Early Historical Landmark / History of COPD
3: EARLY HISTORICAL LANDMARK / HISTORY OF COPD

Some of the earliest references to the description of emphysema include: Bonet’s description of “voluminous lungs” in 1679; According to Morgagni’s (1769) description the lungs were “turgid”. The chronic bronchitis component of COPD can be traced to Badham (1814), who used the word catarrh to refer to the chronic cough and mucus hyper secretion that are cardinal symptoms. He described bronchiolitis and chronic bronchitis as disabling disorders. The Laennec described emphysematous lung are hyper inflated and not emptying well. John Hutchinson who invented spirometer in 1846 was found to be a main factor for the diagnosis of COPD. After 100 years of Hutchinson’s instrument, Tiffeneau added the concept of timed vital capacity as a measure of airflow, for spirometry to become complete as a diagnostic instrument (Tiffeneau and Pinelli 1947). Osler’s believed emphysema was caused by excessive pressure in the alveoli. Gaensler introduced the concept of the air velocity index based on Tiffeneau’s work and later the forced vital capacity, which is the foundation of the FEV$_1$ and FEV$_1$/FVC percent$^{(35)}$.

In 1944, one of the great teachers of emphysema, Ronald Christie, suggested that “The diagnosis should be considered certain when dyspnoea on exertion, of insidious onset, not due to bronchospasm, or left ventricular failure, appears in a patient who has some physical signs of emphysema together with chronic bronchitis and asthma”. It is clear from this statement that Christie recognized the individual components of COPD and relied on the history and physical examination for his diagnosis. Oswald described the clinical features of 1000 cases of chronic bronchitis in 1953 (Oswald et al 1953). Barach and Bickerman (1956) described the treatment for COPD in their book, *pulmonary emphysema*, which nicely describes the treatment in that era. These two physicians were early champions in treatment for emphysema$^{(35)}$.
The Dayman, was first to recognize the spirometry and flow volume patterns indicative of dynamic expiratory airway collapse in emphysema; Dickerson Richards, wrote on the pulmonary circulation and cor pulmonale; Reuben Cherniack, who described respiratory acidosis and has made major contributions to our understanding of the diagnosis and treatment of emphysema for over half a century; and Menelee and Callaway, who described pulmonary function tests in emphysema patients. The first edition of Hinshaw and Garland (1956) *Textbook of respiratory medicine* demonstrates airflow limitation in emphysema (35).

Two landmark meetings: The CIBA Guest Symposium in 1959 and the American Thoracic Society Committee on Diagnostic Standards in 1962 defined the components of COPD, which are the foundation for our definitions today. The American Thoracic Society (ATS) defined chronic bronchitis in clinical terms including chronic cough lasting at least three months for at least two years. By contrast, the ATS defined emphysema in anatomic terms of enlarged alveolar spaces and loss of alveolar walls. Many other attempts to define COPD have not improved on these basic definitions, except that COPD is now defined in functional terms. Other acronyms that predated the COPD designation were chronic obstructive bronchopulmonary disease, chronic airflow obstruction, chronic obstructive lung disease, nonspecific chronic pulmonary disease, and diffuse obstructive pulmonary syndrome. Briscoe and Nash 1965 believed to be the first persons to use the term COPD in discussion at the 9th Aspen Emphysema Conference. This term became established and today we refer to COPD as the designation of this growing health problem (35).
3.1: OVERVIEW AND DEFINITIONS

Chronic obstructive pulmonary disease (COPD), also known as chronic obstructive lung disease (COLD), and chronic obstructive airway disease (COAD), among others, is a type of obstructive lung disease characterized by chronically poor airflow. It typically worsens over time. The main symptoms include shortness of breath, cough, and sputum production \(^{(36)}\). Most people with chronic bronchitis have COPD \(^{(37)}\).

The term chronic obstructive pulmonary disease, abbreviated COPD, or sometimes COLD (chronic obstructive lung disease), refers to a disease state characterized by the presence of airflow obstruction resulting from chronic bronchitis or emphysema. As defined in the recent American Thoracic Society guidelines statement regarding COPD “COLD (chronic obstructive lung disease) is a treatable disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking. Although COPD affects the lungs, it also produces significant systemic consequence” \(^{(38)}\).

Similarly, the recent Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines as “A disease state characterized by airflow limitation that is not fully reversible, is abnormal inflammatory response of the lungs to noxious particles or gases” \(^{(28)}\).

3.2: ETIOLOGY

Tobacco smoking is the most common cause of COPD, with a number of other factors such as air pollution and genetics playing a smaller role \(^{(39)}\). In our country, bidi smoking is an important factor in addition to cigarette smoking that causes COPD \(^{(40)}\).
It also appears that outdoor air pollutants are significant environmental triggers for AECOPD and chronic exposure to the traffic exposure affects the respiratory health conditions, from increasing symptoms to emergency department visits, hospital admissions and even mortality. Improving ambient air pollution and decreasing indoor biomass combustion exposure by improving home ventilation appear to be effective interventions that could substantially benefit the health of the general public \(^{(41)}\) \(^{(42)}\).

In the developing world, one of the common sources of air pollution is from poorly vented cooking and heating fires. Long-term exposure to these irritants causes an inflammatory response in the lungs resulting in narrowing of the small airways and breakdown of lung tissue known as emphysema \(^{(43)}\). The diagnosis is based on poor airflow as measured by lung function tests. In contrast to asthma, the airflow reduction does not improve significantly with the administration of medication \(^{(44)}\).

COPD can be prevented by reducing exposure to the known causes. This includes efforts to decrease rates of smoking and to improve indoor and outdoor air quality. COPD treatments include: quitting smoking, vaccinations, rehabilitation, inhaled bronchodilators and steroids. Some people may benefit from long-term oxygen therapy or lung transplantation \(^{(43)}\). In those who have periods of acute worsening, increased use of medications and hospitalization may be needed \(^{(43)}\).
The spectrum of COPD is shown in figure below:

![Venn Diagram of COPD Subsets](image)

**Figure 19: Schema of COPD with subset**

This non proportional Venn diagram shows subsets of patients with chronic bronchitis, emphysema, and asthma. This subsets composing COPD are shaded. Subset areas are not proportional to actual relative subset sizes. Asthma is by definition associated with reversible airflow obstruction, although in variant asthma special maneuvers may be necessary to make the obstruction evident.

Patients with asthma whose airflow obstruction is completely reversible (subset 9) are not considered to have COPD. Because in many cases it is virtually impossible to differentiate patients with asthma whose airflow obstruction does not remit completely from persons with chronic bronchitis and emphysema who have partially reversible airflow obstruction with airway hyper reactivity, patients with unremitting asthma are classified as having COPD (subsets 6, 7 and 8). Chronic bronchitis and emphysema with airflow obstruction usually occur together (subset 5) and some patients may have asthma associated with
these two disorders (subset 8). Individuals with asthma who are exposed to chronic irritation, as from cigarette smoke, may develop a chronic, productive cough, a feature of chronic Bronchitis (subset 6). Such patients are often referred to as having asthmatic bronchitis and/or the asthmatic form of COPD. Persons with chronic bronchitis and/or emphysema without airflow obstruction (subset 1, 2 and 11) are not classified as having COPD. Patients with airway obstruction caused by diseases with known etiology or specific pathology, such as cystic fibrosis or obliterative bronchiolitis (subset 10), are not included in this definition (28).

3.3: BURDEN OF THE COPD

COPD is a leading cause of morbidity and mortality worldwide and results in an economical and social burden that is both substantial and increasing. There are wide variations in the prevalence of COPD in India subcontinent. Based on this the national burden of chronic bronchitis was estimated as 14.84 million (45) (46) (47).

Chronic obstructive pulmonary disease (COPD) is a name coined for the diseases that were previously known as chronic bronchitis and emphysema. The British Medical Research Council (BMRC) defined chronic bronchitis as “daily productive cough for at least three consecutive months for more than two successive years. American Thoracic Society (ATS) in 1962 defined emphysema as an “anatomic alteration of the lung characterized by an abnormal enlargement of the air spaces distal to the terminal, non-respiratory bronchiole, accompanied by destructive changes of the alveolar walls”. The definition of emphysema put forth by the National Heart, Lung and Blood Institute in 1984 is as “a condition of the lung characterized by abnormal, permanent enlargement of airspaces distal to the terminal bronchiole, accompanied by the destruction of their walls, and without obvious fibrosis”. Reid reported that “the diagnosis of emphysema by
itself is incomplete unless it is taken into account the presence or absence of chronic bronchitis and vice versa”. McDonough et al have recently reported extensive obliteration of terminal bronchioles in patients with COPD who have emphysema, suggesting that “the permanent enlargement of the distal airspaces may serve only as a structural biomarker, being a secondary result of small airway inflammation and destruction”. Thus, COPD has both airway (central and small airways) and airspace abnormalities\(^{(48)}\)\(^{(49)}\).

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) recently defined COPD as “a common preventable and treatable disease characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and co morbidities contribute to the overall severity in individual patient”. It is worthwhile to mention that William Osler had described in 1892 in his Textbook of Medicine hypertrophic emphysema as “a well-marked clinical affection, characterized by enlargement of the lungs due to distension of the air cells and atrophy of their walls, and clinically by imperfect aeration of the blood and more or less dyspnœa” a beautiful clinical description of emphysema\(^{(47)}\). The structure of the normal lung and emphysematous lung is shown below\(^{(50)}\).

\[\text{Figure 20: Normal and emphysematous lung}\]
3.4: PREVALENCE OF COPD GLOBALLY AND IN INDIA

The prevalence and morbidity data greatly underestimate the total burden of COPD because the disease is usually not diagnosed until it is clinically apparent and moderately advanced\(^{(51)}\).

Globally, as of 2010, COPD affected approximately 329 million people (4.8\% of the population) and is slightly more common in men than women.\(^{(52)}\) This is as compared to 64 million being affected in 2004\(^{(53)}\).

By 2020, COPD is predicted to be the third leading cause of death and fifth leading cause of chronic disability worldwide\(^{(54)}\).

The current prevalence of COPD in India is unclear because of variation in study design and lack of adherence to strict protocol of the definition of COPD\(^{(55)}\).

The mean rates in India have not really changed when compared for different time periods. But the total burden of COPD has more than doubled to about 14.84 million in 2011 from about 6.45 million in 1971. This is generally attributable to the overall increase in the population of India\(^{(56)}\).

3.5: DIAGNOSIS

A clinical diagnosis of COPD should be considered in any patients who have dyspnoea, chronic cough or sputum production and history of exposure to risk factors for the diseases.
**Table 1: Key features for considering a diagnosis of COPD**

<table>
<thead>
<tr>
<th>Key indication for considering a diagnosis of COPD</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnoea that is</td>
<td>• Progressive (worsen over time) characteristic worsen with exercise • Persistent</td>
</tr>
<tr>
<td>Chronic cough</td>
<td>May be intermittent and may be non-productive</td>
</tr>
<tr>
<td>Chronic sputum production</td>
<td>Any pattern of chronic sputum production may indicate COPD</td>
</tr>
<tr>
<td>History of exposure to risk factors</td>
<td>• Tobacco, smoking • Smoking from home cooking and heating fuels • Occupational dust and chemicals</td>
</tr>
<tr>
<td>Family history of COPD</td>
<td></td>
</tr>
</tbody>
</table>

Spirometry is required to make the diagnosis in this clinical context, the presence of a post bronchodilator FEV$_1$/FVC <0.70 confirms the presence of persistent airflow limitation and thus COPD. Spirometry criterion for airflow limitation remains a post bronchodilator fixed ratio of FEV$_1$/FVC <0.70. This criterion is simple, independent of reference value and has been used in numerous clinical trials forming the evidence base from which most of our treatment recommendation is drawn$^{(57)}$.

Post bronchodilator spirometry is required for the diagnosis and assessment of severity of COPD, the degree of reversibility of airflow limitations no longer recommended. The degree of reversibility has never been shown to add to the diagnosis, differential diagnosis with asthma or to predicting the reference to long term treatment with bronchodilator and corticosteroids$^{(58)}$. 

29
Here flow volume loop of normal, obstructive and restrictive diseases are shown \(^{(59)}\).

*Figure 21: Flow volume curve during inspiration and expiration*
Figure 22: **Flow volume curve in normal person**

![Flow volume curve in normal person](image)

Figure 23: **Flow volume curve in normal and person with obstruction**

![Flow volume curve in normal and person with obstruction](image)

Figure 24: **Flow volume curve in normal and person with restriction**

![Flow volume curve in normal and person with restriction](image)
3.6: SYMPTOMS

The characteristic symptoms of COPD are chronic and progressive dyspnoea, cough and sputum production that can be variable from day to day. Chronic cough and sputum production may precede the development of airflow limitation by many years. Conversely significant airflow limitation may develop without chronic cough and sputum production (60).

In COPD patients breathing pattern is vary like some patients breathe slowly and deeply whereas some breathe shallow and rapidly (61).

3.6.1: DYSPNOEA:

Dyspnoea, a cardinal symptom of COPD, is a major cause of disability and anxiety associated with the disease. Typical COPD patients describe their dyspnoea as a sense of increased efforts to breathe, heaviness, air hunger or gasping. Here is some classification of dyspnoea, as it is useful to find out severity of disease.

*Table 2: MRC scale for breathlessness (62)*

<table>
<thead>
<tr>
<th>Grade</th>
<th>Degree of dyspnoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No dyspnoea except with strenuous exercise</td>
</tr>
<tr>
<td>1</td>
<td>Dyspnoea when walking up an incline or hurrying on the level</td>
</tr>
<tr>
<td>2</td>
<td>Walks slower than most on the level, or stops after 15 minutes of walking on the level</td>
</tr>
<tr>
<td>3</td>
<td>Stops after a few minutes of walking on the level</td>
</tr>
<tr>
<td>4</td>
<td>With minimal activity such as getting dressed, too dyspneic to leave the house</td>
</tr>
</tbody>
</table>
Table 3: Grades of dyspnoea classification by NYHA (Newyork Heart Association)

<table>
<thead>
<tr>
<th>Grades of dyspnoea</th>
<th>Level of activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1 (minimal dyspnoea)</td>
<td>Dyspnoea on running or on doing more than ordinary effort</td>
</tr>
<tr>
<td>Grade 2</td>
<td>On doing ordinary activity</td>
</tr>
<tr>
<td>Grade 3 (considerable dyspnoea)</td>
<td>On doing less than ordinary activity</td>
</tr>
<tr>
<td>Grade 4</td>
<td>On rest</td>
</tr>
</tbody>
</table>

3.6.2: COUGH:

Chronic cough, often the first symptoms of COPD to develop, is frequently discounted by the patient as an expected consequence of smoking and/or environment exposure. Initially, the cough may be intermittent but later on it is present on every day, often throughout the day. The chronic cough in COPD may be nonproductive. In some cases, significant airflow limitation may develop without the presence of a cough (63).

3.6.3: SPUTUM PRODUCTION:

COPD patients commonly raise small quantities of tenacious sputum after coughing bouts. Regular production of sputum for 3 or more months in 2 consecutive years is the epidemiological definition of chronic bronchitis. Patients produce large amount of sputum may have underlying broncheactesis (63).

3.6.4: WHEEZING AND CHEST TIGHTNESS:

Wheezing and chest tightness are nonspecific symptoms that may vary between days and over a course of a single day. Chest tightness often follows exertion, is poorly localized, is muscular in character, and may arise from the isometric contraction of the intercostals muscles, absence of wheezing or chest tightness does not exclude a diagnosis of COPD nor does the presence of these symptoms confirm a diagnosis (63).
Additional features in severe disease fatigue, weight loss and anorexia are common problems in patients with severe and very severe COPD. Cough syncope occurs due to rapid increase in intrathoracic pressure during prolong attacks of coughing. Coughing spells may also cause rib fracture which are sometimes asymptomatic. Ankle swelling may be the only symptomatic pointer to the development of cor pulmonale. Symptom of depression and or anxiety merit specific inquiry in the clinical history because they are common in COPD and are associated with increased risk of exacerbation and poor health status\textsuperscript{(63)}.

### 3.7: CLASSIFICATION OF SEVERITY OF COPD

The global initiative for chronic obstructive lung disease (GOLD) has proposed a universal guideline for the classification of COPD on the basis of both spirometry and clinical symptoms to define stage of the disease\textsuperscript{(63)}.

**Table 4: Classification of severity of COPD**

<table>
<thead>
<tr>
<th>UNIVERSAL GUIDELINE FOR THE CLASSIFICATION OF DISEASE</th>
<th>PREDICTED FEV\textsubscript{1}/FVC</th>
<th>FEV\textsubscript{1}</th>
</tr>
</thead>
<tbody>
<tr>
<td>MILD COPD</td>
<td>&lt;70%</td>
<td>&gt;80%</td>
</tr>
<tr>
<td>MODERATE COPD</td>
<td>&lt;70%</td>
<td>&lt;50% to 80%</td>
</tr>
<tr>
<td>SEVERE COPD</td>
<td>&lt;70%</td>
<td>&lt;30%</td>
</tr>
</tbody>
</table>

### 3.8: PATHOPHYSIOLOGY OF COPD

COPD comprises pathological changes in four different compartments of the lungs (central airways, peripheral airways, lung parenchyma and pulmonary vasculature), which are variably present in individuals with the disease\textsuperscript{(51)}.  

34
The pathophysiology of chronic obstructive pulmonary disease (COPD) is complex and can be attributed to multiple components: mucociliary dysfunction, airway inflammation and structural changes, all contributing to the development of airflow limitation, as well as an important systemic component \(^{(64)}\).

Inhaled cigarette smoke and other noxious particle such as smoke from bio mass fuels cause lung inflammation; a normal response that appears to be modified in patients who develop COPD. This chronic inflammatory response may induce paranchymal tissue destruction (resulting in emphysema) and disrupt normal repair and defense mechanism (resulting in small airway fibrosis). These pathological changes leads to progressive airflow limitation \(^{(65)}\).

3.8.1: PATHOLOGY:

Pathological changes characteristics of COPD are found in the airways, lung parenchyma and pulmonary vasculature. This pathology increased specific inflammatory cell types in different parts of lungs \(^{(66)}\).

3.8.2: PATHOGENESIS:

The inflammation in the respiratory tract of COPD patients appears to be a modification of the inflammatory response because of the chronic irritant such as smoking \(^{(67)}\).

3.8.3: OXIDATIVE STRESS:

Oxidative stress may be an important amplifying mechanism in the patients with COPD. Oxidants are generated by cigarette smoke and other inhaled particulates and released from activated inflammatory cells such as macrophages and neutrophils \(^{(68)}\).
3.8.4: PROTEASE-ANTIPROTEASE IMBALANCE:

There is evidence which suggest that imbalance of the protease and antiprotease in the lungs of COPD patients. The function of protease is to break down connective tissue components and the function of antiprotease which protects the lung tissue from the effects of the protease. This protease cells are derived from inflammatory cells and epithelial cells which are increased in COPD patients.

Protease mediated destruction of elastin major connective tissue components in the lung parenchyma, is believed to be an important feature of emphysema and is likely to be irreversible (63).

3.8.5: INFLAMMATORY CELLS:

COPD is characterized by a specific pattern of inflammation involving increased number of CD8+ (cytotoxic), TC1 Lymphocytes present only in smokers that develop disease. These cells together with neutrophils and macrophages releases inflammatory mediators and enzymes and interact with structural cells in the airways, lungs parenchyma and pulmonary vasculature (65).

3.8.6: INFLAMMATORY MEDIATORS:

The wide variety of inflammatory mediators that have been shown to be increased in COPD patients attracts inflammatory cells from the circulation (chemo tactic factors), amplify the inflammatory process (pro inflammatory cytokines) and induce structural changes (63).

3.8.7: PATHOPHYSIOLOGY:

There is now a good understanding of how the underlying disease process in COPD leads to the characteristics physiologic abnormalities and symptoms. For example inflammation and narrowing of the airways
leads to decreases FEV$_1$. Paranchymal destruction due to emphysema also contributes to airflow limitation leads to decreased gas transfer \(^{(69)}\).

### 3.8.8: AIRWAY LIMITATIONS AND AIR TRAPPING:

The reduction in FEV$_1$ is because of inflammation, fibrosis, and luminal exudates and other factors also contribute. This obstruction causes the trapping of air during expiration and this causes hyperinflation of the lung, this hyperinflation reduces the inspiratory capacity especially during exercise and leads to dyspnoea on exertion \(^{(70)}\).

![Pathophysiology of COPD](image)

**Image:** Pathophysiology of COPD

### 3.8.9: GAS EXCHANGE ABNORMALITY:

Gas exchange abnormality results in hypoxemia and hypercapnia. In general gas transfer for oxygen and carbon dioxide worsens as the disease progresses. Reduced ventilation may also be due to reduced ventilation drive. This may lead to carbon dioxide retention, when it is combined with reduced ventilation due to high work of breathing.
because of severe obstruction and hyper inflation coupled with ventilatory muscle impairments. The abnormalities in alveolar ventilation and reduced pulmonary vascular bed further worsen the VA/Q mismatch \(^{(71)}\) \(^{(72)}\).

3.8.10: PULMONARY HYPERTENSION (PHT):

P.H.T may develop late in the course of COPD and is due mainly to hypoxic vasoconstriction of small pulmonary arteries eventually resulting in structural changes that include intimal hyperplasia and later smooth muscle hypertrophy/hyperplasia. There is an inflammatory response in vessels similar to that seen in airways and evidence of endothelial cell dysfunction. Progressive pulmonary hypertension may lead to right ventricular hypertrophy and eventually right side cardiac failure \(^{(71)}\) \(^{(72)}\).

3.9: EXACERBATIONS:

An exacerbation of respiratory symptoms often occurs in patients with COPD triggered by infection with bacteria or viruses, environmental pollutants or unknown factors. During this period there is more hyperinflation which leads to increased dyspnoea at the end \(^{(63)}\).

3.10: SYSTEMIC FEATURES:

It is increasingly recognized that many patients with COPD have comorbidities that have a major impact on quality of life and survival. Airflow limitation and particularly hyperinflation affect cardiac function and gas exchange. Inflammatory mediators in the circulation may contribute to skeletal muscle wasting and cachexia and may initiate or worsen comorbidities such as I.H.D., Heart failure, osteoporosis, normocytic anemia, diabetes, metabolic syndrome and depression.
Study show that the earliest manifestation of chronic obstructive pulmonary disease (COPD) is an increase in residual volume suggesting that the natural history of COPD is a progressive increase in gas trapping with a decreasing vital capacity (VC). The reduction in VC forces the forced expiratory volume in 1 s to decline with it. This is aggravated by rapid shallow breathing leading to dynamic hyperinflation (61) (73) (74) (75).

**Figure 26: Systemic effects of COPD**

### 3.11: CHRONIC BRONCHITIS:

Chronic bronchitis is a disease of the airways. It is characterized by excess mucus secretion and productive cough. The cough is called a smokers cough in the early stages but once mucus production has been excessive for 3 months a year for over 2 years, this becomes the inadequate but traditional definition of chronic bronchitis.

Repeated inhalation of tobacco smoke irritates the sensitive lining of the airways, leading to inflammation, mucus hyper secretion and sometimes bronchospasm. Inflammation is a key process. It causes narrowing first in the distal small airways and then the proximal
airways. Acute inflammation resolves but chronic inflammation leads to fibrotic changes and scarring. Mucus hyper secretion due to mucosal damage is associated with rampant increase in the size and number of mucus secreting goblet cells (76).

3.12: EMPHYSEMA

Emphysema usually occurs with bronchitis and shares a similar etiology, but primarily a disease of alveoli and small airways, with secondary effects on other airways. It is usually caused by damage to the alveoli from smoking. Occasionally a congenital lack of alpha 1-antitrypsin causes primary emphysema in earlier life.

Protein breakdown is the villain of emphysema, leading to erosion of alveolar septa, dilation of distal airspaces. The walls of the terminal bronchi are normally supported by radial traction exerted by alveolar septa, but loss of elastic tissue means that, during expiration, compressive force are not opposed by radial traction and the floppy airways collapse.

Two types of emphysema are described, although they may co-exist. Centrilobular emphysema affects mainly the respiratory bronchioles. Pan lobular/Panacinar emphysema affects the alveoli (76).
3.13: CLASSIFICATION

Patients with emphysema have been classified into two groups according to their tendency to develop carbon dioxide retention and underventilation but the majority shows mixed features of these conditions.

3.13.1: BLUE–BLOATER:

This is the commoner of the two groups. The patient is usually obese, with copious sputum production and in whom exacerbations of breathlessness and wheezing are accompanied by infected sputum. Because of an associated tendency to underventilation and insensitivity to carbon dioxide such patients are commonly hypoxemic with hypercapnia especially during acute exacerbations. They frequently develop secondary polycythaemia and renal bicarbonate retention,
peripheral edema because of associated sodium retention. This type of patient will show moderately severe airflow obstruction with relatively normal total lung capacity but increased residual volume \(^{(77)}\).

### 3.13.2: PINK –PUFFERS:

This uncommon type of patient is usually thin, and severely breathless at rest with little or no sputum production. They may show little or no arterial oxygen desaturation at rest but develop marked desaturation on effort. Their sensitivity to carbon dioxide remains normal and hypercapnia only develops as pre-terminal event. Here there is severe airways obstruction with increased total lung capacity \(^{(77)}\).
How do the clinical presentation of emphysema and chronic bronchitis differ? (78)

<table>
<thead>
<tr>
<th>Similarities</th>
<th>Chronic bronchitis</th>
<th>Emphysema</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dyspnoea</strong></td>
<td></td>
<td><strong>Dyspnoea</strong></td>
</tr>
<tr>
<td><strong>Cough</strong></td>
<td></td>
<td><strong>Cough</strong></td>
</tr>
<tr>
<td><strong>Hypoxemia</strong></td>
<td></td>
<td><strong>Hypoxemia</strong></td>
</tr>
<tr>
<td><strong>Hypercapnia</strong></td>
<td></td>
<td><strong>Hypercapnia</strong></td>
</tr>
<tr>
<td><strong>Cor pulmonale</strong></td>
<td></td>
<td><strong>Cor pulmonale</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differences</th>
<th>Chronic bronchitis</th>
<th>Emphysema</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intermittent wheezing</strong></td>
<td></td>
<td><strong>Little or no cough</strong></td>
</tr>
<tr>
<td><strong>SOB</strong></td>
<td></td>
<td><strong>Very little sputum production</strong></td>
</tr>
<tr>
<td><strong>Productive cough often associated with smokers cough</strong></td>
<td></td>
<td><strong>Tachypneic</strong></td>
</tr>
<tr>
<td><strong>Copious amount of sputum, frequent pulmonary infection, cyanosis</strong></td>
<td></td>
<td><strong>Dyspneic prolonged expiration</strong></td>
</tr>
<tr>
<td><strong>Occasional barrel chest dyspnoea usually occurs late in course of illness</strong></td>
<td></td>
<td><strong>Pink puffers or blue bloaters</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Cyanosis is generally not common</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Occasionally wheezy</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Use accessory muscle to assist with ventilation</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Barrel chest</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Clubbing finger</strong></td>
</tr>
</tbody>
</table>

Table 5: Difference between chronic bronchitis and emphysema