Difference between clinical features of Distinguishing Cardiogenic (CPE) from **Noncardiogenic** Pulmonary Edema (NCPE) are as follows table 3.1:

Table 3.1: Distinguishing failures of CPE and NCPE

Cardiogenic pulmonary edema (CPE)	Non-Cardiogenic pulmonary edema (NCPE)
Early signs include exertional dyspnea and orthopnea	Ranges from mild dyspnea to respiratory failure
Evidence of increased intracardiac pressures (S3 gallop, elevated jugular venous pulse along with jugular venous distention, peripheral edema) Increasing airway edema is associated with rhonchi and wheezes on auscultation of the chest (Rales and wheezing is due to airway compression from peribronchial cuffing	Pulmonary findings may be relatively normal in the early stages Patients with NCPE have a warm periphery, a bounding pulse, and no S3 gallop or jugular venous distention.
Hypoxemia is due to ventilation-perfusion V/Q mismatch and responds to the administration of supplemental oxygen	Hypoxemia is due to intrapulmonary shunting and typically persists despite high concentrations of inhaled O_2.
The chest radiograph shows peribronchial thickening, prominent vascular markings in the upper lung zones, and Kerley B lines. Also an enlarged cardiac silhouette, interstitial thickening, and perihilar alveolar infiltrates (seen when pulmonary edema worsens) Pleural effusions are common	The chest radiograph shows normal heart size, patchy alveolar infiltrates with air bronchograms are more indicative of noncardiogenic edema. Pleural effusions are uncommon.
The pulmonary capillary wedge pressure (PCWP) is generally >18 mm Hg in CPE	<18 mm Hg in NCPE But superimposition of chronic pulmonary vascular disease can make this distinction difficult.

Common Diagnostic measures

These include following :

History

A thorough history should be taken about the occurrence, presenting symptoms, past history, presenting clinical features like shortness of breath, profuse diaphoresis, dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, Cough with the characteristic feature mainly the colour of the sputum (Pink, frothy sputum), chest pain.

Physical examination

- Physical findings include to identify patients,with tachypnea, tachycardia, diaphoretic, and hypertension.
- Auscultation of the lungs has to be done to look for adventitious sounds of lung and heart murmers.

Laboratory investigation

Blood tests are performed, which are as follows :

- Electrolytes like sodium, potassium
- Markers of renal function (creatinine, urea).
- Liver enzymes, inflammatory markers (usually C-reactive protein)
- Complete blood count
- Coagulation studies (PT, PTT) are typically requested.
- Arterial blood gas analysis : Arterial blood gas analysis is done to decide whether to start mechanical ventilation.
- Pulse oximetry : Pulse oximetry is useful in assessing hypoxia and, therefore, the severity of CPE. It is also useful for monitoring the patient's response to supplemental oxygenation and other therapies.
- Natriuretic peptide : Atrial natriuretic peptide (ANP) and b-type natriuretic peptide (BNP) are hormones produced by cardiomyocytes. They are produced in response to increased atrial pressure (increased volume and high serum sodium level. They suppress the secretion of aldosterone, renin and ADH, and the action of angiotensin II. When BNP levels are substantially elevated, support heart failure. They act on renal tubules to promote excretion of sodium and water, resulting in a decrease in blood volume and blood pressure.

Other diagnostic measures

- **Echocardiography:** Echocardiography may identify systolic and diastolic ventricular dysfunction and valvular lesions.
- In rare occasions, insertion of a Swan-Ganz catheter may be required to distinguish between the two main forms of pulmonary edema. The use of a Swan-Ganz catheter permits measurement of PCWP and helps differentiate high pressure (cardiogenic) and normal pressure (noncardiogenic) causes of pulmonary edema.
- **Electrocardiography:** Electrocardiography shows ST elevation and evolving Q waves is usually diagnostic of acute MI and should prompt immediate institution of MI protocols and coronary artery reperfusion therapy may be started accordingly.
- **Pulmonary artery catheterization** is indicated when the etiology of the pulmonary edema is uncertain, when it is refractory to therapy, or when it is accompanied by hypotension.

3.2.4 Medical Management and Nursing Management

An easy mnemonic for you to remember the management is given in Box 3.1 which is MOST DAMP.

Box 3.1

M- Morphine
O-Oxygen
S -Sitting up
T- Tourniquet
D- Diuretics
A – Aminophyline
M- Monitoring
P – Physiotherapy of chest

Emergency treatment:

- Initial management of patients with Cronic Pulmonary Edemia (CPE) should address the BAC of resuscitation, that is circulation airway and breathing.
- Oxygen should be administered to all patients to keep oxygen saturation >90%.
- The method of oxygen delivery varies from use of a face mask to bilevel noninvasive positive-pressure ventilation (NPPV) or continuous positive airway pressure (CPAP) or intubation and mechanical ventilation depending on presence of hypoxemia and acidosis and on the patient's level of consciousness.
- In case of persistent hypoxemia, acidosis or altered mental status, intubation and mechanical ventilation may become necessary.
- Any associated arrhythmia or myocardial infarction should be treated appropriately.

Goals of medical management are:

- **Reduction of pulmonary venous return (preload reduction) :** Preload reduction decreases pulmonary capillary hydrostatic pressure and reduces fluid transudation into the pulmonary interstitium and alveoli.
- **Reduction of systemic vascular resistance (afterload reduction) :** Afterload reduction increases cardiac output and improves renal perfusion, which allows for diuresis in the patient with fluid overload.
- **Inotropic support in some cases :** Patients with severe Left ventricular dysfunction or acute valvular disorders may present with hypotension. These patients may not tolerate medications to reduce their preload and afterload. Therefore, the third goal in this subset of patients is to provide inotropic support to maintain adequate BP.

- **Ventilator support:** Patients who remain hypoxic despite supplemental oxygenation and patients who have severe respiratory distress require ventilatory support in addition to maximal medical therapy.

Preload reduction

- **Diuretics:** The "loop diuretics" furosemide, bumetanide, and torsemide are effective in most forms of pulmonary edema, even in the presence of hypoalbuminemia, hyponatremia, or hypochloremia. Furosemide is also a venodilator that can reduce preload rapidly, prior to any diuresis, and is the diuretic of choice. The initial dose of furosemide should be 0.5 mg/kg, but a higher dose (1 mg/kg) is required in patients with renal insufficiency, chronic diuretic use, or hypervolemia or after failure of a lower dose.

- **Nitrates:** Nitroglycerin and isosorbide dinitrate act predominantly as vasodilators, with coronary vasodilating properties as well. They are rapid in onset and effective when administered by a variety of routes. Sublingual nitroglycerin (0.4 mg x 3 every 5 min) is first-line therapy for acute cardiogenic pulmonary edema. If pulmonary edema persists in the absence of hypotension, sublingual may be followed by IV nitroglycerin, commencing at 5–10 g/min. IV nitroprusside (0.1–5 g/kg per min) is a potent venous and arterial vasodilator. It is useful for patients with pulmonary edema and hypertension, but is not recommended in states of reduced coronary artery perfusion. It requires close monitoring and titration, including the use of an arterial catheter for continuous BP measurement in the intensive care unit.

- **Morphine:** Given in 2-to 4-mg IV boluses, morphine is a transient venodilator that reduces preload while relieving dyspnea and anxiety. These effects can diminish stress, catecholamine levels, tachycardia, and ventricular afterload in patients with pulmonary edema and systemic hypertension.

- **Other Preload-Reducing Agents:** IV recombinant brain natriuretic peptide (nesiritide): It is a potent vasodilator with diuretic properties and is effective in the treatment of cardiogenic pulmonary edema. It should be reserved for refractory patients and is not recommended in the setting of ischemia or MI. **Physical Methods :** Reduction of venous return reduces preload. Patients without hypotension should be maintained in the sitting position with the legs dangling along the side of the bed.

After load reduction

- **Angiotensin-Converting Enzyme (ACE) Inhibitors :** ACE inhibitors reduce both after load and preload.

- Nitroprusside : Nitroprusside results in simultaneous preload and afterload reduction by causing direct smooth-muscle relaxation, with an increased effect on afterload. Afterload reduction is associated with increased cardiac output.

- Its use is associated with shunting of blood away from ischemic myocardium toward healthy myocardium (i.e., coronary steal syndrome), which potentiates ischemia.

- If nitroprusside is used, convert therapy to oral or alternative IV vasodilator therapy as soon as possible because prolonged use is associated with thiocyanate toxicity.

Inotropics:

Inotropic support is usually used when preload- and afterload-reduction strategies are not successful or when hypotension precludes use of these strategies. **Two main classes of inotropic agents are available: Catecholamine agents and phosphodiesterase inhibitors (PDIs).**

Catecholamine agents

- **Dobutamine :** Dobutamine, a catecholamine agent, mainly serves as a beta1-receptor agonist, though it has some beta2-receptor and minimal alpha-receptor activity.
    - IV dobutamine induces significant positive inotropic effects with mild chronotropic effects. It also induces mild peripheral vasodilation (decrease in afterload).
    - In general, avoid dobutamine in patients with moderate or severe hypotension (e.g., systolic BP <80 mm Hg) because of the peripheral vasodilation.
- **Dopamine :** The vascular and myocardial receptor effects of dopamine, a catecholamine agent, are dose dependent.
    - Low dosages of 0.5-5 mcg/kg/min stimulate dopaminergic receptors in the renal and splanchnic vascular beds, causing vasodilation and increasing diuresis.
    - Moderate dosages of 5-10 mcg/kg/min stimulate beta-receptors in the myocardium, increasing cardiac contractility and heart rate.
    - High dosages of 15-20 mcg/kg/min stimulate alpha-receptors, resulting in peripheral vasoconstriction (increased afterload), increased BP, and no further improvement in cardiac output.
- **Norepinephrine:** Norepinephrine, a catecholamine agent, primarily stimulates alpha-receptors, significantly increasing afterload (and the potential for myocardial ischemia) and reducing cardiac output. It is generally used for patients with profound hypotension (e.g., systolic BP <60 mm Hg). Nor adrenaline stimulates á receptors in peripheral blood vessels and cause intense vasoconstriction. This increases blood pressure. The increase in BP increases afterload and thereby decreases cardiac output. But it will have positive effect on coronary blood flow due to increase in arterial pressure during diastole. After BP is restored, add other medications to maintain cardiac output.
- **Phosphodiesterase inhibitors (PDIs) :** PDIs increase the level of intracellular cyclic adenosine monophosphate by preventing the breakdown of *cAMP* to *5'AMP* and result in a positive inotropic effect on the myocardium, in peripheral vasodilation (decreased afterload) and in a reduction in pulmonary vascular resistance (decreased preload).

Ventilatory support

Noninvasive pressure-support ventilation

Noninvasive pressure-support ventilation (NPSV) may be considered early when treating patients with severe CPE.

In NPSV, the patient breathes through a face mask against a continuous flow of positive airway pressure. NPSV maintains the patency of the fluid-filled alveoli and prevents them from collapsing during exhalation. As a result, the patient saves the energy spent trying to reopen collapsed alveoli. NPSV improves pulmonary air exchange, and it increases intrathoracic pressure with reduction in preload and afterload.

Two types of NPSV are continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP). In CPAP, a single airway pressure is maintained throughout all phases of the respiratory cycle. In BiPAP, high pressures can be applied during inspiration and low pressures, during expiration, increasing the patient's comfort.

Mechanical ventilation

In general, use endotracheal intubation and mechanical ventilation when patients with CPE remain hypoxic despite maximal noninvasive supplemental oxygenation, when patients have evidence of impending respiratory failure (e.g., lethargy, fatigue, diaphoresis, worsening anxiety), or when the patient is hemodynamically unstable (e.g., hypotensive, severely tachycardic). Mechanical ventilation maximizes myocardial oxygen delivery and ventilation. Positive end-expiratory pressure is generally recommended to increase alveolar patency and to enhance oxygen delivery and carbon dioxide exchange.

Dietary management

Patients admitted with heart failure or pulmonary edema should be given a low-salt diet to minimize fluid retention. Closely monitor their fluid balance.

Other forms of Management

- **Intra-aortic balloon pumping**

The IABP decreases afterload as the pump deflates, and it inflates during diastole to improve coronary blood flow.

Ultrafiltration

Ultrafiltration (UF) is a method of fluid removal that is particularly useful in patients with renal dysfunction and expected diuretic resistance.

Check Your Progress 1

i) Explain the meaning of pulmonary edema.

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ii) List three causes of non cardiogenic pulmonary edema.

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iii) List the cause of cardiogenic pulmonary edema.

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iv) Differentiate between cardiogenic pulmonary edema and non cardiogenic pulmonary edema.

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v) List five points of emergency management of Pulmonary Edema.

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Nursing management

Assessment

- Assessment of cardio-vascular system is very essential. A thorough history with physical examination has to be done. The assessment includes the following :
  - Assess for shortness of breath and profuse diaphoresis, dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea, Cough (Pink, frothy sputum)
  - Assess for chest pain, if present suspect the possibility of acute myocardial ischemia/infarction, or aortic dissection with acute aortic regurgitation as the precipitant of pulmonary edema.

- Assess for tachypnea and tachycardia.
- Auscultation of the lungs for fine crepitant rales, rhonchi or wheeze
- Assess for Cardiovascular findings like S_3, accentuation of pulmonic component of S_2 and jugular venous distension and auscultation of murmurs
- Assess the laboratory values.
- Assess the chest x-ray and look for abnormalities.

Nursing diagnoses

1) Impaired gas exchange related to increased preload secondary to accumulation of fluid in the alveoli as evidenced by increased respiratory rate, shortness of breath, dyspnea on exertion and low saturation in ABG.

   Objective : The client achieves and maintains normal gas exchange as evidenced by improved ABG values, normal respiratory rate, improved oxygen saturation and absence of dyspnea.

   Interventions

   - Assess the respiratory rate, rhythm and effort of respirations of the patient
   - Auscultate for breath sounds for adventitious sounds
   - Monitor for dyspnea
   - Place the patient in semi-fowler's position to increase the thoracic capacity
   - Administer oxygen via mask as ordered
   - Monitor oxygen saturation
   - Perform ABG analysis and do the necessary corrections.

2) Fluid volume excess related to fluid shift secondary to increased hydrostatic pressure or decreased oncotic pressure as evidenced by edema, increased weight gain.

   Objective: The client achieves and maintains normal fluid volume as evidenced by absence or reduction in edema, palpable peripheral pulses and reduction in weight.

   Interventions

   - Assess the weight of the patient, abdominal girth, the nature of edema daily
   - Monitor hemodynamic status including CVP, PAWP to evaluate effectiveness of therapy (refer practical unit on hemodynamic monitoring) block 3 unit 6 course 1
   - Maintain intake output chart. A negative balance is desired
   - Restrict fluid according to the ejection fraction
   - Administer diuretics as prescribed
   - Explain the importance of sodium restricted diet

3) Activity intolerance related to fatigue secondary to cardiac insufficiency and pulmonary congestion as evidenced by easy tiredness with mild activities, weakness.

Objective: The client will achieve activity level as much as tolerable as evidenced by ability to do mild activities.

Interventions

- Assess the activity level of the patient
- Encourage alternate rest and activity periods to reduce cardiac workload
- Provide calming divertional activities to promote relaxation to reduce oxygen consumption and to relieve dyspnea and fatigue
- Teach active and passive exercises as tolerated by the patient
- Encourage mild activities by self
- Advise to avoid heavy or strenuous activities
- Appreciate when able to do activities by self
- Advise to take assistance whenever required and provide calling bell to patient with appropriate instructions.

4) Anxiety related to dyspnea or perceived threat of death as evidenced by restlessness, irritability, expression of feelings of life threat.

Objective: The patient achieves reduction in anxiety as evidenced by verbalization.

Interventions

- Use a calm, reassuring approach to relieve anxiety
- Explain all procedures including sensations experienced during procedure to promote feeling of security
- Allow patient to verbalize
- Clear all the doubts
- Introduce to patients with similar problem who has recovered successfully
- Teach relaxation techniques

Check Your Progress 2

i) Outline any two objective based nursing interventions of pulmonary edema.

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3.3 PULMONARY EMBOLISM

Now lets turn our attention to pulmonary embolism. We shall begin with concept and meaning then focus on other aspects.

3.3.1 Concept and Definition

Pulmonary embolism (PE) is a common and potentially lethal condition that can cause death in all age groups. Prompt diagnosis and treatment can dramatically reduce the morbidity and mortality of the disease. Unfortunately, the diagnosis is often missed, because pulmonary embolism frequently causes only vague and nonspecific symptoms.

Pulmonary embolism (PE) is a blockage of the pulmonary artery or one of its branches, usually occurring when a deep vein thrombus (blood clot from a vein) becomes dislodged from its site of formation and travels, or embolizes, to the arterial blood supply of one of the lungs. This process is termed as thromboembolism.

3.3.2 Risk factors and Causes

The most common sources of embolism are proximal leg deep venous thrombosis (DVTs) or pelvic vein thromboses. Any risk factor for DVT also increases the risk that the venous clot will dislodge and migrate to the lung circulation, which happens in up to 15% of all DVTs. The conditions are generally regarded as a continuum termed venous thromboembolism (VTE).

The development of thrombosis is classically due to a group of pre-disposing factors named as **Virchow's triad. They are hypercoagulability, injury to vascular endothelium, and venous stasis,** among which endothelial injury appears to be the most significant.

Embolization of a deep venous thrombosis to the pulmonary artery or its branches is by far the most common cause of pulmonary embolism. The etiology of venous thrombosis and subsequent thromboembolism results from a distortion in Virchow's triad by venostasis, hypercoagulability, or vessel wall inflammation. Hereditary factors (most result in a hypercoagulable state).

There risk factors for venous thrombosis and pulmonary embolism can be broken down into hereditary factors and acquired factors.

Risk factor

Injury to vascular endothelium may be triggered by local vessel injury, infection, incision atherosclerosis.

- Antithrombin III deficiency
- Protein C deficiency
- Protein S deficiency
- Factor V Leiden (most common genetic risk factor for thrombophilia)
- Plasminogen abnormality

- Plasminogen activator abnormality
- Fibrinogen abnormality
- Resistance to activated protein C.

Acquired factors

The most important clinically identifiable risk factors for DVT and PE are a prior history of DVT or PE, recent surgery or pregnancy, prolonged immobilization or underlying malignancy.

Reduced mobility

- Fractures (fat)
- Immobilization
- Burns
- Obesity

- Old age
- Malignancy of ovary, pancreas, stomach extrahepatic Bile duct system
  - Tumor cells
  - Chemotherapy
- Acute medical illness
  - AIDS (lupus anticoagulant)
  - Behçet disease
  - Congestive heart failure (CHF)
  - Myocardial infarction
  - Cardiomyopathy
  - Polycythemia
  - Systemic lupus erythematosus pulmonae
  - Ulcerative colitis
- **Trauma/major surgery**
  - Spinal cord injury / orthopedic surgery
  - Catheters (indwelling venous infusion catheters) / vascular surgery
  - Postoperative
- **Pregnancy**
  - Postpartum period
  - Oral contraceptives
  - Estrogen replacements (high dose only)

- Amniotic fluid
- Drug abuse (intravenous IV drugs)
- Drug-induced lupus anticoagulant
- Hemolytic anemias
- Heparin-associated thrombocytopenia
- Homocysteinemia
- Homocystinuria
- Hyperlipidemias
- Phenothiazines
- Thrombocytosis
- Varicose veins
- Venography
- Venous pacemakers
- Venous stasis may be triggered by atrial fibrillation decreased cardiac output immobility
- Warfarin (first few days of therapy).

3.3.3 Pathophysiological Changes

Pulmonary embolism arises from anywhere in the body, most commonly it arises from the calf veins. The venous thrombi predominately originate in venous valve pockets (inset) and at other sites of presumed venous stasis. To reach the lungs, thromboemboli travel through the right side of the heart. right atrium (RA), right ventricle (RV), left atrium (LA); left ventricle (LV).

Pulmonary thromboembolism is not a disease in and of itself. Rather, it is a complication of underlying venous thrombosis. Under normal conditions, microthrombi (tiny aggregates of red cells, platelets, and fibrin) are formed and lysed continually within the venous circulatory system. This dynamic equilibrium ensures local hemostasis in response to injury without permitting uncontrolled propagation of clot. Under pathological conditions, microthrombi may escape the normal fibrinolytic system to grow and propagate. Pulmonary embolism (PE) occurs when these propagating clots break loose and embolize to block pulmonary blood vessels.

Patients who have undergone gynecologic surgery, those with major trauma, and those with indwelling venous catheters may have DVTs that start in an area related to their pathology. For other patients, venous thrombosis most often involves the lower extremities and nearly always starts in the calf veins, which are involved in virtually all cases of symptomatic spontaneous lower extremity DVT. Although DVT starts in the calf veins, in cases of pulmonary embolism, it will usually propagate proximally to the popliteal vessels, and from that area embolize. See flow chart Fig.3.1 to understand pathological charge.

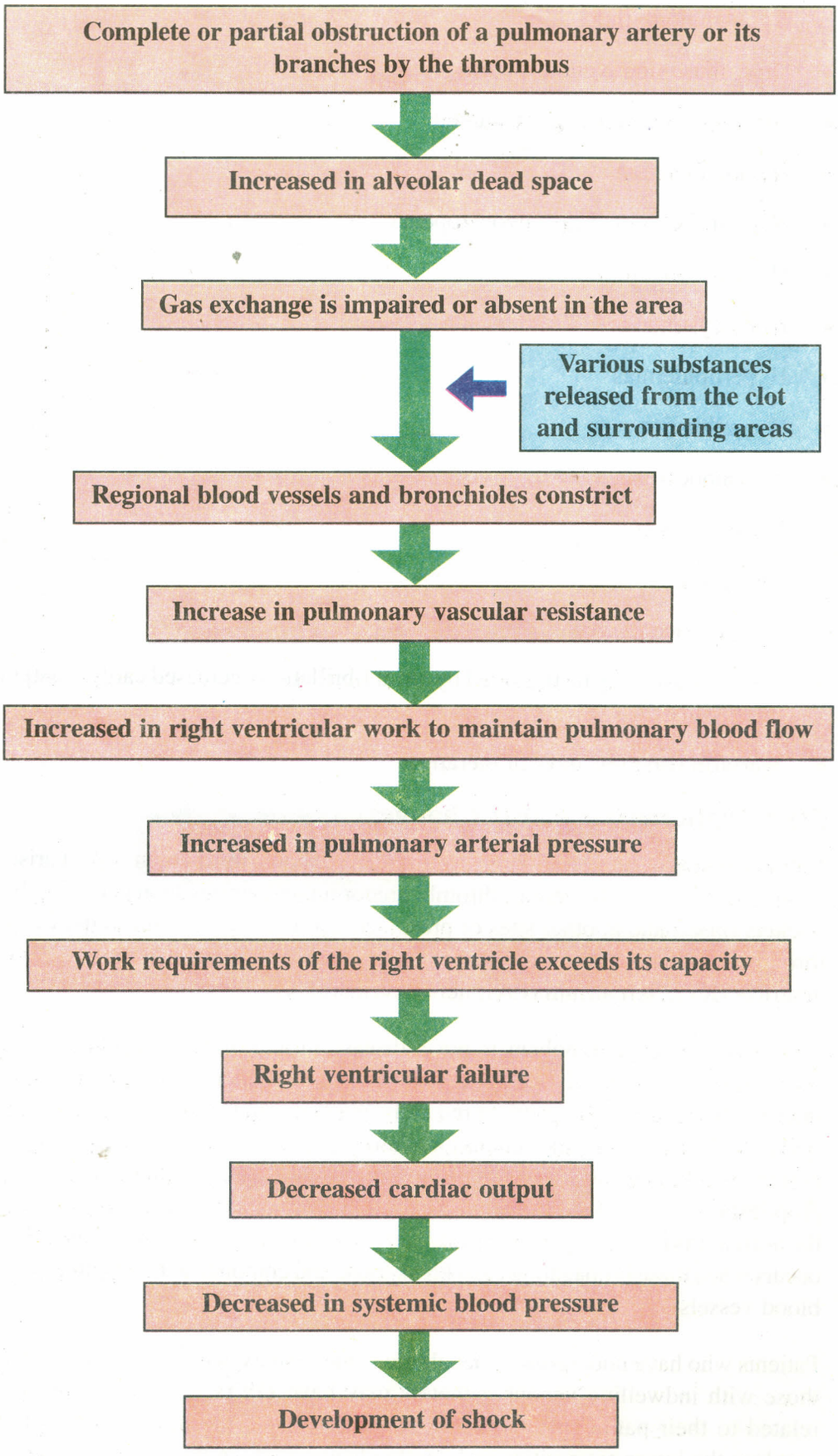

Fig. 3.1: Flow chart showing pathophysiology of pulmonary embolism

3.3.4 Signs, Symptoms and Diagnosis

Symptoms of pulmonary edema (PE) have sudden-onset. These include dyspnea (shortness of breath), tachypnea (rapid breathing), chest pain of a "pleuritic" nature (worsened by breathing), cough, hemoptysis (coughing up blood), which may aid in the diagnosis on more severe cases signs such as pleural rub, cyanosis (blue discoloration, usually of the lips and fingers), collapse, and circulatory instability can occur. About 15% of all cases of sudden death are attributable to PE.

The decision to do medical imaging is usually based on clinical grounds, i.e., the medical history, symptoms and findings on physical examination. The most commonly used method to predict clinical probability is the **Wells score**; it is a clinical prediction rule as is explained below:

The Wells score:

- clinically suspected DVT - 3.0 points
- alternative diagnosis is less likely than PE - 3.0 points
- tachycardia - 1.5 points
- immobilization/surgery in previous four weeks - 1.5 points
- history of DVT or PE - 1.5 points
- hemoptysis - 1.0 points
- malignancy (treatment for within 6 months, palliative) - 1.0 points

Traditional interpretation:

- Score >6.0 - High (probability 59% based on pooled data)
- Score 2.0 to 6.0 - Moderate (probability 29% based on pooled data)
- Score <2.0 - Low (probability 15% based on pooled data)

Alternate interpretation

- Score > 4 - PE likely. Consider diagnostic imaging.
- Score 4 or less - PE unlikely. Consider D-dimer to rule out PE.

Since multiple variations of well score are available it is difficulty to use.

Blood tests

In low/moderate suspicion of PE, a normal D-dimer level (shown in a blood test) is enough to exclude the possibility of thrombotic PE.

When a PE is being suspected, a number of blood tests are done, in order to exclude important secondary causes of PE. This includes a full blood count, clotting status (PT, APTT, TT), and some screening tests (erythrocyte sedimentation rate, renal function, liver enzymes, electrolytes). If one of these is abnormal, further investigations might be warranted.

Imaging

Selective pulmonary angiogram revealing significant thrombus (labelled A) causing a central obstruction in the left main pulmonary artery. ECG tracing shown in following (fig 3.2).

Fig. 3.2: Pulmonary angiography

The gold standard for diagnosing pulmonary embolism (PE) is pulmonary angiography. Pulmonary angiography is used less often due to wider acceptance of CT scans, which are non-invasive (fig 3.3).

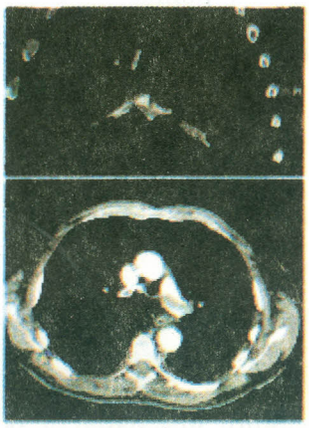

Fig. 3.3: Pulmonary CT scan

CT pulmonary angiography (CTPA) showing a saddle embolus and substantial thrombus burden in the lobar branches of both main pulmonary arteries.

Non-invasive imaging

CT pulmonary angiography (CTPA) is a pulmonary angiogram obtained using computed tomography (CT) with radio-contrast rather than right heart catheterization. Its advantages are clinical equivalence, its non-invasive nature, its greater availability to patients, and the possibility of identifying other lung disorders from the differential diagnosis in case there is no pulmonary embolism.

Tests that are frequently done that are not sensitive for PE, but can be diagnostic are:

- **Chest X-rays** are often done on patients with shortness of breath to help rule-out other causes, such as congestive heart failure and rib fracture. Chest X-rays in PE are rarely normal, but usually lack signs that suggest the diagnosis of PE (e.g., Westermark sign, Hampton's hump).
- **Ultrasonography** of the legs, also known as leg doppler, in search of deep venous thrombosis (DVT). The presence of DVT, as shown on ultrasonography of the legs, is in itself enough to warrant anticoagulation, without requiring the V/Q or spiral CT scans (because of the strong association between DVT and PE). Scanning may be required if the mother is deemed at high risk of having pulmonary embolism.

Electrocardiogram findings

Electrocardiogram of a patient with pulmonary embolism (PE) showing sinus tachycardia of approximately 150 beats per minute and right bundle branch block. The most commonly seen signs in the ECG is sinus tachycardia, right axis deviation and right bundle branch block. atrial fibrillation, ST segment changes T Wave inversion in anterior and inferior leads. The classic ECG changes in PE are S wave in lead T wave Q wave and inverted T in lead III (Fig 3.4).

It provides visualization of any emboli in central Pulmonary artery and hmodynamic effects of PE on right side of heart such as severe obstruction of pulmonary artery and pressure changes.

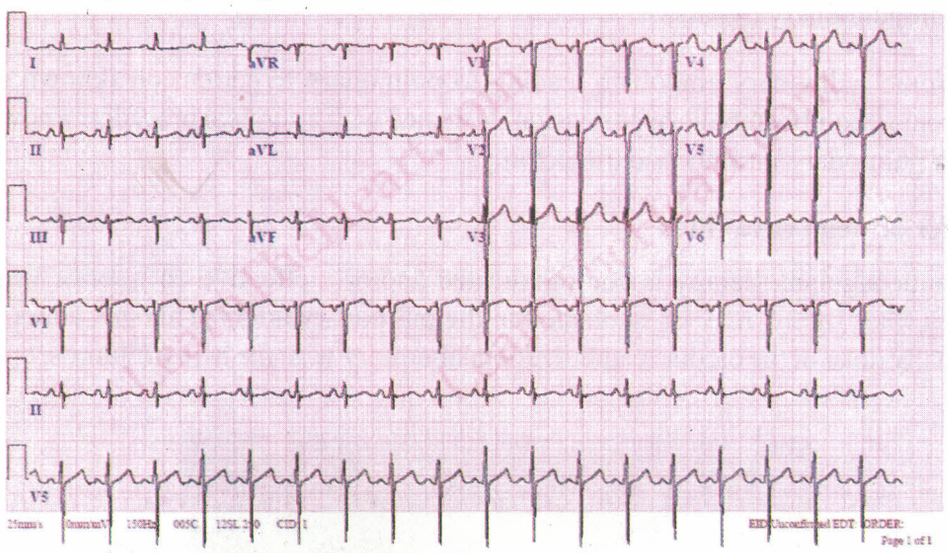

Fig. 3.4: ECG of client with pulmonary embolism

3.3.5 Medical Management and Nursing Management

In most cases, anticoagulant therapy is used as prevention of recurrence. Acutely, supportive treatments, such as oxygen or analgesia, are often required.

Prevention of Recurrence with Anticoagulation

In most cases, anticoagulant therapy is the mainstay of treatment. **Heparin;** low molecular weight heparins (such as enoxaparin and dalteparin), or fondaparinux is administered initially, while **warfarin therapy** is commenced to prevent further clots. These have no effect on existing clots. Heprin should be stopped when **international** ratio reaches 3.0. Warfarin therapy often requires frequent dose adjustment and monitoring of the INR. In PE, INRs between 2.0 and 3.0 are generally considered ideal. Heparin should be adjusted to maintain activated partial thromboplastion time (APTT) in range of 1.5 – 2.3 times **control**.

Warfarin therapy is usually continued for 3 – 6 months, or "lifelong" if there have been previous DVTs or PEs, or none of the usual risk factors is present. An abnormal D-dimer level at the end of treatment might signal the need for continued treatment among patients with a first unprovoked pulmonary embolus.

Thrombolysis

Massive PE causing hemodynamic instability (shock and/or hypotension, defined as a systolic blood pressure <90 mmHg or a pressure drop of 40 mmHg for>15

min if not caused by new-onset arrhythmia, hypovolemia or sepsis) is an indication for thrombolysis, the enzymatic destruction of the clot with streptokinase or **recombinant tissue** type plasminogen activator (rt-pa). The aim of the therapy is to dissolve the clot, but there is an **attendant** risk of bleeding or stroke. The main indication for thrombolysis is in submassive PE where right ventricular dysfunction can be demonstrated on echocardiography.

Surgical management

Surgical management of acute pulmonary embolism i.e., pulmonary embolectomy is done as last resort through an open procedure usually while patient is on cardiopulmonary bypass.

Chronic pulmonary embolism leading to pulmonary hypertension (known as chronic thromboembolic hypertension) is treated with a surgical procedure known as a pulmonary thromboendarterectomy.

Inferior vena cava filter

If anticoagulant therapy is contraindicated and/or ineffective an inferior vena cava filter fig 3.5 may be implanted in the pulmonary artery. This procedure is usually done in the cardiac cathetrization laboratory with the help of fluoroscope.

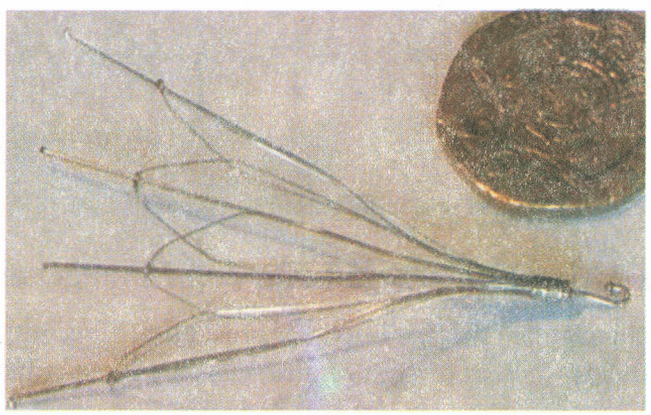

Fig.3.5: Inferior vena cava filter

Role of nurse in taking care of a client with Pulmonary Embolism:

- **Minimizing the risk of pulmonary embolism:** The key role of the nurse is to identify patients at high risk for PE and to minimize the risk of PE in all patients. The nurse must have a high degree of suspicion for PE in any patient, but particularly in those with conditions predisposing to a slowing of venous return.

- **Preventing thrombus formation:** Preventing thrombus formation is one of the major nursing responsibilities. The nurse encourages ambulation and active and passive leg exercises to prevent venous stasis in patients on bed rest. The nurse instructs the patient to move the legs in a "pumping" exercise so that the leg muscle can help increase blood flow. The patient should be instructed not to lie or sit in bed for prolonged periods, not to cross the legs and not to wear constricting clothing. Legs should not be dangled or feet placed in a dependent position while the patient sits on the edge of the bed; instead, catheters should not be left in place for prolonged periods (fig.3.6).

Fig. 3.6: Assessing Potential for Pulmonary Embolism

- **Assessing potential for pulmonary embolism:** The nurse examines patient who are at risk of developing PE for a positive Homans sign, the patient assumes a supine position, lifts the leg, and dorsiflexes the foot. The nurse asks the patient to report whether calf pain occurs during this manoeuver. The occurrence of pain – a positive Homan's sign - may indicate deep venous thrombosis. (Fig.3.7).

Fig. 3.7: Monitoring Thrombolytic Therapy

- **Monitoring thrombolytic therapy:** The nurse is responsible for monitoring the thrombolytic and anticoagulant therapy. Thrombolytic therapy (streptokinase Urokinase, tissue plasminogen activator) causes lysis of deep vein thrombi and pulmonary emboli, which help dissolve the clots. During the thrombolytic infusion, the patient remains on bed rest, vital signs are assessed every 2 hourly and invasive procedures are limited. Tests to determine prothrombin time or partial thromboplastin time are performed 3-4 hours after the thrombolytic infusion is started to confirm the fibrinolytic systems have been activated. Because of the prolonged clotting time, only essential arterial punctures or venipunctures are performed, and manual pressure is applied to any puncture site for at least 30 minutes. Pulse oximetry

is used to monitor changes in oxygenation. The nurse immediately discontinues the infusion if uncontrolled bleeding occurs fig.3.8

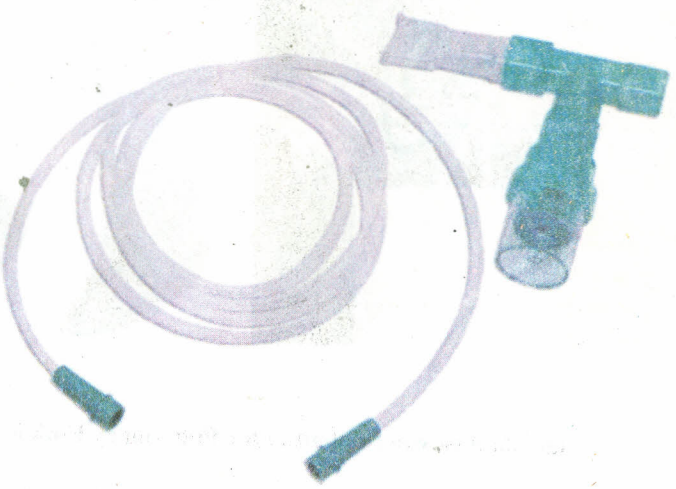

Fig. 3.8: Managing Oxygen Therapy

- **Managing pain:** Chest pain if present is usually pleuritic rather than cardiac in origin. A semi fowler's position provides a more comfortable position for breathing. However, it is important to continue to turn the patient frequently and reposition the patient to improve the ventilation-perfusion ratio in the lung. The nurse administers opiod analgesics as prescribed for severe pain. (fig.3.9 managing pain).

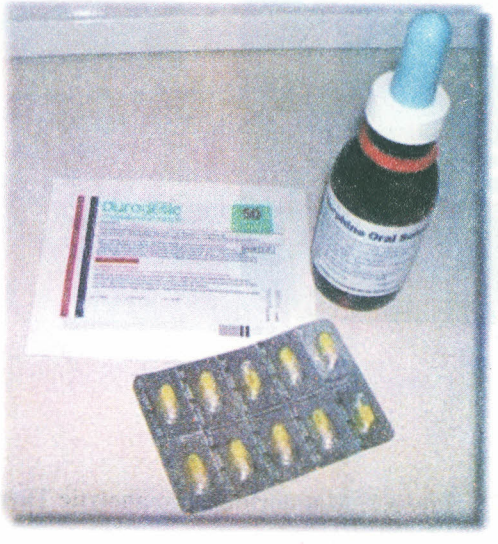

Fig. 3.9: Managing Pain

- **Managing oxygen therapy:** Careful attention is given to proper use of oxygen. It is important to ensure that the patient understands the need for continuous oxygen therapy. The nurse assesses the patient frequently for signs of hypoxemia and monitors the pulse oximetry values to evaluate the effectiveness of the oxygen therapy. Deep breathing and incentive spirometry are indicated for all patients to minimize or prevent atelectasis and improve ventilation. Nebulizer therapy or percussion and postural drainage may be used for management of secretions. (3.7 Managing Oxygen therapy)

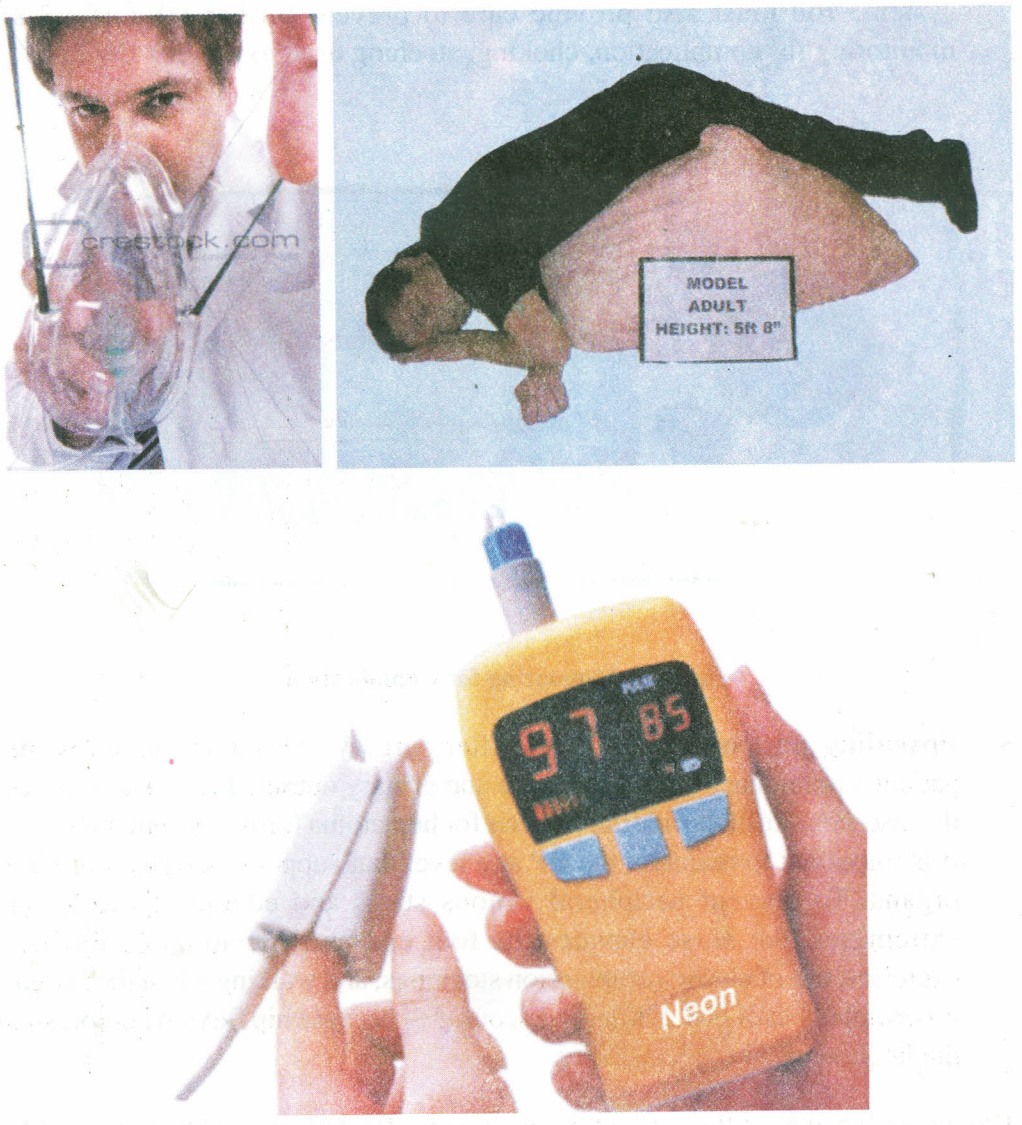

Fig. 3.10: A Managing Oxygen Therapy

- **Relieving anxiety:** The nurse encourages the stabilized patient to talk about any fears or concerns related to this frightening episode, answers the patient's and family's questions concisely and accurately, explains the therapy, and describe how to recognize untoward effects early.

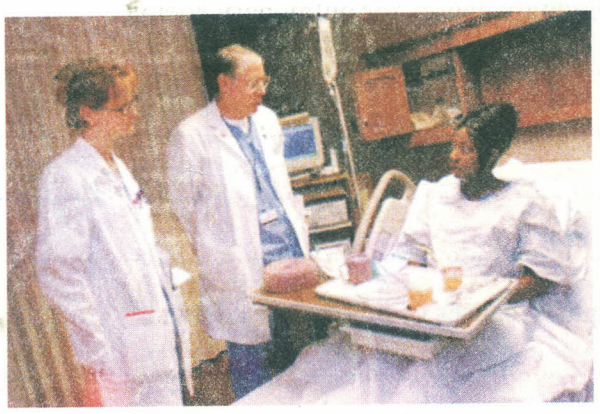

Fig. 3.11: Relieving Anxiety

- **Monitoring for complications:** When caring for a patient who has had PE, you must be alert for the potential complication of cardiogenic shock or right ventricular failure subsequently to the effect of PE on the cardiovascular

system. You must also provide care to prevent further DVT (fig.3.8) monitoring for complication, choking, stoching used to prevent DVT.

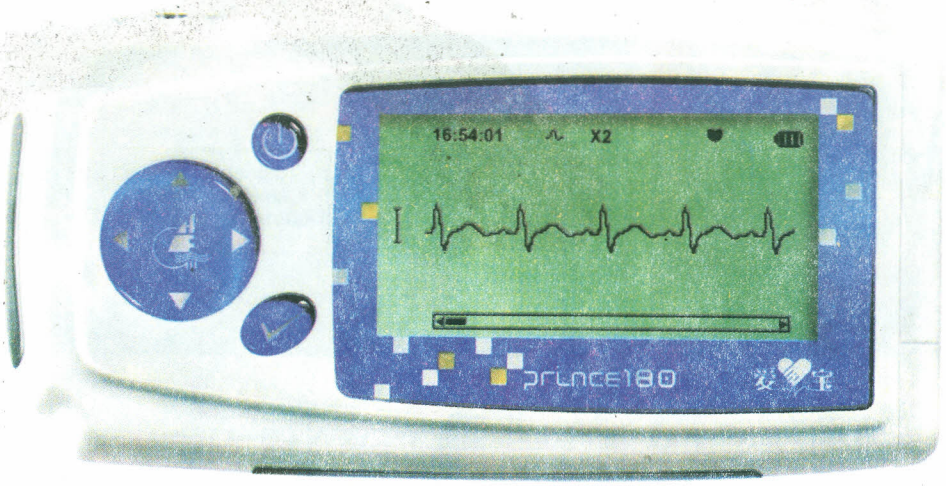

Fig. 3.12: Monitoring for Complication

- **Providing post operative care:** After surgery, the nurse measures the patient's pulmonary arterial pressure and urinary output. The nurse assesses the insertion site of the arterial catheter for hematoma formation and infection. It is important to maintain the BP at a level that supports perfusion of vital organs. To prevent peripheral venous stasis and edema of the lower extremities, the nurse elevates the foot end and encourages isometric exercises, use of elastic compression stockings, and walking when the patient is permitted out of bed. Sitting is discouraged because hip flexion compresses the large veins in the leg.

Prognosis depends on the amount of lung that is affected and on the co-existence of other medical conditions; chronic embolisation to the lung can lead to pulmonary hypertension. There is controversy over whether or not small subsegmental PEs need to be treated at all and some evidence exists that patients with subsegmental PEs may do well without treatment.

Check Your Progress 3

i) List five hereditary causes for pulmonary edema.

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ii) List 10 acquired causes of pulmonary edema.

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iii) What surgical method is used to treat Pulmonary embolism?

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iv) State 5 important roles of the nurse while taking care of a client with Pulmonary embolism..

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3.4 ATELECTASIS

We shall discuss about atelectasis in following subsections.

3.4.1 Concept and Definition

Atelectasis is a medical condition in which the lungs are not fully inflated. It may affect part or all of one lung. It is a condition where the alveoli are deflated, as distinct from pulmonary consolidation.

It is a very common finding in chest x-rays and other radiological studies. It may be caused by normal exhalation or by several medical conditions. Although frequently described as a collapse of lung tissue, atelectasis is not synonymous with a collapsed lung, which is a more specific condition that features atelectasis. Acute atelectasis may occur as a post-operative complication or as a result of surfactant deficiency. In premature neonates, this leads to infant respiratory distress syndrome.

Atelectasis is a collapse of lung tissue affecting part or all of one lung. This condition prevents normal oxygen absorption to healthy tissues.

3.4.2 Causes and Classification

The most common cause is post-surgical atelectasis, characterized by splinting, restricted breathing after abdominal surgery.

- Smokers and the elderly are at an increased risk.
- Atelectasis implies some blockage of a bronchiole or bronchus, which can be within the airway (foreign body, mucus plug), from the wall (tumor, usually squamous cell carcinoma) or compressing from the outside (tumor, lymph node, tubercle).

- Poor surfactant spreading during inspiration, causing an increase in surface tension which tends to collapse smaller alveoli.
- Atelectasis may also occur during suction, as along with sputum, air is withdrawn from the lungs. There are several types of atelectasis according to their underlying mechanisms or the distribution of alveolar collapse; resorption, compression, microatelectasis and contraction atelectasis.
- Pulmonary embolism (PE) another cause of Atelectasis.

Classification

Atelectasis may be an acute or chronic condition. In acute atelectasis, the lung has recently collapsed and is primarily notable only for airlessness. In chronic atelectasis, the affected area is often characterized by a complex mixture of airlessness, infection, widening of the bronchi (bronchiectasis), destruction, and scarring (fibrosis).

Acute Atelectasis

Acute atelectasis is a common postoperative complication, especially after chest or abdominal surgery. Acute atelectasis may also occur with an injury, usually to the chest (such as that caused by a car accident, a fall, or a stabbing). Atelectasis following surgery or injury, sometimes described as massive, involves most alveoli in one or more regions of the lungs. In these circumstances, the degree of collapse among alveoli tends to be quite consistent and complete. Large doses of opioids or sedatives, tight bandages, chest or abdominal pain, abdominal swelling (distention), and immobility of the body increase the risk of acute atelectasis following surgery or injury, or even spontaneously.

In acute atelectasis that occurs because of a deficiency in the amount or effectiveness of surfactant, many but not all alveoli collapse, and the degree of collapse is not uniform. Atelectasis in these circumstances may be limited to only a portion of one lung, or it may be present throughout both lungs. When premature babies are born with surfactant deficiency, they always develop acute atelectasis that progresses to neonatal respiratory distress syndrome. Adults can also develop acute atelectasis from excessive oxygen therapy and from mechanical ventilation.

Chronic Atelectasis

Chronic atelectasis may take one of two forms — middle lobe syndrome or rounded atelectasis. In middle lobe syndrome, the middle lobe of the right lung contracts, usually because of pressure on the bronchus from enlarged lymph glands and occasionally a tumor. The blocked, contracted lung may develop pneumonia that fails to resolve completely and leads to chronic inflammation, scarring, and bronchiectasis.

In rounded atelectasis (folded lung syndrome), an outer portion of the lung slowly collapses as a result of scarring and shrinkage of the membrane layers covering the lungs (pleura). This produces a rounded appearance on x-ray that doctors may mistake for a tumor. Rounded atelectasis is usually a complication of asbestos-induced disease of the pleura, but it may also result from other types of chronic scarring and thickening of the pleura.

Absorption Atelectasis

The atmosphere is composed of 78% nitrogen and 21% oxygen. Since oxygen is exchanged at the alveoli-capillary membrane, nitrogen is a major component for the alveoli's state of inflation. If a large volume of nitrogen in the lungs is replaced with oxygen, the oxygen may subsequently be absorbed into the blood reducing the volume of the alveoli, resulting in a form of alveolar collapse known as absorption atelectasis.

3.4.3 Signs, Symptoms and Diagnosis

- Cough, but not prominent
- Chest Pain
- Breathing Difficulty
- Low oxygen saturation
- Fever — debatable; no evidence to support this, although it is widely accepted
- Pleural Effusion (transudate type)
- Cyanosis (late sign)
- Increased heart rate

Diagnosis

- Chest X-ray
- Computed tomography
- Bronchoscopy

3.4.4 Medical Management and Nursing Management Treatment

Treatment is directed at correcting the underlying cause.

- Post-surgical atelectasis is treated by physiotherapy, focusing on deep breathing and encouraging coughing.
- An incentive spirometer is often used as part of the breathing exercises.
- Ambulation is also highly encouraged to improve lung inflation.
- People with chest deformities or neurologic conditions that cause shallow breathing for long periods may benefit from mechanical devices that assist their breathing. One method is continuous positive airway pressure, which delivers pressurized air or oxygen through a nose or face mask to help ensure that the alveoli do not collapse, even at the end of a breath. This is helpful, as partially-inflated alveoli can be expanded more easily than collapsed alveoli. Sometimes additional respiratory support is needed with a mechanical ventilator.
- The primary treatment for acute massive atelectasis is correction of the underlying cause. A blockage that cannot be removed by coughing or by suctioning the airways often can be removed by bronchoscopy.
- Antibiotics are given for an infection. Chronic atelectasis often is treated with antibiotics because infection is almost inevitable. In certain cases, the affected part of the lung may be surgically removed when recurring or chronic infections become disabling or bleeding is significant.

- If a tumor is blocking the airway, relieving the obstruction by surgery, radiation therapy, chemotherapy, or laser therapy may prevent atelectasis from progressing and recurrent obstructive pneumonia from developing.
- An incentive spirometer can also be used to prevent or help treat atelectasis after surgery.

3.4.5 Prevention of Complications

- Pneumonia may develop rapidly after atelectasis.
- Massive atelectasis may result in the complete collapse of a lung.

Prevention

- Encourage movement and deep breathing in anyone who is bedridden for long periods.
- Keep small objects out of the reach of young children.
- Maintain deep breathing after anesthesia.

Check Your Progress 4

i) List five important causes of atelectasis.

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ii) Explain the 3 types of atelectasis.

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iii) List 3 preventive measures.

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3.5 LET US SUM UP

It this unit you have studied pulmonary edema, pulomonary embolism and atetectasis. Admission to hospital for pulmonary edema is associated with significant morbidity and mortality. A high proportion of patients are at risk of

dying or a re-admission after one admission for pulmonary edema. If the underlying cause is found, appropriate treatment is started accordingly. The prognosis of course varies with severity. Pulmonary embolism is a life threatening condition and many a times person go into a stage of shock before diagnosing itself. So prevention of this problem should be given primary importance and should be considered in the clinical practice. Atectasis is a condition where lung tissue collapse which prevents normal oxygen absorbing by healthy tissues.

To minimizing the risk of pulmonary embolism you should:

a) Identify the high risk clients

b) Preventing Thrombus Formation.

3.6 GLOSSARY

Aspiration : The entry of secretions or foreign material into the trachea and lungs

Smoke inhalation : Smoke inhalation occurs when you breathe in the products of combustion during a fire. Combustion results from the rapid breakdown of a substance by heat (more commonly called burning). Smoke is a mixture of heated particles and gases.

Oxygen toxicity : Oxygen toxicity is a condition resulting from the harmful effects of breathing molecular oxygen (O_2) at elevated partial pressures.

Sepsis : Sepsis is a serious medical condition characterized by a whole-body inflammatory state (called a systemic inflammatory response syndrome or SIRS) and the presence of a known or suspected infection.

DVT : Deep vein thrombosis is a blood clot that forms in a vein deep in the body. Blood clots occur when the blood thickens and clumps together. Most deep vein blood clots occur in the lower leg or thigh. They also can occur in the other parts of the body.

Polycythemia : It is a condition in which there is an increase in the proportion of blood volume that is occupied by red blood cells, which is measured as hematocrit level. It can be due to an increase in the mass of the red blood cells (absolute polycythemia) or to a decrease in the volume of plasma (relative polycythemia).

SLE : It is an auto immune disease characterized by acute and chronic inflammation of the various tissues of the body. Anautoimmune diseases are illness that occur when the body's tissues are attacked by its own immune system. Patients with Lupus produces abnormal antibodies in their blood that target tissues within their own body rather than foreign infectious agents.

Consolidation : Consolidation is a term for solidification into a firm dense mass. It is more markedly defined as an area of the lung that, while previously collapsible, is now filled with a fluid. It is usually applied to a condition marked by induration (swelling or hardening of normally soft tissue) of a normally aerated lung through accumulation of inflammatory cellular exudate in the alveoli and adjoining ducts. Simply, it is defined as alveolar space that now contains the fluid instead of air. Fluid can be pulmonary edema, inflammatory exudate, pus, inhaled water, or blood (from bronchial tree or haemorrhage from a pulmonary artery). It is clinically important in pneumonia: the signs of lobar pneumonia are characteristic and clinically referred to as consolidation.

Surfactants : Surfactants are wetting agents that lower the surface tension of a liquid, allowing easier spreading, and lower the interfacial tension between two liquids. On the other hand The term surfactant is a blend of surface acting agent. Surfactants are usually organic compounds that are amphiphilic, meaning they contain both hydrophobic groups (their "tails") and hydrophilic groups (their "heads"). Therefore, they are soluble in both organic solvents and water. The term surfactant was coined by Antara products in 1950.

Infant respiratory distress syndrome (IRDS) : Also called neonatal respiratory distress syndrome or respiratory distress syndrome of newborn, previously called hyaline membrane disease, is a syndrome caused in premature infants by developmental insufficiency of surfactant production and structural immaturity in the lungs. It can also result from a genetic problem with the production of surfactant associated proteins. RDS affects about 1% of newborn infants and is the leading cause of death in preterm infants.

Bronchiectasis : It is a disease that causes localized, irreversible dilation of part of the bronchial tree. It is classified as an obstructive lung disease, along with bronchitis and cystic fibrosis. Involved bronchi are dilated, inflamed, and easily collapsible, resulting in airflow obstruction and impaired clearance of secretions. Bronchiectasis is associated with a wide range of disorders, but it usually results from necrotizing bacterial infections, such as infections caused by the Staphylococcus or Klebsiella species or Bordetella pertussis.

Oxygen saturation or Dissolved oxygen : It is a relative measure of the amount of oxygen that is dissolved or carried in a given medium. It can be measured with a dissolved oxygen probe such as an oxygen sensor or an optode in liquid media, usually water.

On the other hand oxygen saturation (S_{O2}), commonly abbreviated as "sats", measures the percentage of hemoglobin binding sites in the bloodstream occupied by oxygen. At low partial pressures of oxygen, most hemoglobin is deoxygenated. At around 90% (the value varies according to the clinical context) oxygen saturation increases according to an oxygen-hemoglobin dissociation curve and approaches 100% at partial oxygen pressures of >10 kPa. A pulse oximeter relies on the light absorption characteristics of saturated hemoglobin to give an indication of oxygen saturation. A Sa_{O2} (arterial oxygen saturation) value below 90% causes hypoxemia (which can also be caused by anemia). Hypoxemia due to low Sa_{O2} is indicated by cyanosis.

Cyanosis : It is a blue coloration of the skin and mucous membranes due to the presence of > 5g/dl deoxygenated hemoglobin in blood vessels near the skin surface.

3.7 ANSWER TO CHECK YOUR PROGRESS

Check Your Progress 1

i) Pulmonary edema is an abnormal fluid accumulation in the alveoli and interstitial spaces of the lungs (Due to extravasations of fluid from the pulmonary vasculature into the interstitium and alveoli of the lung). It leads to impaired gas exchange and may cause respiratory failure.

ii)
- Chest trauma, pulmonary contusion
- Aspiration
- Smoke inhalation
- Sepsis
- Non-thoracic trauma
- Leuko-agglutination reactions

iii)
- Myocardial infarction, ventricular septal rupture following infarction:
- High output heart failure due to septicaemia, thyrotoxic crisis, anaemia
- Valvular disorders : Acute aortic regurgitation, mitral stenosis, severe aortic stenosis
- Severe hypertension

iv) **Table 3.1: Distinguishing failures of CPE and NCPE**

Cardiogenic pulmonary edema (CPE)	Non-Cardiogenic pulmonary edema (NCPE)
Early signs include exertional dyspnea and orthopnea	Ranges from mild dyspnea to respiratory failure
Evidence of increased intracardiac pressures (S3 gallop, elevated jugular venous pulse along with jugular venous distention, peripheral edema) Increasing airway edema is associated with rhonchi and wheezes on auscultation of the chest (Rales and wheezing is due to airway compression from peribronchial cuffing	Pulmonary findings may be relatively normal in the early stages Patients with NCPE have a warm periphery, a bounding pulse, and no S3 gallop or jugular venous distention.
Hypoxemia is due to ventilation-perfusion V/Q mismatch and responds to the administration of supplemental oxygen	Hypoxemia is due to intrapulmonary shunting and typically persists despite high concentrations of inhaled O_2.
The chest radiograph shows peribronchial thickening, prominent vascular markings in the upper lung zones, and Kerley B lines. Also an enlarged cardiac silhouette, interstitial thickening, and perihilar alveolar infiltrates (seen when pulmonary edema worsens) Pleural effusions are common	The chest radiograph shows normal heart size, patchy alveolar infiltrates with air bronchograms are more indicative of noncardiogenic edema. Pleural effusions are uncommon.
The pulmonary capillary wedge pressure (PCWP) is generally >18 mm Hg in CPE	<18 mm Hg in NCPE But superimposition of chronic pulmonary vascular disease can make this distinction difficult.

v)
- Initial management of patients with Cronic Pulmonary Edemia (CPE) should address the BAC of resuscitation, that is circulation airway and breathing.
- Oxygen should be administered to all patients to keep oxygen saturation >90%.
- The method of oxygen delivery varies from use of a face mask to bilevel noninvasive positive-pressure ventilation (NPPV) or continuous positive airway pressure (CPAP) or intubation and mechanical ventilation depending on presence of hypoxemia and acidosis and on the patient's level of consciousness.
- In case of persistent hypoxemia, acidosis or altered mental status, intubation and mechanical ventilation may become necessary.

- Any associated arrhythmia or myocardial infarction should be treated appropriately.

Check Your Progress 2

i) Activity intolerance related to fatigue secondary to cardiac insufficiency and pulmonary congestion as evidenced by easy tiredness with mild activities, weakness.

Objective: The client will achieve activity level as much as tolerable as evidenced by ability to do mild activities.

Interventions

- Assess the activity level of the patient
- Encourage alternate rest and activity periods to reduce cardiac workload
- Provide calming divertional activities to promote relaxation to reduce oxygen consumption and to relieve dyspnea and fatigue
- Teach active and passive exercises as tolerated by the patient
- Encourage mild activities by self
- Advise to avoid heavy or strenuous activities
- Appreciate when able to do activities by self
- Advise to take assistance whenever required and provide calling bell to patient with appropriate instructions.

ii) Anxiety related to dyspnea or perceived threat of death as evidenced by restlessness, irritability, expression of feelings of life threat.

Objective: The patient achieves reduction in anxiety as evidenced by verbalization.

Interventions

- Use a calm, reassuring approach to relieve anxiety
- Explain all procedures including sensations experienced during procedure to promote feeling of security
- Allow patient to verbalize
- Clear all the doubts
- Introduce to patients with similar problem who has recovered successfully
- Teach relaxation techniques

Check Your Progress 3

i)
- Antithrombin III deficiency
- Protein C deficiency
- Protein S deficiency
- Factor V Leiden (most common genetic risk factor for thrombophilia)
- Plasminogen abnormality

ii)
- Fractures (fat)
- Immobilization
- Burns
- Obesity
- Old age
- Malignancy of ovary, pancreas, stomach extrahepatic Bile duct system
- AIDS (lupus anticoagulant)
- Behçet disease
- Congestive heart failure (CHF)
- Myocardial infarction

iii) Surgical management of acute pulmonary embolism i.e., pulmonary embolectomy is done as last resort through an open procedure usually while patient is on cardiopulmonary bypass.

Chronic pulmonary embolism leading to pulmonary hypertension (known as chronic thromboembolic hypertension) is treated with a surgical procedure known as a pulmonary thromboendarterectomy.

iv)
- Monitoring thrombolytic therapy
- Managing pain
- Managing oxygen therapy
- Relieving anxiety
- Monitoring for complications

Check Your Progress 4

i)
- Smokers and the elderly are at an increased risk.
- Atelectasis implies some blockage of a bronchiole or bronchus, which can be within the airway (foreign body, mucus plug), from the wall (tumor, usually squamous cell carcinoma) or compressing from the outside (tumor, lymph node, tubercle).
- Poor surfactant spreading during inspiration, causing an increase in surface tension which tends to collapse smaller alveoli.
- Atelectasis may also occur during suction, as along with sputum, air is withdrawn from the lungs. There are several types of atelectasis according to their underlying mechanisms or the distribution of alveolar collapse; resorption, compression, microatelectasis and contraction atelectasis.
- Pulmonary embolism (PE) another cause of Atelectasis.

ii) **Acute Atelectasis**

Acute atelectasis is a common postoperative complication, especially after chest or abdominal surgery. Acute atelectasis may also occur with an injury, usually to the chest (such as that caused by a car accident, a fall, or a stabbing). Atelectasis following surgery or injury, sometimes described as massive, involves most alveoli in one or more regions of the lungs. In these circumstances, the degree of collapse among alveoli tends to be quite consistent and complete. Large doses of opioids or sedatives, tight bandages,

chest or abdominal pain, abdominal swelling (distention), and immobility of the body increase the risk of acute atelectasis following surgery or injury, or even spontaneously.

In acute atelectasis that occurs because of a deficiency in the amount or effectiveness of surfactant, many but not all alveoli collapse, and the degree of collapse is not uniform. Atelectasis in these circumstances may be limited to only a portion of one lung, or it may be present throughout both lungs. When premature babies are born with surfactant deficiency, they always develop acute atelectasis that progresses to neonatal respiratory distress syndrome. Adults can also develop acute atelectasis from excessive oxygen therapy and from mechanical ventilation.

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iii)
- Encourage movement and deep breathing in anyone who is bedridden for long periods.
- Keep small objects out of the reach of young children.
- Maintain deep breathing after anesthesia.

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UNIT 4 ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND STATUS ASTHMATICS

Structure

4.0 INTRODUCTION

Chronic obstructive pulmonary disease (COPD), is characterized by abnormalities in the lungs that make it difficult to exhale normally. Generally, two distinct diseases are involved, emphysema and chronic bronchitis. Emphysema and chronic bronchitis cause excessive inflammatory processes that eventually lead to abnormalities in lung structure permanently obstructing the airflow (hence the term "chronic obstructive"). The adults with asthma are twelve times more likely to develop COPD than those who do not have. Status Asthmaticus is a medical emergency in which asthma symptoms are refractory to initial bronchodilator therapy in the emergency department. Patients report chest tightness, rapidly progressive shortness of breath, dry cough, and wheezing. In this unit we shall discuss about definition, causes, risk factors pathophysiology signs and symptoms and medical and nursing management of Chronic Obstructive Pulmonary Disease (COPD), acute exacerbation of chronic obstructive lung disease and status asthmatics.

4.1 OBJECTIVES

After completing this unit, you will be able to:

- highlight the magnitude of Chronic Obstructive Pulmonary Disease COPD and Status Asthmaticus;

- define the term Chronic Obstructive Pulmonary Disease (COPD) and Status Asthmaticus;
- list the causes of Chronic Obstructive Pulmonary Disease (COPD) and Status Asthmaticus;
- explain the pathophysiological changes in Chronic Obstructive Pulmonary Disease (COPD) and Status Asthmaticus;
- describe the clinical manifestations of Chronic Obstructive Pulmonary Disease (COPD) and Status Asthmaticus;
- discuss the importance of diagnostic features of Chronic Obstructive Pulmonary Disease (COPD) and Status Asthmaticus;
- describe the therapeutic management, nursing management and management of complications; and
- discuss the acute exacerbation of chronic obstructive lung disease.

4.2 CHRONIC OBSTRUCTIVE PULMONARY DISEASE

In this section we shall focus on various aspects and management of chronic obstructive lung disease and in Section 4.3 we shall focus in acute exacerbation of chromic pulmonary disease.

4.2.1 Meaning and Risk Factors

According to W.H.O. Chronic obstructive pulmonary disease (COPD) is a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing and is not fully reversible. The more familiar terms 'chronic bronchitis' and 'emphysema' are no longer used, but are now included within the COPD diagnosis. COPD is not simply a "smoker's cough" but an under-diagnosed, life-threatening lung disease.

Causes and Pathophysiology

As you are aware that the single most significant causes of COPD is smoking. Tobacco causes 80% to 90% of COPD cases. Tobacco smoke stimulates inflammation in the lungs, leading to destruction of the alveoli and narrowing of the airways. Familial emphysema, or alpha-antitrypsin (AAT) deficiency-related emphysema, is caused by the hereditary deficiency of a protein called alpha1-antitrypsin. This deficiency leads to uncontrolled destruction of the alveoli and emphysema. Occupational exposure to dust, fumes, and gases appears to contribute slightly to lung function decline and chronic bronchitis.

Risk factors for COPD

Tobacco use is the number one risk factor for COPD and heavy smokers are at greatest risk. Cigarette smokers are at greater risk than cigar and pipe smokers. All smokers are at greater risk than lifelong nonsmokers.

Alpha–1–antitrypsin (AAT) deficiency, also called familial emphysema, is another risk factor. People with familial emphysema have a rare hereditary deficiency of alpha–1–protease inhibitor. When there is a deficiency of AAT, the activity of elastase—an enzyme that breaks down elastin—is not inhibited and elastin degradation occurs unchecked.

Let us see how emphysema and bronchitis leads to COPD.

Emphysema

As you know that there are millions of tiny, thin-walled, elastic air sacs called alveoli in the lungs. These tiny sacs perform the crucial task of replenishing the blood with oxygen (via inhalation) and removing carbon dioxide (CO2) during exhalation. When the alveoli enlarge due to disease process it may results in emphysema.

- Emphysema is the enlargement of the alveoli accompanied by destruction of their walls. In "smoker's emphysema" cigarette smoke sets off a self-perpetuating, low-grade inflammation that causes the release of enzyme (protease) from inflammatory cells which break down polypeptide chain in lung tissue. Normally protease inhibitors counteract this action. In smokers lung these inhibitors are insufficient. This creates an imbalance between the anti elastin-degrading enzymes and their inhibitors thereby decreasing the amount of active anti elastase available to protect the lung and further upsetting the elastase-antielastase balance.

- This disruption of the alveolar walls and elastin leads to a decrease in the elastic recoil of the lungs, limiting the ability of the alveoli to passively shrink and to exhale. This accounts for the main limitation to exhalation seen in severe COPD. The disruption of the alveolar walls also leads to their increase in size, making the lungs larger and placing the chest at a mechanical disadvantage. Disruption of the alveolar walls also makes exchange of oxygen from the alveoli to the capillaries and carbon dioxide from the capillaries to the alveoli more difficult. Collapse of the bronchial walls occur when the cartilage in the bronchial walls has been weakened.

Familial emphysema

- People with familial emphysema have a hereditary deficiency of alpha-1-protease inhibitor, also called alpha - antitrypsin (AAT). When there is a genetic deficiency of AAT, the activity of elastasean enzyme that breaks down elastin – is not inhibited and elastin degradation occurs unchecked. Individuals with a severe genetic deficiency of AAT usually have symptoms by the time they reach early middle age. It is critical that people with this deficiency never smoke.

- Destruction of alveolar walls, capillaries, and attachments between alveoli and bronchioles causes –

  a) Susceptibility of airways to compression and collapse, impeding airflow out of the lungs;

  b) Entrapment of air in the alveoli.

  c) Poor air-gas exchange, that is, reduced ability to inhale oxygen and exhale carbon dioxide (CO2), resulting in increased levels of CO2 in the blood.

  d) The development of bullae (areas of lung extensively destroyed so that they become large dilated air sacs).

  e) Enlarged lungs.

Chronic Bronchitis

Tobacco smoke causes inflammatory cells (neutrophils and leukocytes) to arrive in the bronchi. These cells worsen airway obstruction by causing inflammation and thickening of the airways. Also, mucus-producing glands deep within the lining of the airways become enlarged (hypertrophy) and increase in number (hyperplasia), and the number of surface cells that produce mucus (goblet cells) increases, resulting in excessive secretion of mucus in the lungs. The resulting chronic cough and expectoration affects the central conducting airway. This mechanism can be shown by following flow diagrams fig 4.1 a and 4.1. b.

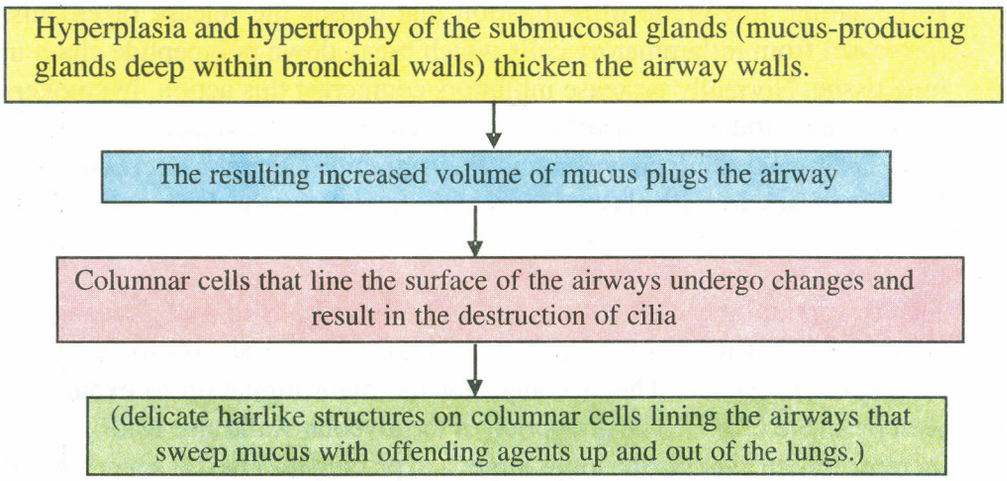

Fig.4.1(a): Flow diagram showing mechanism of COPD

The loss of cilia and the inability to clear bacteria predispose the patient to lung infections.

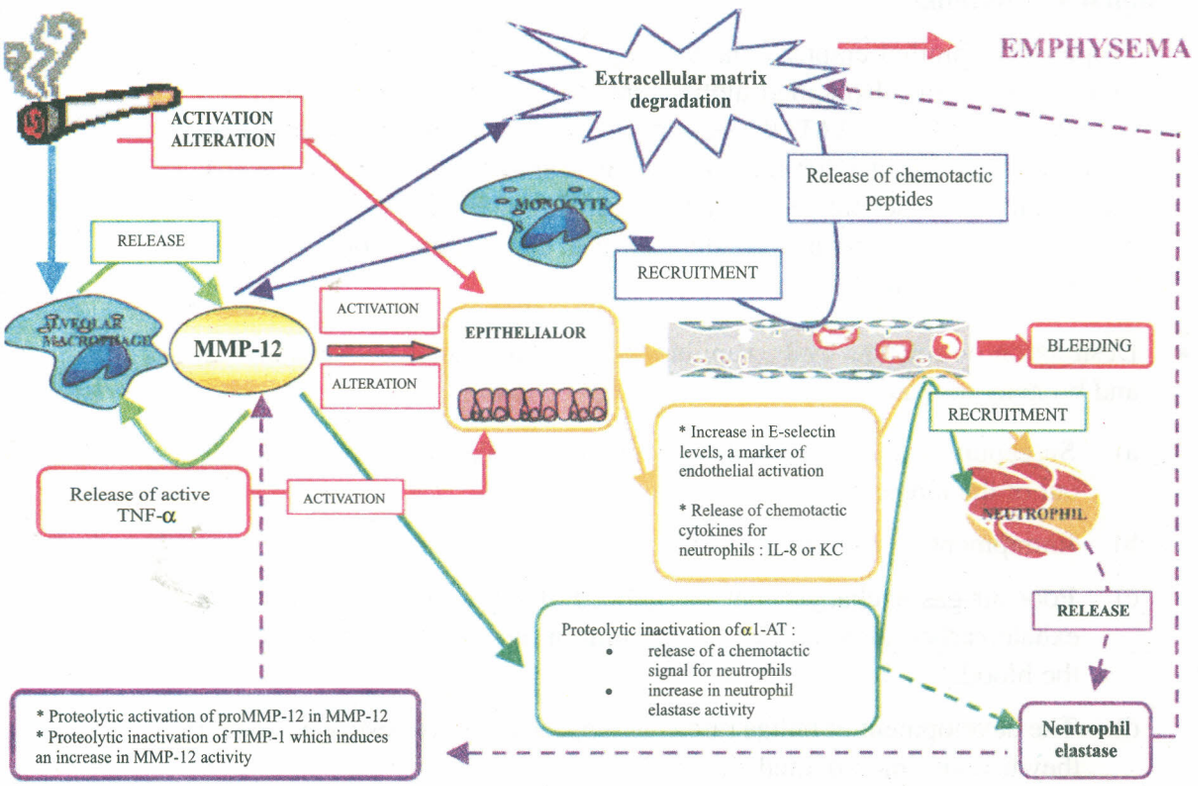

Fig. 4.1(b): Pathophysiological changes

4.2.2 Signs and Symptoms

Signs

Signs of COPD are consequences of the anatomical changes caused by the disease:

- Barrel Chest
- Pursed-Lip Breathing
- Productive Cough
- Cyanosis

Let us explain each of one of the signs:

Many of the signs of COPD are caused by the body's attempt to compensate for a damaged respiratory system. Symptoms develop as a direct result of disease processes.

Barrel Chest

One of the characteristic sign is the change in the shape of the chest, known as barrel chest. When the lungs become enlarged, the diaphragm is displaced downward and is unable to contract efficiently. Furthermore, the chest wall is enlarged, making accessory breathing muscles (muscles in the neck, upper chest, and between the ribs) less efficient as well. These changes contribute to shortness of breath. This becomes apparent when a person with COPD tries to do something with the arms raised above the head, such as changing a light bulb in a ceiling fixture, and becomes short of breath.

To compensate, a person with COPD often sits leaning forward with their arms supported on a surface in front of them or on their knees. This stabilizes the upper chest and shoulders and allows them to use accessory respiratory muscles more efficiently.

Pursed-Lip Breathing

Because airflow out of the lungs becomes limited, exhalation takes longer. Because the alveoli lose their elasticity, one tries to shorten the time needed for exhalation by forcefully exhaling. Unfortunately, forced exhalation increases pressure on the lungs and causes structurally weakened airways to collapse. To prevent airways from closing during forced exhalation, pursed-lip breathing is used: The lips are narrowed together, which slows exhalation at the mouth. This keeps positive pressure in the airways, thus preventing their collapse and allowing some forced exhalation.

Productive Cough

A productive cough is caused by inflammation and excessive amounts of mucus in the airways. Coughing becomes less effective because of obstructed airflow.

Cyanosis

People who have a poor supply of oxygen usually have a bluish tinge to their skin, lips, and nail beds, called cyanosis.

We have focused on signs now let us talk about symptoms.

Symptoms

- **Shortness of Breath (dyspnea):** Dyspnea, the most common symptom of COPD, comes on gradually and is first noticed during physical exertion or during acute exacerbations. It usually begins when patients are in their 60s and 70s and slowly becomes more prominent. It is closely associated with lung function decline and is not always associated with low oxygen in the blood.
- **Chronic Cough:** Chronic cough typically begins as a morning cough and slowly progresses to an all-day cough. The cough usually produces small amounts of sputum (less than 60 mL/day) and is clear or whitish but may be discolored.
- **Wheezing:** Wheezing is the high-pitched sound of air passing through narrowed airways. A person with COPD may wheeze during an acute exacerbation or chronically. Sometimes the wheezing is heard only at night or with exertion. Bronchodilators can relieve wheezing quickly.
- **Hemoptysis:** COPD is one of the more common causes of hemoptysis (coughing up blood). It usually occurs during an acute exacerbation, when there is a lot of coughing with purulent sputum (sputum containing pus). Usually, there are only very small amounts of blood streaking the sputum. Hemoptysis may be a sign of lung cancer in a patient with COPD, so any blood appearing in the sputum should be brought to a doctor's attention.
- **Weight Loss:** Patients with severe COPD work hard and burn a lot of calories just breathing. These patients also become short of breath in the very act of eating, and so may not eat enough to replace the calories they use.
- **Lower Extremity Edema:** In severe cases of COPD, pulmonary artery pressures increase and the right ventricle of the heart contracts less efficiently. When the heart is unable to pump enough blood to meet the needs of the kidneys and liver, edema (swelling) in the feet, ankles, and lower legs results. It can also cause the liver to become swollen and tender or fluid to accumulate in the abdomen (ascites). A distended abdomen can be a sign of ascites.

Clinical Featured/ Characteristics are based on emphysema and chronic bronchitis and are given in table 4.1.

Table 4.1: Distinguishing clinical features of emphasis and chronic bronchitis

Clinical features/ Characteristics	Emphysema	Chronic Bronchitis
Referred to as :	Pink puffers	Blue bloaters
Age at diagnosis	60 years	Approximately 50 years
Appearance	Chest forward, hands on knees, thin, purse-lip breathing	Expanded chest or barrel chest
Bronchial infection	Less frequent	More frequent
Cor pulmonale	Rare	Common
Cough	After dyspnea onset	Before dyspnea onset

Dyspnea	Severe	Mild
Episodes of respiratory insufficiency	Frequent terminal	Repeated
Hematocrit (%)	34-45	50-60
Paco2 mmHg	35-45	50-60
PaO2	65-75	45-60
Sputum	Mucoid, scanty	Purulent, copious

4.2.3 Diagnostic Features

The diagnostic process of COPD includes a thorough examination and following diagnostic procedures:

- History Taking and Examination
- Pulmonary Function Tests
- Oximetry
- Radiological Procedures
- Arterial Blood Gases
- Alpha-1-Antitrypsin Level

History and Examination

Patients with COPD usually are current or past smokers over the age of 40 with a history of shortness of breath upon physical exertion and chronic productive cough. The physical examination may show a barrel chest, decreased breath sounds, and wheezing. Signs of right-sided heart failure, such as edema, tender liver and a scites, clubbing of fingers and central cyanosis may occur.

Pulmonary Function Tests (PFTs)

Pulmonary function tests are the primary diagnostic tools for COPD, after the medical history and physical examination. These tests demonstrate characteristic abnormalities in lung function that confirm or support the diagnosis of COPD and give some idea of the degree of impairment and prognosis.

There are four components to pulmonary function testing: Spirometry, Post bronchodilator Spirometry, lung volumes, and diffusion capacity. In the initial evaluation, all four components are often performed. Periodically, an individual component, most commonly spirometry, is performed to assess progression of disease and to determine the effectiveness of medication. The details of the PFTs is given below:

i) Spirometry

It is most reliable way to determine reversible airway obstruction that measures the amount of air entering and leaving the lungs. The patient sits comfortably in front of the spirometry machine. The machine measures airflow that passes through the inhalation port attached to the machine. The inhalation device is usually a disposable cardboard tube or a reusable tube

that can be sterilized after use. The patient inhales as deeply as possible and forms a seal around the tube with their mouth. Then the patient exhales, as forcefully and rapidly as they can, until they can exhale no more. To be an adequate test, the patient must exhale all the air they possibly can, continue exhaling for at least another 6 seconds. Usually, three separate attempts are made and the best result is used for evaluation.

Multiple measurements are obtained from this manoeuver. Those most commonly used for interpretation are **forced expiratory** volume after 1 second [FEV1], **forced vital capacity** [FVC], and **forced expiratory** flow at 25%-75% of maximal lung volume [FEF 25-75]. They are expressed as percentages of what is predicted for normal lung function, depending on the variables of height, age, race and sex.

The amount of air exhaled (forced vital capacity, or FVC) is reduced, compared to a person with normal lung function. Furthermore, the amount of air exhaled during the initial 1 second (FEV1) is reduced and is reduced to a greater degree than the entire FVC. Therefore, the ratio of air exhaled after 1 second is low compared to the total amount of air exhaled. In healthy lungs, 70%-75% of all the air exhaled after maximum inhalation (FVC) is exhaled within the first second (FEV1), known as the FEV1/FVC ratio. In lungs with COPD, the FEV1/FVC ratio falls below 70%-75%.

The absolute value of the FEV1 is also reduced. The FEV1 is used to quantify the severity of obstruction and can be mild, moderate severe and very severe as given below:

Meld : FEV1 < 70% of what is predicted for age, height, weight and race;

Moderation : FEV1 < 50% to 69%,

Severe : FEV1 < 35%-49%,

Very Severe : FEV1 < 35%, very.

Sometimes the only abnormality is a reduction in the forced expiratory flow rate (FEF) 26-75 Isolated reduction in the FEF25-75 is considered an early detector of very mild obstruction. It can also be a normal variant.

ii) Post bronchodilator Spirometry

Spirometry is often repeated after giving the patient a bronchodilator, such as an inhaled beta-agonist. If the FEV1 (forced expiratory volume after 1 second) improves more than 12%, the obstruction may be reversible or partially reversible. This procedure provides some information on the potential responsiveness of the airways to medication. It is also useful for determining whether steroid treatment has been beneficial, a few weeks after initiating therapy.

Peak expiratory flow rate (PEFR) also can be obtained. PEFR can be compared with readings the patient obtains at home with a peak flow meter. A peak flow meter is a portable device that consists of a small tube with a gauge that measures the maximum force with which one blows air through the tube.

iii) Lung Volumes

Lung volumes are measured in two ways, gas dilution or body plethysmography. The gas dilution method is performed after the patient inhales a gas, such as nitrogen or helium. The amount of volume in which the gas is distributed is used to calculate the volume of air the lungs can hold. Body plethysmography requires the patient to sit in an airtight chamber (usually transparent to prevent claustrophobia) and inhale and exhale into a tube. The pressure changes in the plethysmograph are used to calculate the volumes of air in the lungs.

The most important measurements obtained are residual volume and total lung capacity (TLC). These measurements vary with age, height, weight, and race and are usually expressed as an absolute number and a percentage of what is predicted for a person with normal lung function. A high TLC demonstrates hyperinflation of the lungs, which is consistent with emphysema. Increased residual volume signifies air trapping. This demonstrates an obstruction to exhalation.

iv) Diffusion Capacity

Diffusion capacity is a measurement of gases transfered from the alveoli to the capillary. The patient inhales a very small amount (very safe) of carbon monoxide. How much of it is taken into the blood is measured. A reduced diffusion capacity is consistent with emphysema but is seen in a many other lung diseases as well.

Oximetry

This noninvasive method determines the oxygenation of the blood (O2 saturation; normal is greater than 90%) by measuring the amount of light transmitted through an area of skin. The device must be able to read pulsatile flow, so it must pick up a pulse to be accurate. Oximetry is not as accurate as the measurement of arterial blood gases. It is commonly used during exercise and sleep. Exercise oximetry can determine if a patient's oxygen decreases during activity. If so, oxygen therapy with activity may be beneficial. Overnight oximetry is done to see if oxygen concentrations decrease during sleep.

Radiology

Chest x-ray is an imprecise method of diagnosis of COPD. It is only consistently abnormal in severe cases and should be performed in the initial evaluation to exclude other lung diseases. Findings characteristic of COPD in chest x-ray are hyperinflated lungs with flattened diaphragm, hyperlucent lungs (chest film shows greater than normal film blackening from increased transmission of x-rays), and central pulmonary artery enlargement. Bullae, areas of destroyed lung tissue that create large dilated air sacs, may be seen as well.

CT scan may be used to more accurately diagnose emphysema. This is usually not necessary, however, and abnormal lung anatomy is not always detected.

Arterial Blood Gases analysis

Arterial blood gases are measured using blood drawn from an artery, usually in the wrist. Blood is usually drawn from a vein, but venous blood is inaccurate for these measurements. Drawing blood from an artery, unfortunately, causes more discomfort.

Arterial blood gases are measured to determine the amount of oxygen dissolved in the blood (pO2), the percentage of hemoglobin saturated with oxygen (O2 saturation), the amount of carbon dioxide dissolved in the blood (pCO2), and the amount of acid in the blood pH.

The oxygen measure may be used to determine whether a patient needs oxygen therapy. The carbon dioxide measure gives some idea of lung function and is especially important to know when starting oxygen therapy (see Oxygen).

Alpha-1-Antitrypsin Level

A person suspected of having a genetic deficiency of this enzyme will undergo this test. Alpha-1-antitrypsin deficiencies can also cause liver disease in children, and the level may be measured for that as well. If the level is low, a genetic probe may be used to determine the cause.

4.2.4 Therapeutic Management and Nursing Management

COPD is not a reversible condition, but treatment can slow its progression (smoking cessation being the most important). Treatments available to help manage the disease include the following:

- Smoking cessation
- Nutritional support
- Pulmonary rehabilitation
- Surgical treatment
- Other treatments / Advance Care directives.

Smoking Cessation

It is critically important that COPD patients quit smoking. Once a patient has quit, the rate of decline of lung function slows considerably. It is very difficult to quit smoking cigarettes because they are psychologically and physically addictive, but it can be done. Withdrawal symptoms are caused by withdrawal from nicotine and include depression, insomnia, irritability, anxiety, poor concentration, and weight gain. Some patients gain 10–20 pounds after they stop smoking.

There are several nicotine replacement systems that can help smokers withdraw from nicotine, including nicotine gum, patches, inhalers, and nasal sprays. Nicotine gum was the first nicotine replacement therapy available. Each piece of gum contains 2 mg of nicotine. It is chewed slowly to reduce symptoms of withdrawal. The major disadvantages of nicotine gum are that it takes training to use properly and the peak nicotine blood levels only approximate 40% of what one would obtain by smoking a cigarette. The gum decreases, but does not eliminate, physical withdrawal symptoms.

Nutritional Support

Nutrition is critically important for patients who lose a lot of weight. High fat, low carbohydrate diets are recommended. If a patient is significantly overweight, losing weight may be appropriate.

The treatment for COPD is palliative, not curative. It is probable that longevity cannot be significantly improved with any treatment, except in patients with hypoxemia who benefit from supplemental oxygen therapy.

Surgical Treatment

Types of surgery that may be used to treat COPD include the following:

- Lung Volume Reduction Surgery
- Lung Transplantation.

Lung Volume Reduction Surgery

In lung volume reduction surgery (LVRS), the upper portions of the diseased lungs are removed. This improves symptoms and lung function for some patients. Possibly, the chest wall and breathing muscles return to a place of mechanical advantage. Or perhaps the elastic recoil of the lungs improves as a result.

Lung Transplantation

Single or double lung transplantation may be an option for some severe cases.

Pulmonary Rehabilitation

Patients with COPD become physically unfit. Most hospitals offer pulmonary rehabilitation programs that can improve fitness, even in severe cases. The programs usually include exercises for the lower and upper extremities, education, breathing retraining, and psychosocial support. Exercising the legs particularly can improve endurance. Upper extremity exercises may be beneficial as well. Patients use a lot of energy and tire easily because they breathe rapidly and shallowly. Learning pursed-lip breathing can help relieve these symptoms.

A comprehensive pulmonary rehabilitation (PR) program may lead to significant clinical improvement by increasing exercise tolerance and reducing shortness of breath PR programs may even reduce the number of hospitalizations, although to a lesser extent.

This PR programs are aimed at improving independence and improving quality of life, they do not improve lung function or prolong survival.

Ideally, a variety of health care professionals are required to deliver the wide range of services offered in a comprehensive PR program. Educating patients about their disease is a key component. Exercise training, often with oxygen, may take place at the home or in a clinic setting and often includes stationary bicycling, stair climbing, and walking to improve leg strength, plus weight lifting to improve arm strength. Techniques are taught to decrease shortness of breath during exercise and sexual activity.

Additionally, patient evaluation and goal setting, nutritional evaluation and counselling, psychosocial counselling (addressing depression, anxiety, sexual activity limitations), and the coordination of complex medical services. Medication counselling is an important and integral part of PR, since medication non-adherence is a serious complicating factor in COPD management. Education regarding the appropriate dosing and timing of regularly scheduled and as-needed medications, the proper technique for self-administering inhaled medications, ongoing monitoring, and information for family members and caregivers is imperative.

End-Stage Disease

Mechanical ventilation may be necessary for short- or long-term use; some individuals may become dependent on a ventilator until death. Quality of life is diminished with mechanical ventilation due to the patient's inability to speak or eat during these periods. Therefore, it is important for patients to discuss with their physician and loved ones whether they wish to have this form of therapy. Hospice care is an alternative to mechanical ventilation. Advance care planning should be discussed so that a patient can ensure that his or her wishes are carried out.

Nursing Management

Advance Care Directive

Concept of Advance care planning, involves the patient and health care team to help ensure adherence to a patient's wishes regarding the manner in which s/he will die.

There are two types of advance directives **but in India it is a rare practice.**

- A living will and a durable power of attorney. The living will is a document that describes a patient's preferences for the initiation, continuation, or discontinuation of particular forms of treatment. A durable power of attorney is a document that designates a surrogate who will make medical decisions on behalf of the patient, if the patient become incapable of doing so. It is important for the patient or the advocate to complete these documents while the patient is in full is conscious command of his or her metals faculties; thus, the patient's judgment will not be challenged.

- **"Do-Not-Resuscitate" Orders:** The Do-Not-Resuscitate (DNR) order is a statement indicating that cardiopulmonary resuscitation (CPR) will not be performed in the case of cardiopulmonary arrest. DNR orders do not mean do not treat. Other treatments, including ventilatory support, transfusions, dialysis, and antibiotics are given. In India through some times you may come across verbal requests from client regarding this.

Complications

The complications include following:

- **Cor Pulmonale:** Lower extremity edema (swelling) in a patient with COPD is usually a sign of Cor pulmonale (pulmonary hypertension and right-sided heart failure). COPD makes the heart work harder, especially the right side, which pumps blood into the lungs. Because of poor gas exchange in COPD, there are decreased amounts of oxygen in the blood causing blood vessels to constrict. Many of the capillaries surrounding the alveoli are destroyed in the disease process making the heart work harder to force blood through fewer constricted blood vessels. As a result of this effort, the right ventricle becomes enlarged, the walls of the heart thicken, and the chamber eventually loses its ability to contract efficiently.

End-stage lung disease

When respiratory failure occurs in a patient who has end-stage lung disease, there is a slow decline in lung function and rising levels of carbon dioxide in the blood. The increasing carbon dioxide creates a narcotic effect in the patient, who slowly loses consciousness and stops breathing.

Respiratory failure can occur during an acute exacerbation of COPD or in a patient who has end-stage lung disease.

Other complications

Other complications of COPD includes following:

- Pneumonia
- Polycythemia
- Pneumothorax.

Pneumonia caused by bacterial infection can lead to respiratory failure in these patients. Streptococcus pneumoniae is the most common cause of bacterial pneumonia in patients with COPD.

Polycythemia in COPD is the body's attempt to adjust to decreased amounts of blood oxygen by increasing the production of oxygen-carrying red blood cells. While this may be helpful in the short term, overproduction eventually clogs small blood vessels

A pneumothorax can lead to severe respiratory distress and is treated by inserting a plural tube.

Prognosis

The overall prognosis for a patient with COPD depends on the severity of lung disease and whether the patient continues to smoke. An FEV1 (forced expiratory volume after 1 second) greater than 50% predicted carries a very good prognosis, with the survival of these patients being only slightly less than patients without COPD. Patients with an FEV1 less than 0.75 L (very severe obstruction, less than 30% predicted), have a 1-year mortality rate of 30% and a 10-year mortality rate of 95%. At any level of lung impairment, prognosis improves when the patient quits smoking.

Check Your Progress 1

i) State T for true and F for false for the following statements:

a) COPD is characterised by reversible airways obstruction ❑

b) It rarely occurs in patients under the age of 35 ❑

c) The symptoms rarely vary between days ❑

d) It is linked to a chronic productive cough ❑

ii) Tick Mark the correct answer.

In Severe COPD there is :

a) FEV1 of less than 50% of predicted by NICE / BTS ❑

b) FEV1 of less than 70% of predicted by NICE/BTS ❑

c) FEV1 of less than 50% of predicted by GOLD ❑

d) FEV1 of less than 30% of predicted by NICE / BTS ❑

iii) When the diagnosis of COPD is considered the patient should be questioned about following:

a) occupational history ❑

b) dyspnoea according to the MRC dyspnoea scale ❑

c) ankle swelling ❑

d) All the above ❑

iv) Smoking cessation in COPD patients will

a) help to return the patients FEV1 to normal for their age ❑

b) decline FEV1 ❑

c) have no effect on lung function ❑

d) have no beneficial intervention for all patients ❑

4.3 ACUTE EXACERBATION OF CHRONIC PULMONARY DISEASE

Acute Exacerbations: Acute exacerbations of COPD are characterized by an abrupt increase of symptoms. Cough and sputum production increases. Wheezing is often increased or noted for the first time. Dyspnea (shortness of breath) is increased or apparent for the first time. Exacerbations are caused by bronchial infections in most instances. Fever is uncommon. A person with COPD may initially have one or two acute exacerbations per year, which resolve readily with therapy. The number of exacerbations per year increases as the diseases progresses.

During an acute exacerbation, there is increased airway narrowing due to bronchospasm (contraction of the bronchi and bronchioles), edema, and excessive mucus production. If narrowed airways cause an extreme increase in the work of breathing that cannot be maintained, the patient will die, unless there is intervention (see Mechanical Ventilation). Often the patient needs mechanical support until the acute decline has resolved. Unfortunately, some patients don't recover sufficiently from the acute episode to enable them to breathe on their own. There is no way of knowing who will improve and who will not after such an episode. Generally, those who have worse lung function and functional status are less likely to regain independent breathing.

Refer to box 4.1 which shows for triggering/ predisposing factors for exacerbation of chronic obstructive pulmonary diseases.

Box 4.1 conditions that trigger exacerbation of chronic obstructive pulmonary disease.

- Infections – bacterial e.g., Tracheobronchitis
- Left ventricular failure
- Cardiac dysrhythmias
- Pneumothorax
- Pulmonary throxmoembolism
- Upper airway obstruction
- Aspiration
- Rhinitis or sinusitis
- Asthma
- Gastroesophageal reflux

Signs and Symptoms

Signs and symptoms of acute exacerbations of COPD are given in Box 4.2.

Box 4.2 Signs and symptoms of acute exacerbations COPD.

- Worsening of previously stable condition
- Increased dyspnoea and work breathing
- Increased wheeze and hyperventilation
- Increased cough and chest tightness
- Increased sputum volume
- Increased sputum tenacity and purulence
- Increased water retention
- Worsening V/Q relationship and gas exchange

Therapy for Acute Exacerbations

Acute exacerbation of COPD is an abrupt increase in symptoms of shortness of breath and/or wheezing, often associated with increase in production of purulent sputum (sputum containing pus). Hospitalization may be required if the symptoms are severe.

Antiobiotics: Treatment usually includes antibiotics. Approximately 50% of acute exacerbations are due primarily to the bacteria Streptococcus pneumoniae (causing pneumonia), Haemophilus influenzae (causing flu), and Moraxella catarrhal (causing pneumonia). Numerous antibiotics effectively treat these infections.

- **Medications:** Corticosteroids are beneficial in acute exacerbations of COPD. If the patient is hospitalized, steroids often are given intravenously. Bronchodilator dosages are increased during acute exacerbations to decrease acute bronchospasm. Theophylline may be used during acute exacerbations of COPD.

- **Oxygen:** Oxygen requirements usually increase and supplemental oxygen is generally provided.

- **Mechanical Ventilation:** Patients with acute exacerbations of COPD have a risk for developing respiratory failure. Respiratory failure occurs when respiratory demand exceeds the ability of the respiratory system to respond. Without aggressive intervention at the point of respiratory failure, the patient can die. Aggressive therapy at this point, along with all the above therapies, may include mechanical ventilation. Non invasive ventilation with continuous positive airway pressure (CDAP) bilevel positive airway pressure (BIPAP) masks may be used for cooperative well oriented client/ patient.

- **Decisions About Ventilation :** A patient with severe COPD can decide whether s/he ever wants to be placed on a ventilator. Another decision a person with COPD may want to make is whether to have the ventilator discontinued and to be allowed to die, if s/he is unable to breathe independently. Both decisions should be made in close consultation with the person's physician.

- **Chronic Ventilation:** In the small group of patients unable to be liberated from mechanical ventilation, chronic ventilation may be used. If, after 2 weeks, it becomes apparent that the patient is not likely to come off the ventilator, a tracheostomy is performed, A tracheostomy creates a more stable airway and facilitates movement of the patient and oral care. Patients with a tracheostomy can be maintained on a ventilator indefinitely. So since smoking is the most common cause.

- **Smoking Cessation:** Smoking cessation, including cigarettes, cigars, and pipes, is the most important step in the treatment of COPD. Smoking cessation can revert the decline in lung function equal to the values of nonsmokers. Continuation of smoking essentially will lead to symptoms worsening .

- **Pharmacologic Interventions:** Medication intervention usually consists of life-long chronic therapy with dosage adjustments and additional agents when exacerbations present. According to the American Lung Association, bronchodilators (oral or inhaled) are central to the symptomatic management of COPD. Additional treatment includes antibiotics, oxygen therapy, and systemic glucocorticosteroids.

Check Your Progress 2

i) An infective exacerbation is characterised by:

a) .....

b) .....

ii) The most common causative organisms of infective exacerbations of COPD are:

a) .....

b) .....

4.4 STATUS ASTHMATICUS

In the next section we shall discuss about status asthsmatitics. You will come across such patients in ICU who require ventilator support.

4.4.1 Magnitude, Definition, Causes and Precipitating Factors

Status Asthmaticus is a life threatening form of asthma. It is defined as "a condition in which a progressively worsening attack is unresponsive to the adrenergic therapy drug that leads to pulmonary insufficiency". The primary mechanical event in status asthmaticus is a progressive increase in airflow resistance. Mucous plugging and mucosal edema or inflammation are the major causes for the delayed recovery in status asthmaticus. The combination of hypoxia, hypercapnia, and acidosis, along with the mechanical effects of increased lung volumes may result in cardiovascular depression or cardiovascular arrest.

Precipitating factors

- Infection (pneumonia)
- Pollutants (pollen or smoke)

- Smoking
- Change of local environment sudden change of season, shift into a new house, a new pet, painting the house, taking out old clothes / books
- Drugs (beta blockers) and NSA IDS.
- Acute stress/ anxiety.

4.4.2 Mechanism of Status Asthmaticus

The mechanism of status asthamatics is depicted in following flow chart fig. 4.2.

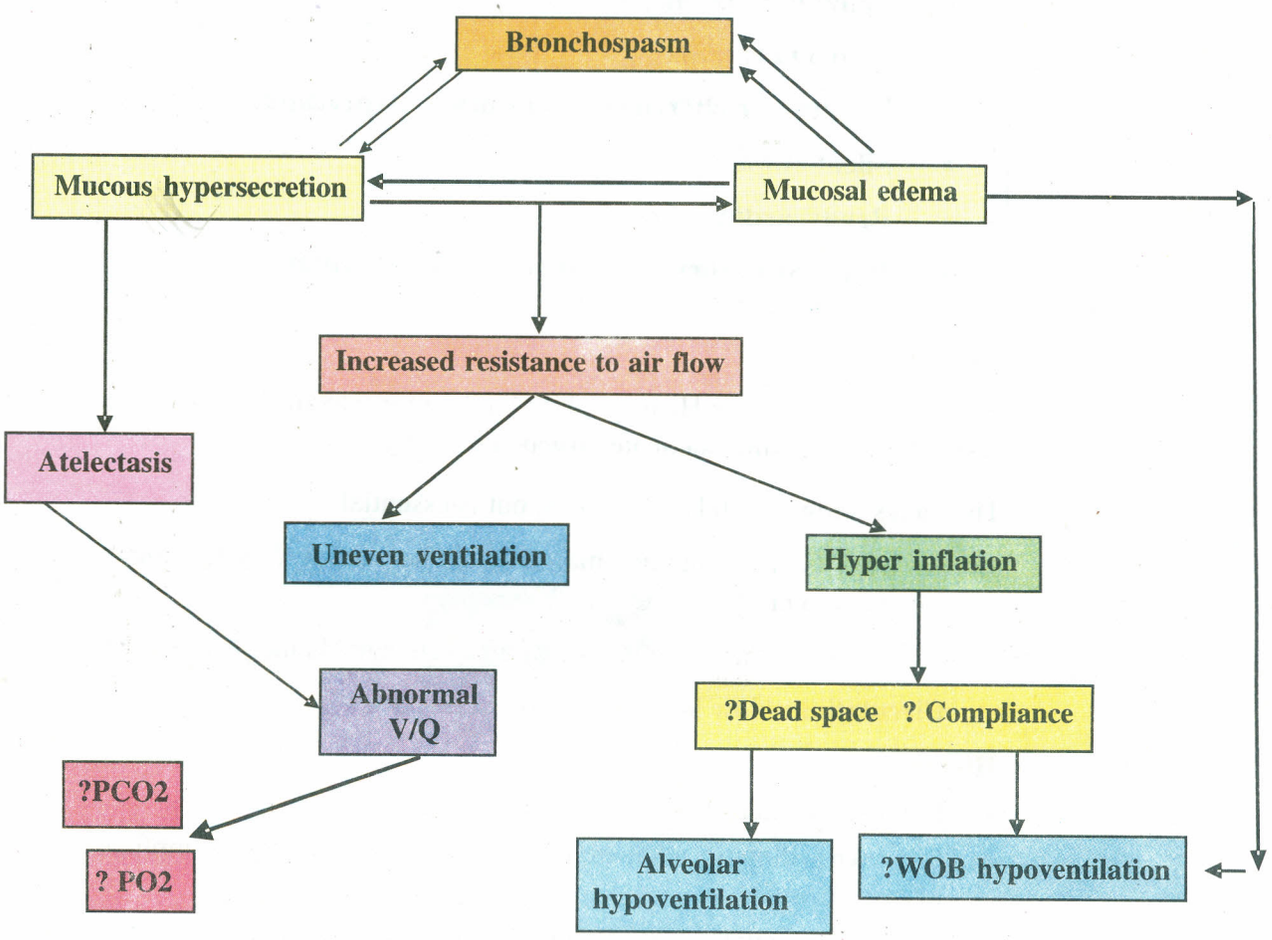

Fig. 4.2: Mechanism of status asthmaticus

4.4.3 Signs, Symptoms and Diagnoses

- Patients with status asthmaticus presents with following signs and symptoms
  - Severe dyspnea that has developed over hours to days.
  - Inability to get out of bed or get proper sleep
  - Inability to complete sentences without becoming breathless.
  - Patients usually present with audible wheezing. Initially, wheezing is heard only during expiration, but, later, wheezing occurs during both expiration and inspiration.
- The chest is hyper-expanded, and accessory muscles, particularly the sternocleidomastoid, sterual, and intercostal muscles, are used. Later, as bronchoconstriction worsens, patients' wheezing may disappear, which may indicate severe airflow obstruction.

- On auscultation you will not hear breath sounds i.e., silent chest.
- Normally, the pulsus paradoxus (i.e., the difference in systolic blood pressure between inspiration and expiration) does not exceed 15 mm Hg. In patients with severe asthma, a pulsus paradoxus of greater than 25 mm Hg usually indicates severe airway obstruction.
- Inspiratory expiratory (ICE) ratio may be upto 1:4 in severe attacks.

Life threatening features include:

- **Inability to speak**
- **Central cyanosis**
- **Reduced or altered consciousness – hypercapnia**
- **Silent chest**
- **Bradycardia**
- **Peak expiratory flow rate (PEF) <100 L/mon**

Diagnosis

Evaluation "ABCs" by History taking and a more detailed examination can be done after you ensure adequate airway, breathing.

This takes a few seconds to minutes, but is essential

a) **Airway:** Can the patient maintain his/her airway? Is the mental status adequate to protect the airway?

b) **Breathing:** What is the degree of air exchange? Is the patient cyanotic?

c) **Circulation:** How is the perfusion? The pulses?

History

- Previous history of wheezing.
- If known asthmatic, what are maintenance medications? Compliance? Time of last aerosol?
- Previous hospitalizations, intubations, last steroid course?
- When did this exacerbation begin?
- Precipitating factors?
- General medical history, including any medications.

Investigation

Chest X-ray for evidence of pneumonia, atelectasis, pneumothorax

Peak expiratory flow rate (PEFR) – to assess the severity. <50% of predicted value suggests severe obstruction

Arterial blood Gas analysis for hypoxemia (PaO_2 < 60 mm of Hg) Acidosis (9pH<7.38)

ECG –for ischemic changes, right heart strain and arrhythmia

TLC/ DLC for infection

Hypercappnia (PaCO2>40 mm of Hg)

Blood chemistry and electrolytes

4.4.4 Collaborative Management

Your patient with status Asthmatics may require admission to the critical care unit. Once in the unit, a multi disciplinary approach from all health team members right from assessment, monitoring, pharmacological and non-pharmacological management to ensure coordination and communication of care is very crucial. This is termed as collaborative management.

Goals of management are follows:

- Early recognition and prompt initiation of therapy.
- Correct hypoxemia
- Overcome airflow obstruction as early as possible
- Reduce recurrence or relapse
- Removal of precipitating factors.

1) Oxygen therapy and nebulization

   Asthmatics are hypoxic due to ventilation-perfusion mismatch (V/Q mismatch). Supplemental oxygen can elevate the pO2. Oxygen in high concentrations (>50-60% for >24 hours),is directly toxic to the lung and can lead to resorbtion atelectasis (replaces nitrogen in the alveolus, oxygen is absorbed into the blood, and the alveolus collapses). Oxygen therapy is continuously given in the concentration of 40-60%.

2) Proper hydration of the patient with I.V. fluids.

3) Nebulisation with salbutamol / turbutaline every 15-30 minutes is given and it is reduced it to 4 hourly once you get the desired response (see fig. 4.1).

4) Betaagonists and corticosteroids are the basis of treatment. Steroids in addition to antiatlammatry action, reduce mucus production and improve oxygenation. For patients on betagonistis and steroids you must monitor for hypokalemia.

5) Terbutaline intravenous terbutaline has become widely used in treating the moderate to severe asthma exacerbation, in which frequent or continuous aerosols have been ineffective, or which is especially severe on initial presentation. It is easily titrated and has a short half life if untoward side effects are encountered. It has almost entirely replaced isoproterenol for use in particularly severe asthmatics.

   - Loading dose 10 micrograms/kg over 10 minutes.
   - Maintenance dose-start at 0.4-1.0 mcg/kg/min. The dose is increased in increments of 0.2-0.4 mcg/kg/min, after assessing for effect and side-effects. Dose can be titrated up quickly if the patient is not excessively tachycardiac. Maximum dose is probably 4 mcg/kg/min.
   - Side effects include tachycardia (most common), hyperglycemia, hypokalemia, worse hypoxia (due to increased V/Q mismatch with infusion), rhabdomyolysis. Monitor Heart rate closely, watch for S-T changes if patient is severe asthmatic and particularly tachycardic.

6) Magnesium

- Magnesium sulfate affects calcium metabolism and promotes smooth muscle relaxation.
- It has been shown to be effective in the treatment of acute, severe bronchospasm.
- Due to.antimylammatery properties the recommended dose is 25-100 mg/kg magnesium sulfate given over 20 minutes (generally 50 mg/kg). "Therapeutic" level is probably around 4 mg/dl. You need to monitor for hypotension during infusion.

7) Theophylline

The use of theophylline in the treatment of an acute asthma exacerbation has become controversial in recent years, as there is evidence that it is not helpful during an acute attack. There remain, however, some patients who are maintained on chronic theophylline or who respond particularly well to theophylline, in whom one might want to continue treatment during an acute exacerbation. One could either continue the oral theophylline preparation, or begin an aminophylline infusion.

- Loading dose 6-7 mg/kg
- Continuous infusion rate depends on patient age
- Therapeutic level 10-20 mg/l. Levels >20 are toxic, though some patients will display side effects (nausea, tachycardia, anxiety, jitteriness) at lower levels. Check levels after the bolus, at 4 hours, and at steady state (12-16 hours).
- Medications that increase theophylline metabolism (and thus lower the level): barbiturates due to phenytoin, isoproterenol.
- Medications that decrease theophylline metabolism (and thus increase the level): allopurinol, cimetidine, erythromycin, propranolol, oral contraceptives.

8) Intubation and Mechanical Ventilation

This is a last resort as it is associated with complications. Increasing CO2 levels and fatigue with reduced respiration are indications for ventilation. Intubation in these cases may produce pneumothorax due to narrow airways increased lung volume, causing high airway pressure. Largest sizes 8 endotracheap tube is used for intubation.

Mechanical ventilation

Ventilator management can be quite challenging. A few general principles are given below:

- Do not try to normalize the pCO2. Tolerate hypercapnia, and use pharmacologic buffering agents if necessary to increase the pH to >7.2. How high a pCO2 you need to tolerate depends on the pressures needed to ventilate the patient.

- Try to keep plateau (alveolar) pressures <30-35 cm H20. Peak pressures may be higher than this due to increased airways resistance.
- Small tidal volumes are usually needed due to high resistance and propensity for air trapping. 5-7 cc/kg is a reasonable place to start.
- Rate should be low and expiratory time long, inspiratory time relatively short. The idea is to leave as much time as possible for expiration, without causing the inspiratory pressure to be too high because you are trying to get the gas in over too short a period. Rates of 10-14 and I:E ratios of 1:4 to 1:6 are typical.
- Volume cycled or pressure cycled ventilation can be used. If using volume cycled ventilation, be sure to watch the pressures generated carefully. If using pressure cycled, the ventilator will usually not reach "plateau" or no flow, and you need to watch the volumes delivered. Frequent reassessment is crucial.
- If you encounter difficulty with oxygenation or just cannot move the chest, manually bag the patient and reassess therapy and ventilator strategy.

The patient must be well sedated and usually paralyzed during mechanical ventilation. Continuous infusions or regularly scheduled doses should be used.

Premedicate with extra sedation and lidocaine before suctioning to reduce the broncho-constriction in response to stimulation.

General management issues

Fluid/electrolytes. The asthmatic patient admitted to the PICU for worsening respiratory distress must be kept nil per orally until such time as you are quite comfortable that s/he is improving, that the risk of deterioration requiring intubation is over, and that the patient will be able to eat/drink and breathe at the same time. Until then you have to maintain hydration with IV fluids. You may see hyperglycemia or hypokalemia due to therapy. Generally it is not of sufficient degree to warrant any additional therapy. Remember that with tachypnea the patient may have excessive fluid losses, or may have been dehydrated on admission due to poor intake orally during illness.

Gastrointestinal prophylaxis: The asthmatic in the ICU is usually on relatively large doses of steroids and is NPO. It is appropriate to treat with an H2 blocker or sucralfate.

Antibiotics: Use antibiotics if you suspect a bacterial pneumonia, sinusitis, otitis media, or other bacterial infection as the cause of the patient's asthma exacerbation.

Chest physiotherapy

Use of chest physiotherapy during an acute simple asthma exacerbation is quite controversial. If used, you should evaluate your patient before and after treatments. Physiotherapy often creates more wheezing immediately, but may be necessary if there is considerable atelectasis or mucous plugging.

Check Your Progress 3

i) Define status asthmaticus?

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ii) List any five risk factors for status asthmaticus.

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iii) State the signs and symptoms of status asthmaticus.

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iv) State the goals of management for Status asthmaticus.

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4.5 LET US SUM UP

In this unit we have discussed about acute exacerbation of Chronic obstructive pulmonary disease (COPD) and status asthmaticus.

The most common cause of COPD is cigarette smoking and the most important step in its treatment is smoking cessation. For COPD patients who continue to smoke, airway obstruction is usually progressive, resulting in early disability and shortened survival. All COPD patients, including the elderly, require tailored medication regimens and medication counselling and should be considered for a Pulmonary rehabilitation program aimed at improving independence and quality

of life. Many clinicians may have difficulty accepting a patient's choice to decline aggressive care and accept impending death. Therefore, patients should discuss their decisions with physicians, family members, and caregivers, and they should prepare advance care directives that articulate their preferences regarding future health care interventions.

4.6 GLOSSARY

Alpha1-antitrypsin (AAT) : (also called alpha antiproteinase or AAP). A protective material produced in the liver and transported to the lungs to help combat inflammation. Deficiency states occur as the result of hereditary defects.

Bronchodilators, fast-acting : (also called "rescue" or "quick relief" medications). These medications quickly relax muscles that tighten around the airways, making the airways wider, breathing easier, and shortness of breath reduced.

Bronchodilators, long-acting: also called ("maintenance" medications). These medications prevent airway spasms throughout the day and night; they take effect more slowly than fast-acting bronchodilators, but work for a longer period of time.

CPAP (continuous positive airway pressure) machine : A breathing machine that provides pressure to keep the upper airways open during breathing. This machine is often used for patients with obstructive sleep apnea.

Cor pulmonale : Enlargement of the right side of the heart. Cor pulmonale weakens the heart and causes increased shortness of breath and swelling in the feet and legs. Patients who have chronic COPD with low oxygen levels may develop this condition.

Incentive spirometer : A device that encourages deep inspiration to expand the lungs and improve cough effectiveness.

Lung volume reduction surgery : surgery in which damaged areas of the lungs are removed so the remaining portion of the lungs can function better. Lung volume reduction surgery is performed only for people with certain types of COPD, and after careful testing and evaluation.

Maximal oxygen uptake : A person's highest rate of oxygen consumption. This measurement is usually expressed in milliliters of oxygen per kilogram of body weight per minute.

Peak expiratory flow rate : A test used to measure how fast air can be exhaled from the lungs.

Pulse oximetry : A non-invasive test in which a device that clips on the finger measures the oxygen level in the blood.

4.7 ANSWER TO CHECK YOUR PROGRESS

Check Your Progress 1

i) a) False

b) True

c) True

d) True

ii) d

iii) d

iv) a

Check Your Progress 2

i) An increased volume of sputum and increased purulence of sputum and increased breathlessness

ii) The most common causative organisms of infective exacerbations of COPD are:

Streptococcus pneumonia

Haemophilus influenzae

Check Your Progress 3

i) Status Asthmaticus is a life threatening form of asthma defined as "a condition in which a progressively worsening attack is unresponsive to the usual appropriate therapy with adrenergic drugs and that leads to pulmonary insufficiency." The primary mechanical event in status asthmaticus is a progressive increase in airflow resistance. Mucous plugging and mucosal edema or inflammation are the major causes for the delayed recovery in status asthmaticus. The combination of hypoxia, hypercapnia, and acidosis, along with the mechanical effects of increased lung volumes may result in cardiovascular depression or cardiovascular arrest.

ii) Precipitating factors

- Infection (pneumonia)
- Pollutants (pollen or smoke)
- Smoking
- Change of season shift to new use, a new pet painting the house taking out old clothes / books
- Drugs (beta blockers)
- Acute stream/ anxiety

iii)
- Patients with status asthmaticus presents with following signs and symptoms
- Severe dyspnea that has developed over hours to days.
- Inability to get out of bed or get proper sleep
- Inability to complete sentences without becoming breathless.
- Patients usually present with audible wheezing. Initially, wheezing is heard only during expiration, but, later, wheezing occurs during both expiration and inspiration.
- The chest is hyper-expanded, and accessory muscles, particularly the sternocleidomastoid, sterual, and intercostal muscles, are used. Later, as bronchoconstriction worsens, patients' wheezing may disappear, which may indicate severe airflow obstruction.
- On auscultation you will not hear breath sounds i.e., silent chest.
- Normally, the pulsus paradoxus (i.e., the difference in systolic blood pressure between inspiration and expiration) does not exceed 15 mm Hg. In patients with severe asthma, a pulsus paradoxus of greater than 25 mm Hg usually indicates severe airway obstruction.
- Inspiratory expiratory (ICE) ratio may be upto 1:4 in severe attacks.

iv)
- Early recognition and prompt initiation of therapy.
- Correct hypoxemia
- Overcome airflow obstruction as early as possible
- Reduce recurrence or relapse
- Removal of precipitating factors.

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UNIT 5 PNEUMONIA, INTERSTITIAL LUNG DISEASE AND PLEURAL EFFUSION

Structure

5.0 INTRODUCTION

Our body needs constant supply of oxygen to support metabolism. The respiratory system brings oxygen through the airway of the lung into the alveoli, where it diffuses into the blood for transport to the tissues. Disorder occurring in this system will alter the normal gas exchange. Interstitial lung disease (ILD) is a broad category of lung diseases that includes more than 130 disorders characterized by scarring. Progressive scarring of lung tissue affects ability to breathe and getting enough oxygen into our bloodstream. When the body diffusions impair inspired due to diseases process, there is risk of infection. For example, in infectious disorders like bronchitis and pneumonia, there is an impairment in mucous and ciliary defenses.

During certain disorders in the lungs excessive pleural fluid may accumulate in pleural spaces leading to pleural effusion. Pleural effusions occur when the rate of fluid formation exceeds that of fluid absorption. Therefore, an appropriate actions needs to be taken to care such patients. In this unit you will learn about

interstitial disease pneumonia and pleural effusion. The causes, Pathophysiological changes, clinical and diagnostic features and management will also be discussed.

5.1 OBJECTIVES

After completing this unit, you will be able to:

- state the meaning of Interstitial Lung Disease (ILD), Pneumonia and Pleural effusion;
- list the causes of Interstitial Lung Disease, Pneumonia and Pleural effusion;
- explain the Pathophysiological changes and discuss the clinical manifestation and diagnostic measures; and
- discuss the Medical, surgical and Nursing management of Interstitial Lung Disease, Pneumonia and Pleural effusion.

5.2 INTERSTITIAL LUNG DISEASE

We shall begin with definition and meaning of interstitial lung disease (ILD).

5.2.1 Definition, Meaning and Etiology

Definition

Interstitial Lung Disease (IDL)

IDL refers to a group of lung diseases affecting the interstitium of the lung, alveolar epithelium, pulmonary capillary endothelium, basement membrane, perivascular and perilymphatic tissues. The term ILD is used to distinguish these diseases from obstructive airways diseases. Also known as diffuse parenchymal lung disease (DPLD) and interstitial pulmonary fibrosis. Interstitial lung diseases is a group of diffuse inflammatory lower respiratory tract disorders, characterized by accumulation of inflammatory cells in the interstitium of the alveolar walls followed by thickened, fibrotic changes.

5.2.2 Causes and Classification

- Idiopathic pulmonary fibrosis
- Sarcoidosis
- Collagen vascular disorders
- Inhalation of inorganic dust such as asbestos, crystalline silica, coal dust
- Organic dust from organisms encountered in farming
- Use of air conditioning
- Animal husbandry
- Radiation
- Infectious agents.

ILD may be classified according to the cause as follows:

Due to Inhaled substances

Inorganic

- Silicosis
- Asbestosis
- Berylliosis

Organic

- Hypersensitivity pneumonitis

Drug induced

- Antibiotics
- Chemotherapeutic drugs
- Antiarrhythmic agents

Connective tissue disease

- Systemic sclerosis
- Dermatomyositis
- Systemic lupus erythematosus
- Rheumatoid arthritis

Infection

- Atypical pneumonia
- Pneumocystis pneumonia (PCP)
- Tuberculosis

Idiopathic

- Sarcoidosis A
- Idiopathic pulmonary fibrosis
- Hamman-Rich syndrome

Malignancy

- Lymphangitic carcinomatosis

Other ILD's

- Idiopathic pulmonary fibrosis
- Connective tissue or autoimmune disease-related pulmonary fibrosis
- Hypersensitivity pneumonitis
- Sarcoidosis
- Eosinophilic granuloma (a.k.a. Langerhan's cell histiocytosis)
- Chronic eosinophilic pneumonia
- Wegener's granulomatosis
- Idiopathic pulmonary hemosiderosis
- Bronchiolitis obliterans
- Lymphangioleiomyomatosis

Thus on the bases of etiology the interstitial lung diseases can be classified into two large groups:

i) Those that have no known cause (idiopathic ILD)

ii) Those with an identifiable cause.

Idiopathic interstitial lung disease is divided into three groups:

- Idiopathic pulmonary fibrosis/usual interstitial pneumonitis (IPF/UIP)
- Non-specific interstitial pneumonitis (NSIP)
- Acute interstitial pneumonitis (AIP).

5.2.3 Pathophysiology

The disease condition damages the lung tissue which causes inflammation in the walls of air sacs in the lungs. Subsequent to inflammation scarring / fibrosis occurs in the interstitium which results in permanent loss of affected tissues to breath and formation of scar tissue also oxygen and also leads to destruction of air sacs and the lung capillaries (fig.5.1) .

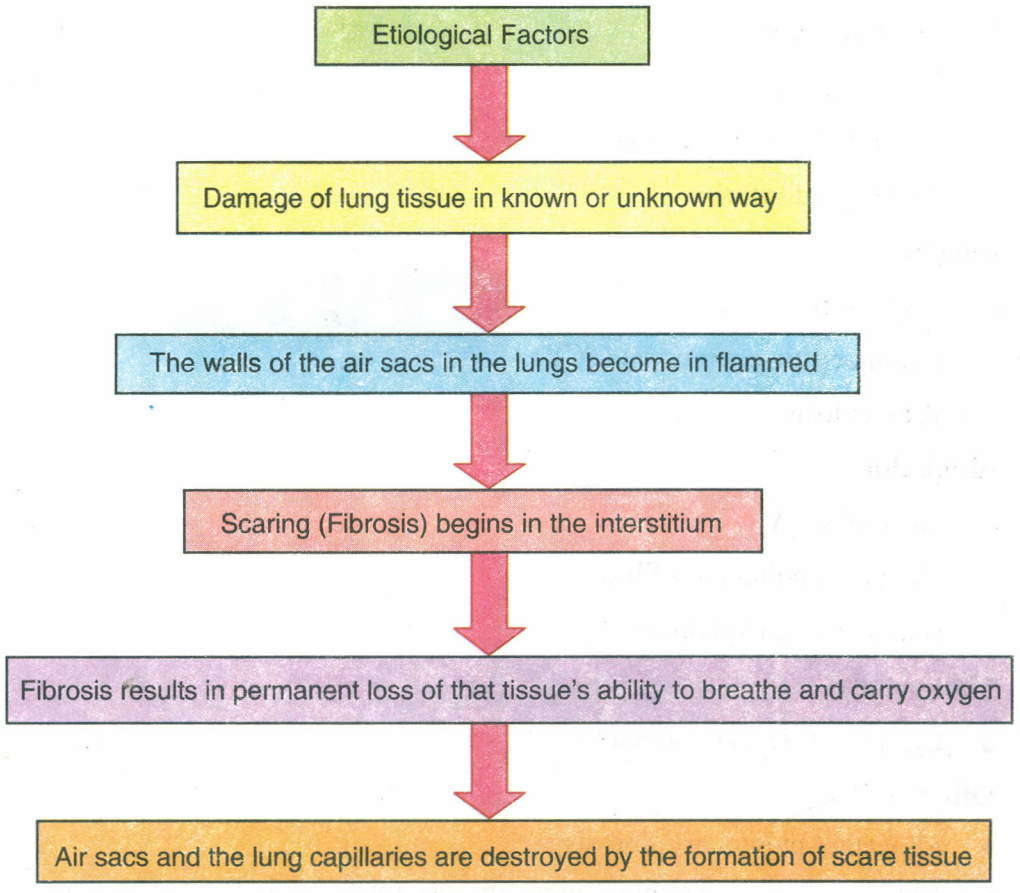

Fig. 5.1: Flow Chart of pathophysiology of ILD

5.2.4 Clinical Manifestations and Diagnostic Measures

It has sudden and non-specific onset. The clinical manifestation includes following:

- Fatigue
- Progressive dyspnea
- Non productive cough

- Dyspnea at rest
- Clubbing of the finger tips
- Shortness of breath, especially with exertion
- Weakness
- Loss of appetite
- Loss of weight
- Discomfort in chest
- Labored breathing
- Hemorrhage in lungs.

Diagnostic measures:

It can be diagnosed by following methods:

- **History and physical examination:** It is important to determine the agents to which the client has been exposed.
- **Physical examination:** It may reveal reduced chest expansion, which is reflected as a decrease in total lung capacity. Inspiratory and expiratory crackles are frequently heard. The crackles have a characteristic sound, like the sound of hook-and-loop tape [Velcro] being pulled apart. Changes in the skin, joints and fingernails can help direct further evaluation.
- **Gallium ventilation perfusion scans:** These scans usually reveal impaired perfusion in the lower lobes and multiple areas of impaired ventilation. The ventilation – perfusion mismatch results in hypoxemia and carbon dioxide retention.
- **Bronchoscopy** and biopsy may also be used to confirm interstitial lung diseases.
- **Pulmonary function tests** to determine characteristics and capabilities of the lungs.
- **Spirometry** to measure the amount of air that can be forced out.
- **PEAK expiratory flow meter** to evaluate changes in breathing and response to medications.
- **Blood tests** to analyze the amount of carbon dioxide and oxygen in the blood.
- **X-ray** computerized axial tomography (CAT) scan.
- **Bronchoalveolar lavage** to remove cells from lower respiratory tract to help identify inflammation and exclude certain causes.
- **Lung biopsy** to remove tissue from the lung for examination in the pathology laboratory.

5.2.5 Therapeutic Management and Nursing Management

Management of a client with ILD is based on the level of respiratory impairment. The goals of treatment are to:

- Identify the specific disease,

- Start the therapy early to decrease inflammation and prevent further lung scarring.
- Remove the source of the problem, if possible.
- Minimize and manage potential complications of ILD.
- Improve or prevent deterioration in a patient's quality of life.

The initial treatment is to remove the client from exposure to the agent. The type of medication and length of therapy depends on the specific type of interstitial lung disease. In some types of ILD, removing the source of exposure, including cigarette smoke, some medications, and environmental irritants may be all that is needed. With other types of ILD, medications and even surgery may be required.

Common treatment options include following:

- Medications
- Oxygen Therapy
- Pulmonary Rehabilitation
- Lung Transplant (in some advanced cases)

Let us explain each one of above treatment modalities.

1) Medications

- **Corticosteroids (Antifibrotic / Anti-Inflammatory Drugs)** Inflammation is controlled with corticosteroids - Prednistone.
- As the disorder progresses inhaled corticosteroids and bronchodilator help to mobilize the secretions.
- **Cytotoxic Agents** or Immunosuppressive Drugs may be used if steroid therapy has failed to be effective or if corticosteroid treatment is not possible. In some cases, a combination of a corticosteroid and cyclophosphamide is used with good results. This medication reduces inflammation by killing some inflammatory cells and suppressing their function. Response to therapy may be slow and require up to 6 months or longer.
- **Azathioprine** [Imuran] used in combination with corticosteroids for the treatment of ILD.
- **Mycophenolate** [CellCept]. It help reduce the amount of steroids required. It works to prevent the immune system from attacking cells in the body that result in fibrosis.

2) Oxygen Therapy

Oxygen therapy is required for some patients with ILD. Decreased lung function and/or pulmonary hypertension may cause blood oxygen levels to be too low. Some may need oxygen therapy all the time while others may need it only during sleep and exercise.

3) Lung transplantation

If the above therapies fail to adequately treat ILD, lung transplant is an option for some advanced cases. With improved surgical techniques and post-transplant care, transplantation may offer improved quality of life and prolonged survival to selected patients.

4) Pulmonary Rehabilitation

A pulmonary rehabilitation program is often recommended to help patients with ILD achieve their highest possible level of functioning. This program includes education, exercise conditioning, breathing techniques, energy saving techniques, respiratory therapy evaluation, nutritional counselling and psychosocial support.

Nursing management

The primary goals of nursing management of the client with interstitial lung disease are given below:

- Promotion of adequate oxygenation
- Maintenance of patent airway
- Achievement of the highest possible functional level
- Note the degree of dyspnea, presence of orthopnea, decreased breath sounds. Assessment of degree of dyspnea, presence of orthopnoea restricted chest wall movement and manifestations of heart failure.
- Evaluate mental status because confusion and restlessness may be early indicators of increasing hypoxia and hypercapnia.
- Record the client's baseline oxymetry and the level of inspired oxygen.
- Advise the client to stop smoking
- Provide psycho social support
- Monitor arterial blood gas (ABG) values
- Maintain intensive pulmonary toilet
- Help deep coughing and breathing exercises
- Give Chest physiotherapy every 4th hourly
- Provide Nebulised aerosol treatment [Nebulization].

Complications of the disease or therapy

This may include conditions such as pulmonary hypertension, hypoxemia, respiratoy failure, right heart failure. Some therapies may result in infection, muscle weakness and osteoporosis.

Health teaching tips

If as a critical nurse you are taking care of a patient with ILD. You should teach / counsel the patient regarding following measures:

- **Stop smoking:** There is an association between smoking and some types of idiopathic interstitial lung disease, the most severe and potentially lethal form of the disorder. Talk to your patient about options for quitting, including smoking cessation programs, which use a variety of proven techniques to help people quit.
- **Enroll in a pulmonary rehabilitation program:** These programs vary widely, but in general they focus on improving the ability to exercise and carry out normal activities, managing shortness of breath with breathing techniques, improving appetite and nutritional status, dealing with the difficult psychological aspects of living with lung disease, and improving overall quality of life.

- **Exercise regularly, as prescribed by your doctor:** Exercise is a double-edged sword for people with lung disease. It requires an increased intake of oxygen, and makes symptoms worse. At the same time, exercise is essential for maintaining lung function, reducing stress and depression, and maintaining overall health and well-being. Refer the patient to a pulmonary rehabilitation program before he starts or resumes exercising.

- **Eat well:** People with lung disease may lose weight both because it is uncomfortable to eat and because of the extra energy it takes to breathe. Yet a nutritionally rich diet that contains adequate calories is essential. The type of food your patient eats, the time of day he eats and the size of portions can all play a role in getting the nourishment he needs.

- Because it's often easier to breathe when the stomach isn't completely full, the patients may want to eat smaller meals throughout the day rather than two or three large ones. He might also try choosing lighter fare, such as fruit and salads, rather than rich or fatty foods, which take more energy to digest. A dietitian can give further guidelines for healthy eating.

- If the patient is overweight, losing weight to achieve a healthy body mass index (BMI) can have dramatic effect on ability to breathe and exercise tolerance.

Check Your Progress 1

i) Explain the meaning of IDL.

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ii) Classify ILD according to the cause.

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iii) List the clinical features of ILD.

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iv) Discuss the common treatment options.

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So far you have studied the interstitial lung diseases, it causes, classification and management, in the next section we shall focus on pneumonia.

5.3 PNEUMONIA

As you have already studied in basic training programme that Pneumonia is a major health problem. You may be aware of a variety of situations that pneumonia. They may be community or hospital acquired. Imposed immobility, depressed gag cause and cough reflex, increased secretions, decreased lymphatic flow, can all lead to pneumonia. We shall review it briefly.

5.3.1 Definition and Meaning

Pneumonia (pneumonitis) is an inflammatory process in lung parenchyma usually with a marked increase in interstitial and alveolar fluid.

It is a leading cause of death, particularly among older adults and people with debilitating disease. Pneumonia is the second largest cause of nosocomial infections.

5.3.2 Causes, Risk Factors and Types

Causes of pneumonia include following:

- **Bacteria:** Pneumococcal pneumoniae is the most common. Streptococcus pneumonia, staphylococcus aureus (hematogenous spread).
- **Viruses**: Influenza virus
- Mycoplasma
- **Fungal:** Histoplasmosis,aspergillosis
- **Protozoa:** Pneumocystis carinii.

Other causes

- Aspiration of food, fluid, or vomitus
- Inhalation of chemicals,dust,smoke.e.g., Mycoplasma pneumonia,and fungal pneumonia.

Risk factors

- Advanced age
- History of smoking
- Upper respiratory tract infection
- Tracheal intubation
- Prolonged immobility
- Immunosuppressive therapy e.g., Steroid, chemotherapy
- Malnutrition, Dehydration
- Chronic diseases like diabetes, heart failure
- Exposure to air pollution
- Altered consciousness.

Types of Pneumonia

The pneumonia can be classified on the basis of causative organism, location and extent and location in radiological appearance.

According to the causative organism

- Bacterial, viral, mycoplasmal (atypical), fungal, protozoal.

According to the location and extent

a) **Segmental pneumonia :** It involves one or more lobe segments of the lungs.

b) **Lobar pneumonia:** It involves one or more entire lobes.

c) **Bilateral pneumonia:** Involves lobes in both the lungs.

According to the location and radiological appearance

a) **Bronchopneumonia:** It involves the terminal bronchioles and alveoli.

b) **Interstitial or reticular pneumonia:** It is inflammatory responses within the lung tissue surrounding the air spaces or vascular structures.

c) **Alveolar (acinar) pneumonia:** In this Fluid accumulates in a distal air spaces.

d) **Necrotizing pneumonia:** It is death of the portion of the lung tissue surrounded by normal tissue.

5.3.3 Pathophysiology

The invading organism causes symptoms, in the part, by provoking an overtly exuberant immune response in the lungs. The small blood vessels in the lungs i.e., capillaries become leaky, and protein-rich fluid seeps into the alveoli. This results in a less functional area for oxygen-carbon dioxide exchange. The patient becomes relatively oxygen deprived, while retaining potentially damaging carbon dioxide. The patient breathes faster and faster, in an effort to bring in more oxygen and blow off more carbon dioxide.

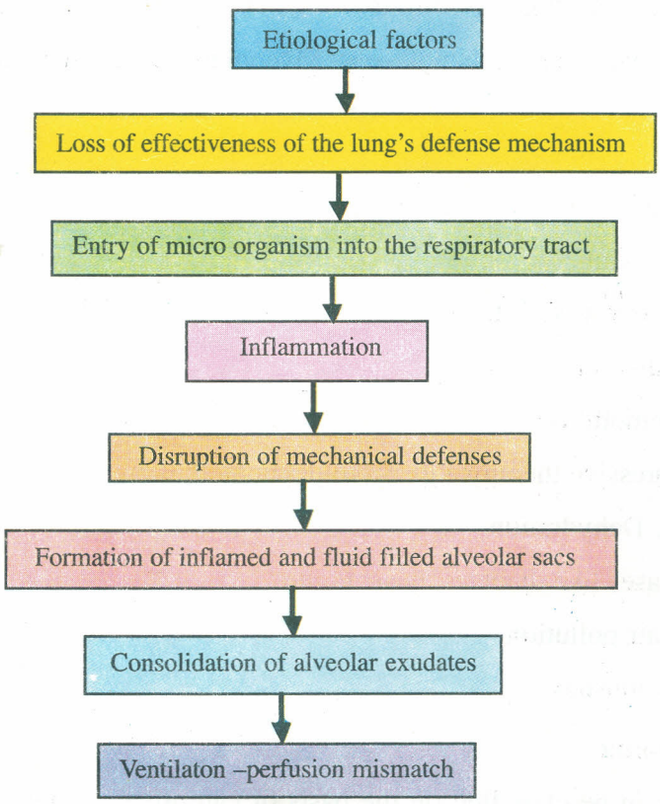

Fig. 5.2: Flow Chart of Pathological Changes

Mucus production is increased, and the leaky capillaries may tinge the mucus with blood. Mucus plugs actually further decrease the efficiency of gas exchange in the lung. The alveoli fill further with fluid and debris from the large number of white blood cells being produced to fight the infection. Hence consolidation, a feature of bacterial pneumonias, occur when the alveoli, which are normally hollow air spaces within the lung, instead become solid, due to quantities of fluid and debris. See fig. 5.2 flow chart for step wise changes.

Stage of Diseases:

There are 4 characteristic stages of the disease process.

Congestion: After the organisms reach alveoli via droplets or saliva, there is an outpouring of fluid into the alveoli. The organism multiply in the serous fluid and the infection is spread. It interfere with the lung function.

Red hepatization: There is massive dilation of the capillaries and alveoli are filled with organisms, neutrophils, RBC and fibrin. The lung appears red and granular, similar to the liver,which is why the process is called hepatization.

Gray hepatization: Blood flow decreases and leucocytes and fibrin consolidate in the affected part of the lung.

Resolution: Complete resolution and healing occur if there are no complications.

5.3.4 Clinical Manifestation and Diagnostics Measures

The Signs and Symptoms of pneumonia are given below:

- Cough with sputum production
- Dyspnoea, chest pain
- Decreased breath sounds and crackles over affected areas
- Dullness on percussion
- Fever, chillness, headache, fatigue.
- Advanced cases hemoptysis
- Unequal chest expansion during inspiration if a large area of lung tissue is involved.

Diagnostic measures

- History and physical examination
- **Blood investigation :** It shows elevated WBC count.
- **Sputum Culture and sensitivity :** To identify the causative organism.
- **Chest X- ray :** It helps identify the extent and pattern of the lung involvements and lung infiltration. Fluid consolidated lung tissue and atelectasis can also be identified.
- **Fibroptic bronchoscopy :** It is one to obtain a sputum specimen or remove secretions from the bronchi.
- **Pulse oximetry:** It is used to monitor arterial oxygen saturation. If it is less than 95 % it may indicate impaired gas exchange or ventilation.

Arterial blood gas analysis : May be done to evaluate gas exchange.

5.3.5 Therapeutic Management and Nursing Management

Treatment of pneumonia should include Specific antibiotic therapy to eradicate causative organism.

Antibiotic used for treatment of pneumonia include following according to the causative organisms.

Streptococcal pneumonia: Penicillin G, Erythromycin,Clindamycin

Staphylococcal pneumonia: Methicillin,Oxacillin,Vancomycin for MRSA.

Klebsiella: Gentamicin,Tobramicin, Cefotaxim.

Pseudomonas: Gentamicin, Piperacillin.

H.Influenza: Ampicillin, Amoxycillin.

Mycoplasma: Erythromicin,Tetracyclin.

Viral pneumonia: Symptomatic management.

Pneumocystis carinii: Pentmidine methane sulphonate.

Fungal pneumonia: Amphotericin B, Ketoconazole

Respiratory support

O2 administration: For severely hypoxic client. Endotracheal intubation and mechanical ventilation may be required to supply oxygen.

Bronchodilators: To reduce bronchospasm and improve ventilation.

Chest physiotherapy: When mucous secretions are thick and viscous or the cough is weak, percussion is done rhythmically striking or clapping the chest wall with cupped hands.Cupping traps the air between the palm and skin,causing vibrations that loosen respiratory secretions.

Postural drainage: It helps to remove secretions from a particular lung segment with the help of gravity. The client is positioned with the lung area to be drained above the trachea or mainstem bronchus.Drainage of all lung segments require a variety of positions.

Tracheal suctioning: May be required incase of tracheotomy.

Nutritional support: Provide small frequent meals, avoid gas forming foods, High caloric liquid supplement may be required in case of tracheotomy.

Fluid and electrolyte management: Increasing fluid intake to 2500 to 3000 ml/ day helps to liquefy secretions and making them easier to cough out and expectorate. If oral intake is inadequate I/V fluid may be required.

Immunization : Pneumococcal vaccine is recommended for people over the age of 65 years. Annual influenza vaccine is also recommended.

Surgical management: The following surgical procedures are performed

Lobectomy

Segmentectomy

Wedge resection

Pneumonectomy

Complications: The complication of pneumonia include following:

- Pleurisy
- Pleural effusion
- Atelectasis
- Delayed resolution
- Lung abscess
- Empyema
- Pericarditis
- Arthritis: It result from systemic spread of the organism.
- Meningitis: Can be caused by S.pneumoniae.

Nursing Management

Nursing Assessment:

- Monitor vital signs,skin colour
- Assess the level of consciousness and mental status
- Assess respiratory rate,O2 saturation,dyspnoea,cough,lung sounds.
- Monitor blood values, ABG values and report it to the physician.

Nursing Diagnosis:

- Ineffective airway clearance related to excessive secretions and weak cough or abnormal viscosity of mucus.
- Ineffective breathing pattern related to inflammatory process of the lung parenchyma as evidenced by tachypnoea.
- Impaired gas exchange related to ventilation / perfusion mis-match and intrapulmonary stunting.
- Acute pain related to inflammation of the lung parenchyma.
- Activity intolerance related to decreased O2 levels on metabolic demands.
- Imbalanced nutrition less than the body requirements related to increased metabolism, fatigue and anorexia.
- Risk for infection related to invasive devices used for therapeutic purpose.
- Power lessens related to lack of control over prognosis / threat to social, psychological integrity.

Nursing interventions are prioritized related to following :

- o Optimizing oxygenation and ventilation
- o Preventing spread of infection
- o Educating and supporting patients and Family members

Check Your Progress 2

i) What do you mean by pneumonia?

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ii) List 5 causes of pneumonia.

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iii) Write three risk factors of pneumonia.

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iv) Describe the main changes that taking place in pneumonia. Support your answer with the flow chart.

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v) Describe signs and symptoms of a patient with pneumonia.

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vi) Explain three classifications of pneumonia.

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vii) Describe the aspects of management.

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You have completed studying pneumonia its causes and management. Now we will move on it to pleural effusion.

5.4 PLEURAL EFFUSION

Healthy individuals have 20-25 ml of fluid in each pleural space. Normally, fluid enters the pleural space from the capillaries in the parietal pleura and is removed via the lymphatics situated in the parietal pleura. Fluid can also enter the pleural space from the interstitial spaces of the lung via the visceral pleura or from the peritoneal cavity via permeable membrane of the diaphragm. The lymphatics have the capacity to absorb 20 times more fluid than is normally formed but when the accumulation of fluid exceeds absorption pleural effusion results.

5.4.1 Definition and Meaning

Pleural effusion is excess fluid that accumulates in the pleural cavity, the fluid-filled space that surrounds the lungs, thereby restricting normal lung expansion.

5.4.2 Causes and Classification

The etiology of pleural effusion can be based on following:

- Transudates
- Exudates.

Exudative plleural effusion occurs when local factors that influence formation and absorption of pleural fluid are altered e.g., bacterial pneumonia, viral infection, malignance pulmonary embolism etc. A **transudative** pleural effusion occurs when systemic factors that influence the formulation and absorption are altered such as left ventricular failure and cirrhosis.

Let us now discuss about the causes of transudative and exudative pleural effusion.

The causes of transudative pleural effusions including following:

i) **Congestive Heart Failure:** This is the most common cause of pleural effusion. Frequently the effusions are bilateral (approximately 75% of the time) but may occur alone on either side with the right side being more common. Fluid is usually straw colored, with low white blood cell counts (<500 cells/mm3) and a mononuclear cell predominance. With severe congestive heart failure, fluid may persist in spite of vigorous diuresis.

ii) **Cirrhosis, Nephrotic Syndrome, and Hepatic Hydrothorax:** In disorders associated with low serum proteins and ascites, bilateral effusions are common. Cell counts are low and lymphocytes predominate. Glucose remains normal (>60 mg/dl). Hepatic hydrothorax occurs in about 5% of patients with ascites and cirrhosis. The effusion occurs (usually on the right side) because of direct movement of peritoneal fluid through communications in the hemidiaphragm.

Causes of exudative pleural effusion

The most common causes of exudative pleural effusions are parapneumonic (associated with pneumonia), malignancy, pulmonary embolism, trauma (including hemothorax and esophageal perforation), collagen vascular disease (especially rheumatoid arthritis), post-cardiac injury (including surgery), tuberculosis, trapped lung, and atelectasis.

- **Parapneumonic Effusion:** Bacterial pneumonias are frequently associated with pleural effusions and when they become complicated, require drainage. Complicated parapneumonic effusions include empyema (the finding of gross pus in the pleural space), those with positive pleural fluid cultures or Gram stains, and those in which the microbiology is negative but the patient continues to show signs of infection with fever, severe pleuritic pain and leukocytosis. In this last category the pleural fluid usually shows high white blood cell counts with polymorphonuclear predominance, glucose <30 mg/dl, and high LDH (>500 units/dl). Complicated parapneumonic effusions require drainage by tube thoracoscopy. The patient who has pneumonia with a small amount of pleural fluid present and is clinically responding to antibiotic therapy (afebrile, no pleuritic pain, normal white blood cell count) does not require thoracentesis. By contrast, rapid accumulation of pleural fluid in a patient with pneumonia is an indication for immediate thoracentesis.

- **Malignant Effusions:** Malignancy is the second most common cause of exudative pleural effusions with lung (36%), breast (25%) and lymphoma (10%) being the most frequent causes. Typical pleural fluid characteristics include a mononuclear predominant exudate (average 2500 cells/mm3), with an average red blood cell count of 40,000 cells/mm3, normal glucose (>60mg/dl) and positive cytology. At the time of diagnosis one-third of patients have a low pleural fluid glucose (<60mg/dl), which is associated with more extensive disease and a poorer prognosis.

- **Effusion Secondary to Pulmonary Embolism:** These exudative effusions are usually bloody, and associated with pleurisy and dyspnea. The effusion may increase in size the first 24-48 hours after initial anticoagulation. Unless there is significant pulmonary compromise, or the effusion continues to

increase, these effusions can be observed. There are reports of transudative effusions associated with pulmonary embolism, but atelectasis secondary to splinting from pleurisy is a more likely cause.

- **Tuberculous Effusion:** Typically, this predominantly lymphocytic exudate is devoid of mesothelial cells and may occur without any obvious parenchymal involvement. The glucose may be low (<60 mg/dl) and adenosine deaminase levels are usually elevated (>70 IU/l). Historically, in the non-immunocompromised host, pleural fluid smears are rarely positive but pleural fluid cultures are positive in 25%.

- **Miscellaneous:** Atelectasis is a common cause of small to moderate effusions. Frequently they are seen postoperatively or with prolonged bed rest and inactivity. There are no unique diagnostic features and these effusions usually fit exudative criteria, have normal glucose levels, and WBC counts of 1000 to 2000 cells/mm3 with mononuclear cell predominance. Transudates may occur with atelectasis.

Let us see what types of fluids accumulate in the pleural spaces in case of pleurisy

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Types of fluid that accumulates in the pleural space.

Four types of fluids can accumulate in the pleural space:

- Serous fluid (hydrothorax)
- Blood (hemothorax)
- Chyle (milky white lymphatic fluid) (chylothorax)
- Pus (pyothorax or empyema)

Refer unit 2 chest trauma for the detailed explanation.

5.4.3 Pathophysiology

Normally, fluid enters the pleural space from the capillaries in the parietal pleura, from interstitial spaces of the lung via the visceral pleura, or from the peritoneal cavity through the semipermable membrane of the diaphragm. This fluid is normally removed by lymphatics in the parietal pleura, which have the capacity to absorb 20 times more fluid than is normally formed. When this capacity is overwhelmed, either through excess formation or decreased lymphatic absorption, a pleural effusion develops.

5.4.4 Clinical Manifestations and Diagnostic Measures

Symptoms depend on the size of the effusion and the rapidity of the development. Small effusion in a healthy person may not get clinically manifested.

- Pleural effusion with increasing size will cause respiratory distress compressing and restricting airflow and finally causing meditational shift and compromised cardiac function.
- If fluid is present Chest X-ray will show following changes:
    - Blunting of costophrenic angle
    - Flattening of diaphragm
    - Obscuring of cardiac borders due to Pneumothorax

- Pleural edges evident through image of ribs

5.4.5 Therapeutic Management and Nursing Management

Treatment of pleural effusion includes following :

- Respiratory support
- Resolution of underlying cause
- Hydrothorax with congestive heart failure to be treated with diuretics.

Hydrothorax due to inflammatory responses to be reduce with anti inflammatory agents.

Drainage of hemothorax, chlothorax, emphyema thoracentesis.

Repeated effusions may require chemical (talcbleomycin, and tetracyline/ doxycycline) or surgical pleuraodesis, in which the two pleural surfaces are attached to each other so that no fluid can accumulate between them. Detailed management of different causes have been discussed in unit 2 under the different conditions.

Complication of pleural effusion

The complications are given in the following fig. 5.3.

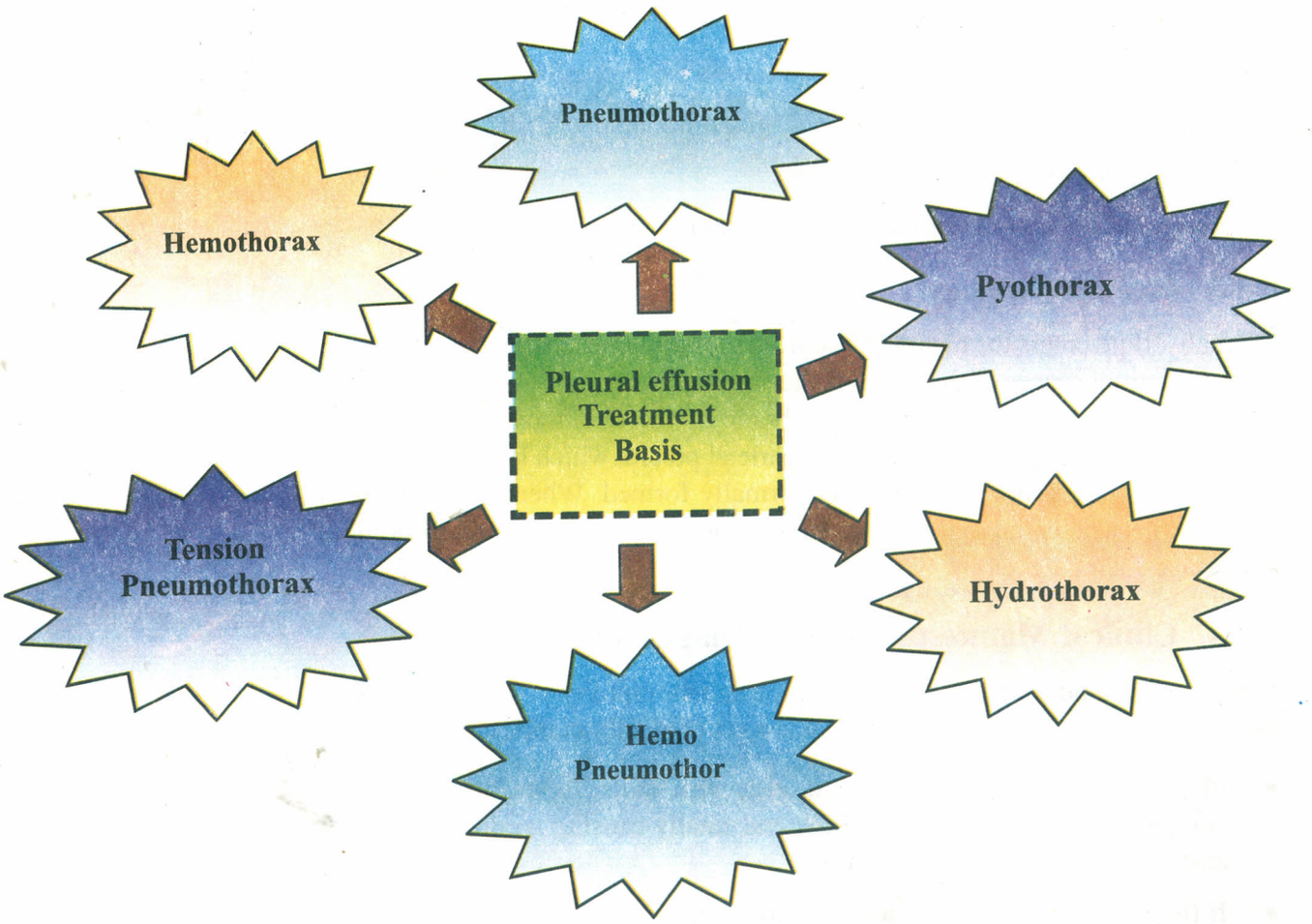

Fig. 5.3: Complications of pleural effusion

Check Your Progress 3

i) List the conditions where Pleural effusions occur.

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ii) Explain the types of pleural effusion.

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iii) List clinical features of pleural effusion.

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5.5 LET US SUM UP

Pulmonary system plays an important role in our life. The proper functioning of the system delivers gas exchange. ILD is the group of disorder which alters the normal gas exchange. The symptoms and course of these diseases may vary from person to person, but the common link between the many forms of ILD is that they all begin with an inflammation. The various diagnostic as well as treatment procedures are used to assess and maintain respiratory status.

Pneumonia is inflammation of respiratory bronchioles and alveoli. It require intense medical and nursing care.Client needs rest and supportive management and awarness about the importance of follow up to ensure cure of the disease.

Pleural effusions are associated with many systemic disorders. Thoracentesis is done to determine whether the pleural fluid is a transudate or an exudate coupled with other appropriate diagnostic studies helps in diagnosing the problem. Because pleural fluid findings are often nonspecific (except for positive cytology and bacteriology), clinical correlation and response to therapy are critical.

In this unit we have discussed in detail about the meaning causes, classification, pathophysiology and nursing management of interstitial lung diseases pneumonia and pleural effusion.

5.6 GLOSSARY

Sarcoidosis : Also called sarcoid or Besnier-Boeck disease, is a multisystem disorder characterized by non-caseating granulomas (small inflammatory nodules). The cause of the disease is still unknown. Virtually any organ can be affected; however, granulomas most often appear in the lungs or the lymph nodes. Symptoms usually appear gradually but can occasionally appear suddenly. The clinical course generally varies and ranges from asymptomatic disease to a debilitating chronic condition that may lead to death.

Pneumocystis pneumonia : Pneumocystis pneumonia (PCP) or pneumocystosis is a form of pneumonia, caused by the yeast-like fungus Pneumocystis jirovecii. This pathogen is specific to humans. Pneumocystis is commonly found in the lungs of healthy people, but being a source of opportunistic infection it can cause a lung infection in people with a weakened immune system. Pneumocystis pneumonia is especially seen in people with cancer, HIV/AIDS and the use of medications that affect the immune system.

Silicosis : (also known as Potter's rot) is a form of occupational lung disease caused by inhalation of crystalline silica dust, and is marked by inflammation and scarring in forms of nodular lesions in the upper lobes of the lungs.

Berylliosis, or chronic beryllium disease (CBD), is an occupational lung disease that is most classically associated with exposure to fluorescent lighting and missile silos. It is a chronic allergic-type lung response and chronic lung disease caused by exposure to beryllium and its compounds. The condition is incurable, but symptoms can be treated.

Exudates : An exudates is any fluid that filters from the circulatory system into lesions or areas of inflammation.

Transudates : Transudates is extravascular fluid with low protein content and a low specific gravity (< 1.012). It has low nucleated cell counts (less than 500 to 1000 /microlit) and the primary cell types are mononuclear cells: macrophages, lymphocytes and mesothelia cells.

Asbestosis : It is a chronic inflammatory medical condition affecting the parenchymal tissue of the lungs. It occurs after long-term, heavy exposure to asbestos, e.g., in mining, and is therefore regarded as an occupational lung disease. Sufferers have severe dyspnea (shortness of breath) and are at an increased risk regarding several different types of lung cancer.

Pleural fremitus : It is a palpable vibration of the wall of the thorax caused by friction between the parietal and visceral pleura of the lungs.

Subjective fremitus : It is a vibration felt by the patient on humming with the mouth closed.

Tactile Fremitus : Known by many other names including pectoral fremitus, tactile vocal fremitus, or just vocal fremitus, is a vibration felt on the patient's chest during low frequency vocalization. Commonly, the patient is asked to repeat a phrase while the examiner feels for vibrations by placing a hand over the patient's chest or back. Phrases commonly used in English include 'ninety-nine', 'boy oh boy', 'toy boat', and 'blue balloons'.

Vocal resonation : It is the process by which the basic product of phonation is enhanced in timbre and/or intensity by the air-filled cavities through which it passes on its way to the outside air. Various terms related to the resonation process include amplification, enrichment, enlargement, improvement, intensification, and prolongation, although in strictly scientific usage acoustic authorities would question most of them. The main point to be drawn from these terms by a singer or speaker is that the end result of resonation is, or should be, to make a better sound.

5.7 ANSWER TO CHECK YOUR PROGRESS

Check Your Progress 1

i) Interstitial Lung (IDL)

IDL refers to a group of lung diseases affecting the interstitium of the lung, alveolar epithelium, pulmonary capillary endothelium, basement membrane, perivascular and perilymphatic tissues. The term ILD is used to distinguish these diseases from obstructive airways diseases. Also known as diffuse parenchymal lung disease (DPLD) and interstitial pulmonary fibrosis. Interstitial lung diseases is a group of diffuse inflammatory lower respiratory

tract disorders, characterized by accumulation of inflammatory cells in the interstitium of the alveolar walls followed by thickened, fibrotic changes.

ii) **ILD may be classified according to the cause as follows:**

Due to Inhaled substances

Inorganic

- Silicosis
- Asbestosis
- Berylliosis

Organic

- Hypersensitivity pneumonitis

Drug induced

- Antibiotics
- Chemotherapeutic drugs
- Antiarrhythmic agents

Connective tissue disease

- Systemic sclerosis
- Dermatomyositis
- Systemic lupus erythematosus
- Rheumatoid arthritis

Infection

- Atypical pneumonia
- Pneumocystis pneumonia (PCP)
- Tuberculosis

Idiopathic

- Sarcoidosis A
- Idiopathic pulmonary fibrosis
- Hamman-Rich syndrome

Malignancy

- Lymphangitic carcinomatosis

iii)

- Fatigue
- Progressive dyspnea
- Non productive cough
- Dyspnea at rest
- Clubbing of the finger tips
- Shortness of breath, especially with exertion
- Weakness
- Loss of appetite

- Loss of weight
- Discomfort in chest
- Labored breathing
- Hemorrhage in lungs

iv) The Common Treatment Options

Medications

- **Corticosteroids (Antifibrotic / Anti-Inflammatory Drugs)** Inflammation is controlled with corticosteroids - Prednistone.

- As the disorder progresses inhaled corticosteroids and bronchodilator help to mobilize the secretions.

- **Cytotoxic Agents** or Immunosuppressive drugs may be used if steroid therapy has failed to be effective or if corticosteroid treatment is not possible. In some cases, a combination of a corticosteroid and cyclophosphamide is used with good results. This medication reduces inflammation by killing some inflammatory cells and suppressing their function. Response to therapy may be slow and require up to 6 months or longer.

- **Azathioprine** [Imuran] used in combination with corticosteroids for the treatment of ILD.

- **Mycophenolate** [CellCept]. It help reduce the amount of steroids required. It works to prevent the immune system from attacking cells in the body that result in fibrosis.

Oxygen Therapy

Oxygen therapy is required for some patients with ILD. Decreased lung function and/or pulmonary hypertension may cause blood oxygen levels to be too low. Some may need oxygen therapy all the time while others may need it only during sleep and exercise.

Lung transplantation

If the above therapies fail to adequately treat ILD, lung transplant is an option for some advanced cases. With improved surgical techniques and post-transplant care, transplantation may offer improved quality of life and prolonged survival to selected patients.

Check Your Progress 2

i) Pneumonia (pneumonitis) is an inflammatory process in lung parenchyma usually with a marked increase in interstitial and alveolar fluid.

It is a leading cause of death, particularly among older adults and people with debilitating disease. Pneumonia is the second largest cause of nosocomial infections.

ii) Causes of Pneumonia

Bacteria: Pneumococcal pneumoniae is the most common. Streptococcus pneumonia, staphylococcus aureus (hematogenous spread).

Viruses: Influenza virus

Mycoplasma

Fungal: Histoplasmosis,aspergillosis

Protozoa: Pneumocystis carinii.

iii) Risk Factor

Advanced age

History of smoking

Upper respiratory tract infection

iv) Flow Chart

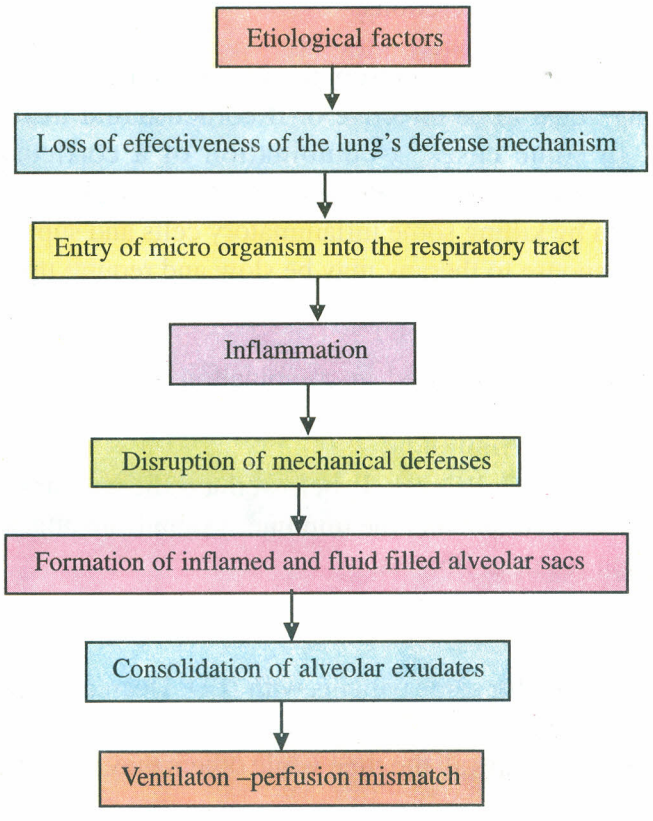

Fig. 5.2: Flow Chart of Pathological Changes

Stage of Diseases:

There are 4 characteristic stages of the disease process.

Congestion: After the organisms reach alveoli via droplets or saliva, there is an outpouring of fluid into the alveoli. The organism multiply in the serous fluid and the infection is spread. It interfere with the lung function.

Red hepatization: There is massive dilation of the capillaries and alveoli are filled with organisms, neutrophils, RBC and fibrin. The lung appears red and granular, similar to the liver,which is why the process is called hepatization.

Gray hepatization: Blood flow decreases and leucocytes and fibrin consolidate in the affected part of the lung.

Resolution: Complete resolution and healing occur if there are no complications.

v) The Signs and Symptoms of pneumonia are given below:

- Cough with sputum production

- Dyspnoea, chest pain
- Decreased breath sounds and crackles over affected areas
- Dullness on percussion
- Fever, chillness, headache, fatigue.
- Advanced cases hemoptysis
- Unequal chest expansion during inspiration if a large area of lung tissue is involved.

vi) According to the causative organism

- Bacterial, viral, mycoplasmal (atypical), fungal, protozoal.

According to the location and extent

a) **Segmental pneumonia :** It involves one or more lobe segments of the lungs.

b) **Lobar pneumonia:** It involves one or more entire lobes.

c) **Bilateral pneumonia:** Involves lobes in both the lungs.

According to the location and radiological appearance

a) **Bronchopneumonia:** It involves the terminal bronchioles and alveoli.

b) **Interstitial or reticular pneumonia:** It is inflammatory responses with in the lung tissue surrounding the air spaces or vascular structures.

c) **Alveolar (acinar) pneumonia:** In this Fluid accumulates in a distal air spaces.

d) **Necrotizing pneumonia:** It is death of the portion of the lung tissue surrounded by normal tissue.

vii) Respiratory support

O2 administration: For severely hypoxic client. Endotracheal intubation and mechanical ventilation may be required to supply oxygen.

Bronchodilators:- To reduce bronchospasm and improve ventilation.

Chest physiotherapy: When mucous secretions are thick and viscous or the cough is weak, percussion is done rhythmically striking or clapping the chest wall with cupped hands.Cupping traps the air between the palm and skin,causing vibrations that loosen respiratory secretions.

Postural drainage: it helps to remove secretions from a particular lung segment with the help of gravity. The client is positioned with the lung area to be drained above the trachea or mainstem bronchus.Drainage of all lung segments require a variety of positions.

Tracheal suctioning: may be required in case of tracheotomy.

Nutritional support: Provide small frequent meals, avoid gas forming foods, High caloric liquid supplement may be required in case of tracheotomy.

Fluid and electrolyte management: Increasing fluid intake to 2500 to 3000 ml/day helps to liquefy secretions and making them easier to cough out and expectorate .If oral intake is inadequate I/V fluid may be required.

Immunization: Pneumococcal vaccine is recommended for people over the age of 65 years. Annual influenza vaccine is also recommended.

Surgical management: The following surgical procedures are performed

Lobectomy

Segmentectomy

Wedge resection

Pneumonectomy

Check Your Progress 3

i) Congestive heart failure cirrhosis, Nephrotic syndrome hepatic hydrothorax malignancy etc.

ii)
- Transudates
- Exudates.
- **Exudative** plleural effusion occurs when local factors that influence formation and absorption of pleural fluid are altered e.g., bacterial pneumonia, viral infection, malignance pulmonary embolism etc. A transudative pleural effusion occurs when systemic factors that influence the formulation and absorption are altered such as left ventricular failure and cirrhosis.

iii)
- Pleural effusion with increasing size will cause respiratory distress compressing and restricting airflow and finally causing meditational shift and compromised cardiac function.

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NOTES

BNS-032 Nursing Management in Critical Care Conditions

Block 1 Care of Critically ill Patients with Problems of Gastrointestinal System

Unit 1 Acute Gastrointestinal bleeding
Unit 2 Acute intestinal obstruction, Intra abdominal Compartment Syndrome, Perforative Peritonitis.
Unit 3 Hepatic disorders – Fulminent hepatic failure and hepatic encephalopathy
Unit 4 Acute Pancreatitis

Block 2 Care of Critically Ill Patients with Problems of Respiratory System
Unit 1 Acute Respiratory Distress Syndrome
Unit 2 Chest trauma (Hemothorax, Pneumothorax Chylothorax, Pyothorax, Hydrothorax)
Unit 3 Pulmonary Edema, Pulmonary Embolism, Atelectasis
Unit 4 Acute exacerbation of Chronic Obstructive Pulmonary Diseases And Status Asthmatics
Unit 5 Interstitial Lung Disease, Pneumonia, Pleural Effusion

Block 3 Care of Critically Ill Patients with Problems of Cardiothoracic System
Unit 1 Acute Coronary Syndrome
Unit 2 Hypertensive Crisis
Unit 3 Cardiac dysrrhythemia and cardiac arrest
Unit 4 Conductive Disturbances and Heart Block (Pacemaker)
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Block 4 Care of Critically Ill Patients with Problems Of Nervous System
Unit 1 Altered Sensorium And Increased Intracranial Pressure (↑ ICP)
Unit 2 Cerebro-vascular Accident (Stroke)
Unit 3 Traumatic Brain injury, Spinal Cord Injury and Statin Epileptics[
Unit 4 Myasthenic Crisis Multiple Scterosis And Guillain Barre Syndrome

Block 5 Care of critically ill patients with problem of renal and endocrine system
Unit 1 Care of Patients With Renal Transplantation
Unit 2 Acute Tubular Necrosis And Bladder Trauma
Unit 3 Diabetic Ketoacidosis
Unit 4 Thyroid crisis, Myxedema coma, Adrenal crisis and Syndrome of inappropriate hypersecretion of anti diuretic hormone (SIADH))

Block 6 Care Of Critically Ill Patients With Various Emergency Conditions
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Unit 2 Poly Trauma (Multiple Organ Failure /organ transplantation/ triage/coding)
Unit3 Shock, Septicemia, Multiple Organ Dysfunction Syndrome, Disseminated Intravascular Coagulation, Status Epilepticus
Unit 4 Drug Over Dose And Poisoning, Anaphylaxis
Unit 5 Drowning

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