Chronic Obstructive Pulmonary Disease

Goals and Objectives

Course Description
"Chronic Obstructive Pulmonary Disease" is an online asynchronous continuing education course for occupational therapists and occupational therapy assistants. This course presents contemporary information about chronic obstructive pulmonary disease including sections on pathophysiology, symptomatology, diagnosis, classification, complications/comorbidities, treatment, pulmonary rehabilitation, and psychosocial implications.

Course Rationale
The information presented in this course is applicable for occupational therapy professionals in all settings. A greater understanding of chronic obstructive pulmonary disease will enable occupational therapists and occupational therapy assistants to provide more effective and efficient rehabilitative care to individuals affected by this condition.

Course Goals and Objectives
Upon completion of this course, the participant will be able to:

1. Recognize the symptoms and risk factors associated with COPD
2. Identify significant history and clinical presentation associated with COPD
3. Classify COPD utilizing accepted guidelines
4. Identify medications used to treat COPD
5. List and define the components of a comprehensive pulmonary rehabilitation program
6. Recognize complications and comorbidities frequently associated with COPD
7. Identify and differentiate surgical interventions which may be utilized to treat COPD
8. Recognize the quality-of-life issues experienced by individuals with COPD
9. Identify the psychosocial challenges experienced by individuals with COPD
10. Identify challenges faced by the caregivers of individuals with COPD
11. Recognize the end-of-life issues relevant to individuals with COPD

Course Provider – Innovative Educational Services

Provider Contact Information – information@cheapceus.com

Course Instructor – Michael Niss, DPT

Financial/Non-financial Disclosure - Neither the Provider nor the instructor have any financial or non-financial conflict of interest related to the presentation of this CE program.

Target Audience – Occupational Therapists, Occupational Therapy Assistants

OT Scope of Practice – OT Service delivery (evaluation, intervention, outcomes); Foundational knowledge (Human body diagnoses and conditions)

Course Educational Level – This course is applicable for introductory learners.

Course Prerequisites – None

Method of Instruction – Distance Learning – Independent; Asynchronous online text-based home study

Location - Cheapceus.com

Date – Continuously available on-demand

Course Completion Requirements / Criteria for Issuance of CE Credits – Completion of instructional materials and a score of 70% or greater on the course post-test.

Continuing Education Credits – Three (3) contact hours / .3 AOTA CEUs / NBCOT 3.75 PDUs

Course Fee - $29.95

Registration Information – No pre-registration required; available on-demand at Cheapceus.com

Special Needs Requests – Email: information@cheapceus.com or phone: 954-663-4101

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Refund Policy - Unrestricted 100% refund upon request. The request for a refund by the learner shall be honored in full without penalty or other consideration of any kind. The request for a refund may be made by the learner at any time without limitations before, during, or after course participation.

American Occupational Therapy Association
Approved Provider

Innovative Educational Services is an AOTA Approved Provider of professional development. PD activity approval ID# 5471. This Distance Learning – Independent PD activity is offered at .3 CEUs; Introductory; OT Service delivery, Foundational knowledge. The assignment of AOTA CEUs does not imply endorsement of specific course content, products, or clinical procedures by AOTA.

Chronic Obstructive Pulmonary Disease

Course Outline

Overview

Introduction

Chronic Obstructive Pulmonary Disease (COPD) has a high impact on an individual's wellbeing, health care utilization, and mortality and causes a substantial and increasing economic and social burden.[1] Living with COPD can be challenging, as the disease dramatically impacts patients' daily life.[2] As COPD worsens and individuals experience increasing respiratory symptoms, a vicious cycle develops whereby activity declines, walking speed is reduced, fitness levels decline, and activities of daily living become too difficult to carry out, eventually causing disability and dependence.[1] When disease symptoms, particularly dyspnea, affects the performance of daily activities, the potential exist for important changes to occur in individual's overall quality of life. As the disease progresses, any kind of physical activity or social interaction may prove difficult. COPD is a significant disease which affects the individual physically, emotionally, and socially and leads to an increase in the social support needs of the patients.[2]

Definition

Chronic obstructive pulmonary disease (COPD) is a debilitating and progressive disease that primarily affects the respiratory system.[3] COPD is characterized by chronic airflow limitation, progressive and largely irreversible, associated with an abnormal inflammatory reaction. Chronic obstructive pulmonary disease (COPD) encompasses two phenotypically related diseases, chronic bronchitis and emphysema. Although the hallmark of COPD is inflammation and inability to maintain efficient gas exchange, emphysema is often characterized by atypical over-distension of the alveoli and permanent destruction of the surrounding supporting structures leading to irreversible damage to gaseous exchange.[4]

Pathophysiology

The pathogenesis of COPD involves interplay of several over-lapping and co-existing injuries, defects, inflammation and disorganized repair in a vicious cycle, ultimately leading to a chronic progressive impairment of lung function. These processes are shared by other airway and parenchymal diseases of lungs and aggravated by other pulmonary and systemic co-morbidities. The key steps involved are epithelial injury, inflammatory cell activation, protease-antiprotease imbalance, airway inflammation, goblet cell hypertrophy and hypersecretion, recurrent infection, acute exacerbations, attempts to disorganized repair and fibrosis, ultimately leading to chronic progressive permanent airway obstruction.[5]

Most of the major pathophysiologic changes associated with advanced COPD are attributed to systemic inflammation. Systematic inflammation is induced by inflammatory cytokines, such as tumor necrosis factor (TNF-α), interleukin (IL-6) and IL-8. Bronchiectasis, permanent damage and widening of one or more of the large

connecting bronchi (airways), occurs in nearly one third of individuals with COPD. Individuals with bronchiectasis have elevated levels of inflammatory cytokines that are associated with decreased fat-free mass, increased proteolysis and worse respiratory function. These inflammatory cytokines and endocrine hormones contribute to the reduction in exercise tolerance and poor quality of life caused by skeletal myopathy in COPD patients.[1]

Epidemiology

Chronic obstructive pulmonary disease (COPD) is the most common chronic lung disease, and a major cause of lung-related death and disability[6]. Fifteen million Americans report that they have been diagnosed with COPD.[7] Surprisingly, more than 50% of adults with low pulmonary function are not aware that they have the disease[8] therefore, the actual number may be higher. COPD is responsible for about 10.3 million physician visits and 1.5 million emergency department visits each year in the US.[9] Health care costs associated with COPD are an estimated $32 billion per year.[10] It is the third leading cause of death in the United States.[11] COPD is a major cause of disability and mortality worldwide and the prevalence increases with age. It is predicted that COPD will increase by more than thirty percent in the next ten years[6]. Statistically, it is rapidly approaching a leading cause of mortality in the United States, with a morbidity of 4.9 million and mortality rate at 4.2 per 100,000 [12].[4]

Risk Factors

Cigarette smoking

Cigarette smoking is the most significant and predictable risk factor in pathogenesis of COPD. Almost 80% of individuals who have COPD and 80% who die from COPD in the United States are smokers[13]. The estimated fraction of COPD mortality attributable to smoking was 54% for men 30–69 years of age and 52% for men 70 years of age or older[14]. There is a consistent exposure–response relationship which is demonstrated in evidence from cohort studies fulfilling the causal criterion of temporality (exposure preceding onset of disease). Although only 15% of smokers have clinically significant COPD, smoking leads to a predictable dose-dependent loss of lung function in pre-symptomatic phase which accelerates with age and has prognostic implications[14]. Smoking has supra-additive effect in worsening lung function and prognosis when combined with other risk factors like A1PI deficiency or occupational exposures[14].[5]

Genetic predilection

Genetically determined deficiency of alpha1–protease inhibitor (A1PI) represents a proven genetic abnormality that predisposes to COPD[14]. It is prevalent globally but most commonly found in whites of Northern European ancestry. Twin and familial aggregation studies suggest that genetic factors likely influence variation in pulmonary function in nonsmokers, but may not necessarily increase the risk of developing a clinical diagnosis of COPD[15].[5]

Occupational and environmental exposures
Farming and occupations with dusty environments increase the risk of developing chronic bronchitis two to threefold and, in combination with smoking, the risk increases to almost sixfold above average population. Environmental particulate air pollution and indoor smoke from biomass fuels have also been linked to COPD[14]. There is strong evidence of an association between outdoor pollution (particulate matter, O3, NO2) and decreased pulmonary function[14]. Exposure to air pollutants, occupational exposure, second-hand smoke exposure, fumes from burning biomass fuel, etc. can produce deleterious effects on the airway. Oxidative stress, pulmonary and systemic inflammation, reduction in airway ciliary activity, amplification of viral infections, and increases in bronchial reactivity could lead to irreversible loss of pulmonary function over time and COPD[15].[5]

Gender
Epidemiological studies show a male gender predominance related to the higher cigarette smoking habit or other inhaled toxins and occupational exposure among men within a population[16]. Increase in smoking among women has diminished the difference among gender prevalence. Mortality may be peaking among men in the United States but, among women, mortality continues to rise and deaths from COPD among women now even exceeds those among men.[5]

Asthma
Accelerated loss of lung function has been noted among asthma patients[14]. Functional changes in both the small airways and the alveolar parenchyma have been reported. Many individuals have bronchial inflammation with features of both asthma and chronic bronchitis/emphysema[14].[5]

Socioeconomic status
Morbidity and mortality rates have been shown to be inversely related to socioeconomic status[13]. Relative lack of awareness, diagnostic and therapeutic facilities and poorer health conditions may in part be connected to the socioeconomic status of the affected population.[5]

Developmental events
Impairment in early lung growth and development appears to increase the risk of development of COPD[14]. Maternal smoking, low-birth weight and recurrent childhood respiratory infections have been associated with higher incidence of adulthood COPD[14].
[5]

Dietary factors
Observational studies strongly suggest that dietary factors, such as a higher intake of vitamin C and other antioxidants (carotenoids, Vitamin E, lutein, flavanoids) are significantly associated with better lung function[15]. Some dietary elements like fruits and vegetables (antioxidants), fish (omega-3 polyunsaturated fatty acids) and Vitamin D seem protective while processed foods like cured meats (nitrites) may be deleterious for lung function preservation[15].[5]

Tuberculosis
Pulmonary tuberculosis can lead to scarring and accelerated decline in lung function. Some population-based surveys (PLATINO and PREPOCOL) reported strong association between previous tuberculosis and a greater risk of COPD[14].[5]

Intravenous drug abuse
Emphysema is prevalent in approximately 2% of intravenous drugs abusers which can be attributed to pulmonary vascular damage possibly from the insoluble filler. Bullous cysts are found in upper lobes of cocaine or heroin abusers whereas basilar and panacinar emphysema are associated with methadone and methylphenidate injections.[5]

Immune deficiency syndromes
Human immunodeficiency virus (HIV) infection was found to be a risk factor for COPD, independent of confounding variables[14]. Apical and cortical bullous lung damage occurs in autoimmune deficiency syndrome and Pneumocystis carinii infection.[5]

Connective-tissue disorders
Several connective disorders have been implicated in causation of or co-existence with emphysema and poor lung function. Cutis laxa is a congenital disorder of elastin tropoelastin that is characterized by premature aging and occasionally emphysema. Marfan syndrome (autosomal dominant inherited disease of type I collagen), Ehlers-Danlos syndrome, Salla disease (autosomal recessive storage disorder with intralysosomal accumulation of sialic acid), Birt-Hogg-Dube' syndrome and familial spontaneous pneumothorax syndrome (mutations in folliculin gene) have been associated with poor lung function, blebs, pneumothorax and emphysema[15].[5]

Symptomatology

The most common symptoms of COPD include:

- breathlessness (dyspnea)
- chronic cough
- wheezing
- sputum production
- recurrent respiratory infection
- weight loss
- fatigue
- exercise limitation
- muscle weakness

Diagnosis

The disease is diagnosed based on the combination of history, physical examination, and spirometry. Confirming information can also be obtained via arterial blood gases, sputum evaluation, and imaging studies.[6]

History

COPD is a gradually progressive chronic disease presenting with clinically obvious symptoms late in the course, usually in their fifth decade of life with productive cough or breathlessness or acute chest illness. A1PI-deficient patients present earlier than other COPD patients usually in 3rd-4thdecades and by then they have significant liver disease, which usually starts in childhood. Early COPD results in gradual progressive worsening of pulmonary function, which results in patients unknowingly avoiding exertional dyspnea (the most common early symptom of COPD) and fatigue by shifting their expectations and limiting their activity. Patients who have an extremely sedentary lifestyle but few complaints require further evaluation for possibility of underlying COPD as many patients reset their expectations with regard to health, termed "response shift"[14]. Generalized muscle weakness found in COPD patients can also contribute to this finding. Most patients initially present in the fifth or sixth decade of life when they have dyspnea with mild exertion and usually the forced expiratory volume in 1 second (FEV1) has fallen to 50% of predicted. Moderate to severe COPD patients report variability in symptoms over the course of the day or week-to-week; morning is typically the worst time of day. Dyspnea is related to both respiratory (hyperinflation and impaired gas exchange) and extra-respiratory (like muscle dysfunction, heart disease, anemia and depression) features of COPD. The chronic cough is characterized by the insidious onset of sputum production, which occurs in the morning initially, but may progress to occur throughout the day. The sputum is usually mucoid, but becomes purulent during exacerbations. Hemoptysis complicating chronic bronchitis usually occurs in association with acute exacerbation. Wheezing may also be found in some patients due to co-existence of asthma or COPD alone. Acute exacerbations are characterized by increased cough, sputum, dyspnea, and fatigue, are increasingly frequent as the disease worsens. Each exacerbation may last for a few weeks and followed by prolonged recovery over months and may be difficult to distinguish from other causes of dyspnea, cough, and/or sputum including pneumonia, congestive heart failure, pulmonary embolism, or pneumothorax[14]. A history of cigarette smoking or alternative inhalational exposure is usually found in majority of COPD patients. A1PI deficient patients may develop disease without smoking, however presence of smoking significantly worsens the course of disease. Some patients develop COPD without an obvious risk factor. Other historical features that may accompany COPD include certain comorbidities (eg, lung cancer, coronary artery disease, osteoporosis, depression, skeletal muscle weakness). Although most patients are usually obese, weight loss can also occur in COPD and is associated with a worse prognosis.[5]

Clinical Presentation

Physical findings in early COPD is highly non-specific and unreliable. Early stage patients may have coarse crackles and rhonchi. Wheezing may be found occasionally especially associated with asthma or acute exacerbations. The hallmark finding is obstruction of expiratory airflow. As the airway obstruction worsens, physical examination may reveal hyperinflation, decreased breath sounds, wheezes, crackles at

the lung bases, and/or distant heart sounds. In addition, the diaphragm may be depressed and limited in its motion, and the anteroposterior diameter of the chest may be increased.[5]

Patients with end-stage COPD may present with barrel-shaped chest, increased span of hyperresonant lung percussion, distended neck veins, full use of the accessory respiratory muscles of the neck and shoulder girdle, purse-lipped breathing, paradoxical retraction of the lower interspaces during inspiration (Hoover's sign), emaciation, and frequently, inguinal hernias. They may adopt positions that relieve dyspnea, such as leaning forward with arms outstretched and weight supported on the palms (Tripod sign). This position stabilizes the shoulder girdle and helps to maximize intrathoracic volume. Late signs may include cyanosis, clubbing, myoclonus due to severe hypercapnia, and an enlarged, tender liver due to right heart failure.[5]

Spirometry

Spirometry is a simple test to measure the amount of air a person can breathe out, and the amount of time taken to do so. A spirometer is a device used to measure how effectively and how quickly the lungs can be emptied. Objective measurement of airflow obstruction is the mainstay of workup for diagnosis, staging and follow-up of COPD[17]. The most important values measured are the forced expiratory volume in one second (FEV1) and the forced vital capacity (FVC) or the forced expiratory volume after 6 seconds, (FEV6), which is the recommended substitute for FVC. COPD is confirmed when a patient, who has symptoms that are compatible with COPD, is found to have airflow obstruction (FEV1/FVC ratio less than 0.70 and an FEV1 less than 80 percent of predicted) and there is no alternative explanation for the symptoms and airflow obstruction (eg, bronchiectasis, vocal cord paralysis, tracheal stenosis). If airflow is abnormal, post-bronchodilator testing should be performed. Correction of airflow to the normal range suggests a diagnosis of asthma and could exclude COPD. Because of variability in the FVC (or FEV6) measure, the FEV1/FVC ratio can establish a diagnosis of obstruction but is not useful to monitor disease progression[18]. Other spirometric findings include decreased inspiratory capacity and vital capacity, accompanied by increased total lung capacity, functional residual capacity, and residual volume.[5]

Spirometric assessment is performed according to the guidelines of the American Thoracic Society (ATS)[19]. The subject performs three exhaling exercises and the best is used for the analysis[20]. If the Tiffenau rate (value of FEV1/FVC) is less than seventy percent, COPD exists[18].[6]

Spirometry measurements used for diagnosis of COPD include:

- **FVC** (forced vital capacity): maximum volume of air that can be exhaled during a forced maneuver.
- **FEV_1** (forced expired volume in one second): volume expired in the first second of maximal expiration after a maximal inspiration. This is a measure of

how quickly the lungs can be emptied. FEV_1 is influenced by the age, sex, height, and ethnicity, and is best considered as a percentage of the predicted normal value.

- **FEV_1/FVC**: FEV_1 expressed as a percentage of the FVC, gives a clinically useful index of airflow limitation. The ratio FEV_1/FVC is between 70% and 80% in normal adults; a value less than 70% indicates airflow limitation and the possibility of COPD.

Arterial Blood Gases

Arterial blood gases reveal mild or moderate hypoxemia (low oxygen levels) without hypercapnia (elevated carbon dioxide levels) in the early stages of COPD. In the later stages of the disease, hypoxemia tends to become more severe and may be accompanied by hypercapnia with increased serum bicarbonate levels. The changes in ABG represent ventilation perfusion mismatch, which may be worsened during exercise, sleep and episodes of exacerbation.[5] Arterial blood gases (ABG) are used to correlate symptoms with blood oxygenation levels but is not needed in mild to moderate airflow obstruction. ABG is optional in moderately severe airflow obstruction however, for severe disease, ABG then becomes the major monitoring tool once hypoxemia with hypercapnia develops.[4]

Sputum Evaluation

In patients with stable chronic bronchitis, the sputum is mucoid and the predominant cells are macrophages[14]. With an exacerbation, the sputum becomes purulent, with excessive neutrophils and a mixture of organisms visualized through Gram staining. *Streptococcus pneumoniae* and *Haemophilus influenzae* are pathogens frequently cultured during exacerbations.[5]

Imaging Studies

Chest radiographs and CT-scan of chest are the mainstay of COPD imaging. Although not contributing to diagnosis of COPD, they may add valuable information regarding severity, stage and special findings during the course of disease.[5]

Chest X-ray

Chest radiography is usual the initial study performed or ordered but will not be diagnostic except in severe cases, however, is still important to exclude other lung diseases.[4] Radiographic features suggestive of COPD are prominent usually in advanced disease and include:

- **Signs of hyperinflation -** Prominent hilar vascular shadows and encroachment of the heart shadow on the retrosternal space, increased radiolucency of the lung, a flat diaphragm, and a long and narrow heart shadow on a frontal radiograph, accompanied by a flat diaphragmatic contour may be seen.[5]

- **Bullae** - defined as radiolucent areas larger than one centimeter in diameter and surrounded by arcuate hairline shadows. They are due to locally severe disease.[5]
- **Rapidly tapering vascular shadows and cardiac enlargement -** may become evident only on comparison with previous chest radiographs. These findings are due to pulmonary hypertension and cor pulmonale, which can be secondary to COPD.[5]

Computed Tomography

High-resolution CT (HRCT) scanning is more sensitive than standard chest radiography and is highly specific for diagnosing emphysema and outlines bullae that are not always observed on radiographs.[5] Computed tomography (CT) is better able to characterize the involvement pattern as either centriacinar or panacinar. Centriacinar usually involves the upper lobes in the center of secondary pulmonary lobules, in contrast to panacinar with involves the lung bases and the entire secondary pulmonary nodule with generalized paucity of the vascular structures.[4] A CT scan is not indicated in the routine care of patients with COPD but is helpful when the patient is being considered for a surgical intervention such as bullectomy or lung volume reduction surgery.[5]

Dyspnea

Dyspnea is a sensation of respiratory discomfort and the evaluation of the degree of dyspnea provides an independent dimension that is not provided by pulmonary function tests or by measuring dyspnea in an exercise laboratory. One of the major goals of COPD treatment is a reduction in dyspnea. The severity of the disease can be determined by the intensity of dyspnea[21,6].

Changes in breathing are so slow and insidious that for a long time the decline is normalized; put down to getting older or being less fit. Eventually, the breathlessness begins to impact on the person's ability to conduct their day-to-day activities and is accompanied by other respiratory symptoms and poor exercise tolerance. Distressing breathlessness can be precipitated by certain body positions, by activities such as walking and climbing stairs and by extremes of emotion. Environmental triggers such as excessive heat or cold, smoke or perfumes exacerbate breathlessness and people may need to anticipate and avoid these triggers. This avoidance of the triggers of breathlessness can isolate people from locations and activities that once afforded them pleasure.[22]

Breathless people experience good days and bad days and this means that despite planning ahead, a bad day may rule out hoped for activities. Certain times of the day can be more problematic, with breathing often worse in the mornings, coinciding with the need to clear sputum and the need to attend to washing and dressing, and at night interfering with sleep. Certain times of the year can also worsen breathlessness due to extremes of temperature. Acute breathlessness is associated with panic, fear of

suffocation, and fear of dying during an attack. People feel helpless and out of control of their bodies at these times. Strategies can be taught that help bring respiratory distress under control. Breathing techniques such as consciously slowing breathing, diaphragmatic breathing or purse-lipped breathing are reported widely by patients as effective ways to help manage frightening breathlessness.[22]

Because dyspnea is a subjective symptom, it is assessed through the use of standardized symptom reports or questionnaires[23]. For the most part, questionnaires used to measure dyspnea as an outcome of pulmonary rehabilitation are evaluative instruments and each of this instruments measure different aspect of dyspnea[24].[2]

Modified Medical Research Council Dyspnea Scale (mMRC)[6] - The mMRC has five levels that increase with the level of activity in which dyspnea appears. It assesses common tasks the patient can develop without displaying dyspnea. Levels of Dyspnea are graded as follows:

- Grade 0: “I only get breathless with strenuous exercise”
- Grade 1: “I get short of breath when hurrying or walking up a slight hill”
- Grade 2: “I walk slower than people of the same age because of breathlessness or have to stop for breath when walking at my own pace”
- Grade 3: “I stop for breath after walking 100 yards or after a few minutes”
- Grade 4: “I am too breathless to leave the house”.

Baseline Dyspnea Index (BDT) – The BDT analyzes dyspnea from a triple perspective; the difficulty of the task, magnitude of effort and functional impairment, each of the sections is assessed from 0 (severe) to 4 (none), so total amount can range between 0 and 12[25].[6]

The Oxygen Cost Diagram (OCD) - This scale was developed in an effort to match a range of tasks with the occurrence of dyspnea[32]. The OCD is a 100-mm vertical visual analog scale with 13 activities listed at various points along the line corresponding to increasing oxygen requirements for their completion, ranging from sleeping (at the bottom) to brisk walking uphill (at the top)[27].[2]

The University of San Diego Shortness of Breath Questionnaire (SOBQ) - The University of San Diego Shortness of Breath Questionnaire (SOBQ) is a 24-item measure that assesses self-reported shortness of breath while performing a variety of activities of daily living[28]. Patients are asked to rate their dyspnea associated with the 21 different activity, from 0 = "not at all" to 5 = "maximally or unable to do because of breathlessness." Three additional questions about limitations due to shortness of breath, fear of harm from overexertion, and fear of shortness of breath are included for a total of 24 items.[28].[2]

The Borg Scale - The Borg scale is a category-ratio scale. It is commonly used to evaluate the effects of exercise on dyspnea. The original and modified scales have ratio

properties ranging from 0 = nothing at all to 10 = very, very severe, with descriptors from 0 to 10. The Borg scale has been used in pulmonary rehabilitation programs to evaluate dyspnea before, during, and after progressive exercise[29].[2]

The Visual Analog Scale (VAS) - The VAS is usually a 100 mm line anchored at either end with descriptors, such as "none" to "very severe." When used to measure dyspnea, these anchors are qualified to read "no shortness of breath" to "maximum shortness of breath," or some similar variation[30]. The VAS can be used to quantify a number of aspects of symptoms besides the sensation of dyspnea, such as effort and distress with dyspnea. The visual analogic scales and the Borg scale are the simplest tools available; both are completed by the patient, and allow a follow-up of the impact of treatment on dyspnea[31].[2]

The Chronic Respiratory Questionnaire (CRQ) - The Chronic Respiratory Questionnaire (CRQ), a 20-item, disease-specific, quality-of-life questionnaire[32], has been used extensively in pulmonary rehabilitation settings. The CRQ consists of four domains (dyspnea, fatigue, emotional function, and mastery), rated on a seven-point scale. The dyspnea component of the CRQ asks patients to identify five activities of importance to them. These same activities are rated with 1 = most dyspnea and 7 = least dyspnea, before and after a pulmonary rehabilitation program[24]. The CRQ has a fatigue subscale consisting of five items, scored on a 7-point scale. The CRQ fatigue domain is reliable, valid with the same clinically important differences as the other components. To determine the outcomes of pulmonary rehabilitation, it is safe to say that the CRQ is the most widely used and tested instrument that measures both dyspnea and fatigue[33].[2]

The Pulmonary Functional Status Scale (PFSS) - The Pulmonary Functional Status Scale (PFSS) is a 53-item, self-administered questionnaire measuring physical, mental, and social function. The dyspnea subscale evaluates dyspnea related to activities, as well as dyspnea independent of activities[34].[2]

The Pulmonary Functional Status and Dyspnea Questionnaire (PFSDQ) - The Pulmonary Functional Status and Dyspnea Questionnaire (PFSDQ) is a 164-item, self-administered questionnaire that evaluates dyspnea and activity levels. The pulmonary functional status and dyspnea questionnaire-modified version (PFSDQ-M), measure dyspnea, fatigue, and activity levels[35]. PFSDQ-M has been used to measure fatigue in COPD patients. The PFSDQ-M comprises three domains: influence of dyspnea on ADLs, influence of fatigue on ADLs and change experienced by the patient in ADLs. The patient reports to what degree dyspnea and fatigue affect 10 specific ADL items, assigning a score from 0 to 10 for each activity as follows: 0 (no interference); 1-3 (mild); 4-6 (moderate); 7-9 (severe); and 10 (extremely severe). Higher values on the scale indicate greater ADL limitation.[2]

Classification (Staging)

Two different methodologies are commonly utilized to classify COPD; the GOLD Guidelines and the BODE Index.

GOLD Guidelines

The GOLD classification system separates COPD patients into the four different stages[18].

Stage I: Mild COPD
Mild airflow limitation (FEV1/FVC < 70%; FEV1 >80% predicted) and sometimes, but not always, chronic cough and sputum production. At this stage, the individual may not be aware that his or her lung function is abnormal.

Stage II: Moderate COPD
Worsening airflow limitation (FEV1/FVC < 70%; 50% < FEV1 < 80% predicted), with shortness of breath typically developing during exertion. This is the stage at which patients typically seek medical attention because of chronic respiratory symptoms or an exacerbation of their disease.

Stage III: Severe COPD
Further worsening of airflow limitation (FEV1/FVC <70%; 30% < FEV1 < 50% predicted), greater shortness of breath, reduced exercise capacity, and repeated exacerbations which have an impact on patients' quality of life.

Stage IV: Very Severe COPD
Severe airflow limitation (FEV1/FVC < 70%; FEV1 <30% predicted) or FEV1 < 50% predicted plus chronic respiratory failure. Patients may have Very Severe (Stage IV) COPD even if the FEV1 is > 30% predicted, whenever this complication is present. At this stage, quality of life is very appreciably impaired and exacerbations may be life-threatening.

BODE Index

The BODE index is a multidimensional classification system that systemically determines the degree of mortality in individuals with COPD, that provides useful prognostic information in patients with COPD and might be able to measure health status.[6] The GOLD staging system has been criticized for underestimating the importance of the extrapulmonary manifestations of COPD in predicting outcome. The BODE index addresses this criticism.[5] It encompasses the body mass index (B), the degree of airflow obstruction as expressed by the FEV1 (O), dyspnea with the modified medical research council (D), and exercise (E) measured with six-minute walk distance.[6] This index provides better prognostic information than the FEV1 alone to assess an individual's risk of death or hospitalization due to COPD. However, it is not

used to guide therapy.[5] Table 1 below shows the point value used for the computation of the BODE index[36]. Table 2 shows the estimated mortality rate associated with given BODE Index scores.[6]

Table 1. Variables and cutoff values for points 0 to 3 in the BODE index computation

	Point on BODE Index			
	0	1	2	3
FEV_1 (% of predicted)	>65	50-64	36-49	<35
Distance walked in 6 minutes (meters)	>350	250-349	150-249	<149
Dyspnea scale score (mMRC)	0-1	2	3	4
Body mass index measure	>21	<21	xx	xx

Table 2. BODE Index Scoring to Estimate Mortality

BODE Index Score	12-month mortality (%)	24-month mortality (%)	52-month mortality (%)
0-2	2	6	19
3-4	2	8	32
4-6	2	14	40
7-10	5	31	80

Index Score is utilized to predict 12, 24, and 52-month mortality.
Index score is obtained via Table 1

Pharmacological Treatment

The U.S. Food and Drug Administration (FDA) recommends five treatment end points be considered for COPD:

1. Improvement in airflow obstruction
2. Providing symptom relief,
3. Modifying or preventing exacerbations
4. Altering disease progression (including mortality)
5. Modifying lung structure.

Effective treatment of the COPD patient requires effective integration of pharmacologic treatment and non-pharmacologic therapy.[5]

Bronchodilators

Bronchodilators are the mainstay of any COPD treatment plan. The mechanism of action is primarily by dilating airways and thereby decreasing airflow resistance increasing airflow and decreasing dynamic hyperinflation which is the origin of early stage symptoms. Many patients with COPD will have reduced dyspnea and improved

exercise tolerance with bronchodilator therapy, even if improvement in resting spirometry is very modest. Unlike asthma, COPD patients mostly need bronchodilators both on a chronic basis as well as for “rescue”. All symptomatic patients with COPD should be prescribed a short-acting bronchodilator for as-needed basis and a regularly scheduled long-acting bronchodilator should be added if symptoms are inadequately controlled. Bronchodilators include beta agonists and anticholinergics. The initial choice of agent remains debated. Historically, beta 2 agonists were considered first line and anticholinergics added as adjuncts.[5]

Beta 2-agonists

This group of medications bind to the beta-adrenergic receptor present on airway smooth muscle, resulting in bronchodilation and improvement in airflow. They may also help by increasing ciliary beating frequency and improving mucus transport and may improve endurance of fatigued respiratory muscles. Beta agonists are available in short-acting and long-acting inhaled formulations. The shortacting- beta agonists (SABA) have a relatively rapid onset of action after inhalation, in about 5 to 15 minutes, and the bronchodilation lasts for 2 to 4 hours. Long-acting beta-agonists (LABAs) have a longer onset and bronchodilation lasting for up to 12 hours or more. Inhaled route is preferable owing to more favorable ratio of therapeutic effect to undesirable side effects.[5]

A metered dose inhaler (MDI), dry powder inhaler (DPI) is the preferred mode to deliver a bronchodilator medication by inhalation as it simplifies therapy, improves compliance, and may reduce extra medication usage and patient cost. Nebulizers may be more effective in patients too weak to use an inhaler device, in those with altered mental status, or in those whose inspiratory capacity is too limited to permit effective inhalation.[5]

The benefits of treatment include improvement in airflow obstruction and symptom relief. Side-effects commonly include tremor, palpitations, anxiety, and insomnia. Ventricular arrhythmias and hypokalemia may also occur.[5]

Anti-cholinergics

Anticholinergic agents block M2 and M3 cholinergic receptors and result in bronchodilation. In airway smooth muscle cells, acetylcholine stimulates the production of neutrophil chemotactic activity and anticholinergics could, theoretically, have anti-inflammatory action. Short-acting anticholinergic agents improve lung function and symptoms.[5]

Benefits of treatment are similar to beta-agonist agents including improvement in airflow obstruction and symptom relief. However, as with other bronchodilators, anticholinergics have no effect on disease progression and alteration of lung structure. Reported adverse effects include dry mouth, metallic taste, and prostatic symptoms.[5]

Corticosteroids

COPD is characterized by both airway and systemic inflammation as discussed in the pathogenesis and the primary reason of disease progression. Bronchodilators achieve temporary symptom control but have failed to show any effect on the underlying inflammation. Corticosteroids are by far the leaders of this class of medication, and some newer phosphodiesterases have shown promise.[5]

Inhaled glucocorticoids decrease frequency of exacerbations and modestly slow the progression of respiratory symptoms, but appear to have little impact on lung function and mortality. Because of their lack of effect on bronchodilation, inhaled glucocorticoids can be used only as part of a combined regimen, but are not as sole therapy. Benefits from therapy include reduction in the frequency and severity of exacerbations of COPD. Inhaled corticosteroids are only minimally absorbed and therefore systemic adverse effects are limited. Local effects include oral candidiasis and dysphonia. Systemic effects include increased bruising and reduced bone density, and possible susceptibility for pneumonia.[5]

Appropriate caution and monitoring is recommended although the clinical importance of these effects remain uncertain. Systemic steroids have been widely used in the treatment of acute exacerbation of COPD. The use of oral steroids in persons with chronic stable COPD is not recommended given the adverse effect profile, which includes hypertension, glucose intolerance, osteoporosis, fractures, and cataracts, among others[14]. Inhaled glucocorticoids are typically used in combination with a long-acting bronchodilator for patients in GOLD stage III-IV, who have significant symptoms or repeated exacerbations, despite an optimal bronchodilator regimen.[5]

Oxygen Therapy

For individuals whose resting arterial Po2 is between 56 and 59 mmHg, long-term oxygen therapy is indicated if they demonstrate erythrocytosis (hematocrit ≥ 55%) or evidence of cor pulmonale.[5] Oxygen therapy is one of the therapies currently available to reduce COPD mortality. Long term oxygen therapy (LTOT) reduces pulmonary hypertension and improves survival in patients with COPD and resting hypoxemia (arterial partial pressure of oxygen ≤55 mmHg).[1] Exercise-induced hypoxemia is another accepted indication for supplemental oxygen. Supplemental oxygen during exercise training improves functional outcomes such as symptoms, health-related quality of life, and ambulation.[1] Three distinct oxygen-conserving devices are available, and they include reservoir cannulas, demand pulse delivery devices, and transtracheal oxygen delivery. Oxygen-conserving devices function by delivering all of the oxygen during early inhalation. Each liter of oxygen flow adds 3-4% to the fraction of inspired oxygen (FiO2). The continuous-flow nasal cannula is the standard means of oxygen delivery for stable hypoxemic patients. The cannula is simple, reliable, and generally well tolerated.[5]

Cessation of Cigarette Smoking

Smoking cessation is the single most effective therapy for the majority of COPD patients[37]. The transition from smoking to nonsmoking status involves following five stages: precontemplation, contemplation, preparation, action, and maintenance. Smoking intervention programs include self-help, group, physician-delivered, workplace, and community programs. Setting a target date to quit may be helpful. Physicians and other health care providers should participate in setting the target date and should follow up with respect to maintenance. Successful cessation programs should include patient education, target date to quit, follow-up support, relapse prevention, advice for healthy lifestyle changes, social support systems, pharmacological agents. According to the US Preventive Services Task Force (USPSTF) guidelines, recommends "5-A" approach to counseling that includes[5]:

i) Ask about tobacco use,
ii) Advise to quit through personalized messages,
iii) Assess willingness to quit,
iv) Assist with quitting,
v) Arrange follow-up care and support.

Behavioral counseling and pharmacotherapy are most effective when used together.[38] Supervised use of pharmacologic agents is an important adjunct as withdrawal from nicotine may cause unpleasant adverse effects during the first weeks after quitting smoking. Nicotine replacement therapies are available in
the form of chewing gum and transdermal patches to counter the withdrawal symptoms[37]. Long-term success rates have been 22-42%, compared with 2-25% with placebos. The use of an antidepressant medication, bupropion (Zyban,150 mg bid) has been shown to be effective for smoking cessation and may be used in combination with nicotine replacement therapy. Varenicline (Chantix), is a partial agonist selective for a4, b2 nicotinic acetylcholine receptors and action is thought to result from partial agonist activity at a nicotinic receptor subtype while simultaneously preventing nicotine binding. Nortriptyline and clonidine have also been proposed to help in cessation of smoking[37].[5]

Pulmonary Rehabilitation

Multidimensional pulmonary rehabilitation has emerged as a recommended standard of care for patients with chronic lung disease based on a growing body of scientific evidence. The primary goal of rehabilitation interventions for people with COPD is to optimize function.[6] Comprehensive pulmonary rehabilitation has been shown to improve exercise capacity, improve independence quality of life, decrease dyspnea, and decrease health care utilization and it may also reduce mortality[36]. Although airflow obstruction (FEV1) is not improved, the effects of rehabilitation on health status ("quality of life") are generally much greater than seen with pharmacologic treatments[14]. Pulmonary rehabilitation should be considered as an addition to medication therapy for symptomatic patients who have GOLD Stage II, III, or IV COPD. Pulmonary

rehabilitation program usually requires a team approach, including physicians, nurses, dietitians, respiratory therapists, exercise physiologists, physical therapists, occupational therapists, athletic trainers, cardiorespiratory technicians, pharmacists, and psychosocial professionals. This multidisciplinary approach emphasizes on patient and family education, smoking cessation, medical management (including oxygen and immunization), respiratory and chest physiotherapy, physical therapy with bronchopulmonary hygiene, exercise, and vocational rehabilitation and psychosocial support.[5]

There is no consensus of opinion regarding the optimal duration of the pulmonary rehabilitation intervention[39]. The duration depends on changes in the patient´s lifestyle. A number of external factors also influence program duration including health-care systems and reimbursement policies, access to programs, level of functional disability, health-care provider referral patterns, and the ability of individual patients to make progress toward treatment goals.[6]

Physical Activity

Physical inactivity is one of the most potent predictors of mortality in COPD[40]. Low levels of physical activity have been associated with a higher risk of hospitalization and re-hospitalization[40]. Physical activity is also the most important factor in determining self-rated general health and quality of life (QoL) in patients with COPD, with the most physically active patients reporting the best health and QoL[41]. Overall, patients with COPD who are more physically active have generally better functional status in terms of diffusing capacity of the lung carbon monoxide, expiratory muscle strength, exercise capacity, maximal oxygen uptake and systemic inflammation, compared with those who are less active[42]. Considering this relationship between physical activity and meaningful patient outcomes, improving physical activity levels is an important goal in the management of COPD.[3]

It is important to make the distinction between physical activity and exercise capacity, which are both closely related to clinical outcomes in COPD. Physical activity is ‘any bodily movement produced by skeletal muscles that results in energy expenditure. By contrast, exercise capacity indicates an individual’s ability to endure exercise, where exercise comprises physical activities that are specifically performed with the intention of improving physical fitness. Exercise capacity indicates what a person is capable of doing, while physical activity reflects what someone actually does.[3]

Physical activity levels are remarkably lower in stable outpatients with COPD than in healthy individuals[41]; even in patients with early-stages disease[43]. Physical activity level is recognized as a predictor of mortality and hospitalization in patients with COPD and contributes to disease progression and poor outcomes[42].[3]

The assessment of physical activity in healthy populations and in those with chronic diseases is challenging. Instruments used to quantify physical activity may be either subjective or objective.[1]

Subjective Instruments

Specifically designed questionnaires and diaries are subjective measures that have been used to quantify physical activity in daily life. These tools are helpful for evaluating the patients' perspectives on their ability to carry out daily activities. Self-reported questionnaires and diaries rely on memory and recall of the patients and several variables such as the design of the questionnaire, patient characteristics (age, cognitive capacity, cultural factors) and interviewer characteristics may affect the reliability of the results. It has been shown that patients' estimation of time spent on physical activities in daily life disagreed with objective assessment.[3]

The most frequently used subjective tools with a better-documented validation include:

- The Minnesota Leisure Time Physical Activity Questionnaire (MLTPAQ) or Survey (MLTPAS)
- The Baecke Physical Activity Questionnaire
- Follick's diary
- The Physical Activity Scale for the Elderly (PASE)
- The Zutphen Physical Activity Questionnaire (ZPAQ).

Objective Instruments

The clinical evaluation and validation of objective measures of assessing physical activity continues to be investigated. Motion sensors, which include pedometers used for measuring steps and accelerometers used for detecting body acceleration, can be used for the objective quantification of physical activity over time. Accelerometers are electronic devices, generally worn on the arm (multisensory armband devices) or waist, which estimate physical activity outcomes such as body posture, quantity and intensity of body movements, energy expenditure, and physical activity level based on measurements of body's acceleration.[3]

Exercise

Exercise aims to reverse the systemic consequences of COPD, in particular the skeletal muscle dysfunction, enhances the mechanical efficiency of physical activities (particularly walking) and reduces the sensitivity to dyspnea and the ventilation required to overcome a specific task. In many patients, COPD also has detrimental extra-pulmonary effects, such as weight loss and skeletal muscle dysfunction/wasting. The pulmonary and skeletal muscle abnormalities limit the pulmonary ventilation and

enhance the ventilatory requirements during exercise resulting in exercise-associated symptoms such as dyspnea and fatigue. These symptoms make exercise an unpleasant experience, which many patients try to avoid, and along with a depressive mood status, further accelerates the process, leading to an inactive life-style. Muscle deconditioning, associated with reduced physical activity, contributes to further inactivity and as a result patients get trapped in a vicious cycle of declining physical activity levels and increasing symptoms with exercise.[3]

Exercise Tolerance - Six Minute Walk Test

Exercise tolerance is a well-accepted clinical marker in COPD and provides information about disease stage, prognosis, functional capacity, and the effects of treatment.[1] The 6-min walk test (6MWT) remains the most popular test for the evaluation of exercise tolerance in COPD patients. It is simple and well standardized.[5] The 6MWT measures the global and integrated responses of all organ systems involved during exercise. It has been shown to be an important parameter related to morbidity and mortality in COPD[44], and is also part of the BODE Index.[6].

Aerobic Exercise

Aerobic exercise is the main non-pharmacological treatment better tolerated by patients with COPD. The exercise prescription for the training program is guided by the following three parameters: intensity; frequency; and duration. Aerobic exercise training for older people should have a target intensity of 50-85% of the oxygen uptake reserve – a range that includes both moderate exercise (minimum of 30 minute five days a week) or vigorous exercise (20 minutes three days each week)[45].[6]

There are two different types of aerobic exercise for COPD patients: interval (a.k.a. incremental) training and continuous (a.k.a. endurance) training. Interval training is recommended as an alternative to continuous training in patients with severe symptoms of dyspnea during exercise due to an inability to sustain continuous training at the recommended intensities. During interval training, short exercise bouts (30-180 seconds) are performed at high intensity (at least 70-80% of peak work rate).[1]

There is high level evidence that continuous training is effective for aerobic capacity and there is moderate evidence that interval training is effective for strength, endurance, functionality and psychosocial parameters[46].[6]

Continuous

Supervised continuous training is recommended for patients in all stages of the disease who are able to perform continuous training of at least moderate intensity. Training frequency should be three times weekly in the first weeks of the exercise program[47]. Patients with severe symptoms of dyspnea during exercise are frequently not capable of performing high-intensity (70 to 80% of the peak work rate) continuous type training[48]. It seems that moderate intensity continuous training (50 to 60% of the peak work rate or

5-6 out of 10 according to the modified Borg Scale) is minimally required to achieve changes in physical fitness. Improvements in health-related quality of life after training at moderate intensities were comparable with those observed after high intensity training[49].[6]

Lower extremity exercise training at higher exercise intensity produces greater physiologic benefits than lower intensity training in patients with COPD. Moreover, both low-intensity and high-intensity exercise training produce clinical benefits for patients with COPD[50].[6]

Two categories of tasks can be found during everyday activities, endurance and strength tasks. Endurance tasks require repetitive actions over an extended period of time (walking, cycling and swimming). While strength tasks require explosive performance over short time periods (jumping, lifting weights, sprinting)[39]. The addition of a strength-training component to a program of pulmonary rehabilitation increases muscle strength and muscle mass[50].[6]

Interval training

Most patients with severe COPD are not able to sustain a continuous exercise protocol. For these patients, interval exercise represents an alternative because it offers the same benefit as high-intensity exercise. Short high intensity (at least 70-80% of peak work rate) exercise bouts of 30-180 seconds are necessary during interval training. Recommended frequency of training is the same as with continuous training[47].[6]

Resistance Exercise

Given that muscle weakness is a common problem in this population, progressive resistance exercise represents a beneficial treatment for improvements in muscle strength. Moreover, improvements in muscle strength can be obtained when progressive resistance exercise is conducted alone or in combination with aerobic training, indicating that it can be successfully performed in conjunction with other training types during pulmonary rehabilitation. Resistance training is an ideal intervention for patients with peripheral muscle weakness and pronounced symptoms of dyspnea during exercise[51]. There is not consensus on the optimal method of resistance training (calisthenics, resistance weight training, isometrics or isokinetic-type training) in patients with COPD. Each type produces strength gains highly specific to the type of training. There are no studies that compared different intensities of resistance training in patients with COPD. It is recommended to use (lower limb) resistance training according to ACSM (two or three times a week)[45]. Exercises should be performed at 60-80% of the first repetition maximum (RM), resistance exercises should train 8-10 exercises involving the major muscle groups in bouts of 8-15 repetitions at least 30 minutes a day of moderate-intensity activity on two or three non-consecutive days each week[52]. Multiple sets of repetitions (2-5 sets) provide greater benefit[47]. Resistance activities include a progressive-weight training program, done with therabands (wrist or ankle weights) or progressive weight.[6]

Careful consideration is also required when prescribing progressive resistance exercise programs for people with COPD who have comorbid health conditions. Therefore, progressive resistance exercise may not be appropriate for all people with COPD attending pulmonary rehabilitation, and it is recommended that prescription be targeted to the individual.[6]

Respiratory System Exercise

Breathing Control Exercises
Breathing Control Exercises (BCEs) aim to decrease the effort required for breathing and assist relaxation by deeper breathing, which may result in an improved breathing pattern through decreased respiratory rate and reduced breathlessness. Examples of breathing control exercises are[53]:

- Diaphragmatic breathing – Slow and deep breathing is performed by breathing with the diaphragm muscles. The work of the accessory muscles during inspiration is reduced at the same time.
- Yoga breathing – Enhanced breathing based on somatic relaxation
- Body position exercises – Favorable positions of the body that encourage the upper chest, shoulders, and arms to relax; allowing movement of the lower chest and abdomen. Examples include: high side lying, relaxed sitting, forward lean standing, and knee learning positions.
- Pursed lip breathing – Exhalation through pursed lips.

Respiratory Muscle Training
The goal of Respiratory Muscle Training (RMT) is to improve muscle strength and endurance of impaired respiratory muscles, hopefully resulting in greater control of breathing patterns and a reduction breathlessness. RMT requires the use of an adjusted breathing resistance device and can focus on either inspiratory or expiatory muscle groups.[53]:

Inspiratory muscle training – Three examples of inspiratory muscle training are[53]:

- Inspiratory resistive load breathing – inhalation is performed through a mouthpiece with an adjustable diameter adapter.
- Inspiratory threshold loading – breathing performed through a flow of independent resistance.
- Normocapnic hyperpnea – respiratory muscles are trained at a high level for an extended period of time.

Expiratory muscle training – Can be performed utilizing low-intensity endurance training or high intensity strength training.

Chest Mobility

Biomechanics of Chest Movement

Movement of the chest wall is a complex function within the rib cage, sternum, thoracic vertebrae, and muscles. Basic observation reveals chest configuration for abnormality of the spine or chest shape, for example, scoliosis, kyphoscoliosis, barrel, or pectus excavatum. Normally, in all joint movement at the end of expiration, the intercostal muscles are at a suitable length before contraction during inspiration. In assessment, chest stiffness may be caused by muscle structure being applied directly in the supine, side lying or sitting position. Stretching the rib cage, rotating the trunk or lateral flexion of the trunk can be evaluated. Furthermore, suitable lengthening of soft tissue around the chest wall and respiratory muscles is related to the efficiency of contraction force and chest movement. In the case of emphysematous lung or air trapping in COPD, abnormal chest configurature and reduced chest movement with shortened muscle length and weakness are experienced. Increasing chest movement with stronger contraction of respiratory muscles can help in gaining lung volume, breathing control and coughing efficiency, and reducing symptoms by improving aerobic capacity, endurance, functional ability, and quality of life.[54]

The thoracic cage is composed of three parts: thoracic spine, ribs, and sternum, which connect to costovertebral and condrosternal joints, and so movement occurs in three dimensions; transverse, antero-posterior and vertical directions. True ribs (2nd to 8th rib) move more flexibly because of no clavicle obstruction, whereas the 11th and 12th ribs connect to the cartilage, therefore causing less freedom to move.[54]

Thoracic Flexion

The basic structure of the costovertebral joint comprises both the angle and neck articulation of the rib with the spine, and is attached to costotransverse and radiate ligaments. In the direction of thorax flexion, there is anterior sagittal rotation, when the costovertebral joint moves as anterior gliding that slightly rotates, whereas downward rotation and gliding occur during extension. The lower thoracic spine moves more freely than the upper one. The sternum is composed of the manubrium, body, and xiphoid process, and is anterior with upward expansion when breathing deeply. In fact, when it comes to movement, the manubrium is somewhat fixed to the first rib, whereas the body is more flexible around the 2nd to 7th rib. Thus, movement of the sternum looks like a hinge joint during deep inspiratory and relaxed expiratory phases.[54]

Thoracic Extension

For extension, the extensor muscle group is the most active, with a motion range of approximately 20-25 degrees. Thorax extension presents the opposite movement to flexion, with backward sagittal rotation by posterior translation and slight distraction of the spine.[54]

Lateral flexion (Side-bending)
In lateral flexion, the thoracic body rotates slightly on the flexion side, while the posterior rotates in the opposite direction so that the costovertebral joint is opened and inferior gliding occurs to increase rib space. Mobility of the thorax on flexion, either to the right or left, is found more in lower than upper thoracic parts. Thus, stretching of the lower thorax is rather more successful than that of the upper part. A normal range of motion is approximately 45 degrees: 25 degrees at the thorax and 20 degrees at the lumbar spines. During lateral flexion to the left, the inferior facet of T6 on the left side moves above the superior facet of the T7 spine. In thorax movement, lateral flexion directly affects the rib space in both approximation and stretch away, which results in the transverse process, when the head of the rib glides in the opposite direction.[54]

Trunk rotation
Trunk rotation is a complex movement that involves many joints. For example, during rotation to the left, three events occur; 1) rib rotation with costotransverse posterior gliding on the rotating side, whereas anterior rotation of the rib and gliding are on the opposite side, 2) thoracic body that is elevated and depressed in each segment, and 3) vertical asymmetrical torsion. Upper thoracic spine can move like pure axial rotation as well as thoracolumbar and cervicothoracic rotation. However, sometimes movement of the upper and lower thoracic spines also co-move with lateral flexion or rotation. Thus, articular facet between high and low spines is a sliding movement.[54]

Assessing Chest Mobility

Observation of respiratory symptoms and chest wall mobility
General screening of respiratory problems can be assessed from the signs or symptoms of respiratory depression such as tachypnea, use of accessory muscles, abnormal breathing pattern, cyanosis, nasal flaring etc. which refer to hard work in breathing. Normal shape of the chest can be observed by the diameter of anterior and lateral views, where the diameter ratio between anterior and lateral measurement should be more than 1.0. However, in the case of COPD, this ratio may be less than 1.0 and the shape is called barrel chest. In COPD, the barrel chest is shown simply from intrapulmonary air trapping or emphysema, which depresses the diaphragm downward and intercostal outward in a shortened position. The shortening of muscle length before inspiration causes insufficient contractile force. Shortness of breath and decreased chest expansion can be observed clinically.[54]

Manual evaluation of chest expansion
Evaluation of chest expansion is relatively simple for the clinician. Various protocols such as the three levels of upper, middle, and lower lobes can be performed manually. Circumferential change from full expiration to maximal inspiration at supine position can be applied with a tape at the axilla (upper lung) and xiphoid (lower lung) levels. Another level that can be measured to present chest expansion by tape is the 4th intercostal rib space. Furthermore, the chest caliper is a new tool that can be used to evaluate chest expansion.[54]

Palpation of the chest wall for flexibility can be evaluated in sitting, side lying, supine, or prone position. Conventional chest movement can be performed with manual evaluation.[54]

Upper costal chest expansion [54]

- Patient position: Sitting.
- Clinician hand position: All finger tips are placed at the upper trapezius with palm on the upper chest above the 4th rib at the mid clavicle line, and the tips of both thumbs close to the midline at the mid- sternum line.
- Command: Gentle compression and order the subject to breathe in deeply and release following chest expansion.
- Results: Approximate calculation of different distances between the tips of thumbs in centimeters (cm) before and after full inspiration.
- Direction: Upper costal expansion should be upward with anterior expansion.

Middle costal chest expansion[54]

- Patient position: Sitting or lying supine.
- Clinician hand position: All finger tips placed at the posterior axillary line with tips of both thumbs close to the horizontal mid line. The palm should be placed on the middle chest area (4th to 6th rib anteriorly at the mid-clavicle line).
- Command: Gentle compression and order the subject to breathe in deeply and release following chest expansion.
- Results: Approximate calculation of different distances between the tips of thumbs in centimeters (cm) before and after full inspiration.
- Direction: Middle chest expansion should be outward and slightly up ward.

Lower costal chest expansion[54]

- Patient position: Sitting.
- Clinician hand position: All finger tips placed at the anterior axillary line with tips of both thumbs close to the horizontal mid line. The palm placed on the lower chest area (below the scapular line and not lower than the 10th rib posteriorly).
- Command: Gentle compression and order the subject to breathe in deeply and release following chest expansion.
- Results: Approximate calculation of different distance between the tips of thumbs in centimeters (cm) before and after full inspiration.
- Direction: Lower costal expansion should be outward.

Sternocostal Movement Evaluation[54]

- Patient position: Sitting
- Clinician hand position: Palm placed to cover all sternum (head and body).
- Command: Gentle compression and order the subject to breathe deeply.

- Result: Anterior expansion during sternum expansion, then upward expansion during sternum (head part) movement.

Tape and caliper evaluation

Both of these methods can be applied in a sitting position, which is better than lying supine. The three levels: upper, middle and lower, can be measured at the axillary, nipple line, and xiphoid process. The anatomic landmarks on the chest wall are as follows[54]:

- Upper thoracic expansion is seen as the third intercostal space at the midclavicular line and the fifth thoracic spinous process.
- Lower thoracic expansion is seen at the tip of the xiphoid process and the 10th thoracic spinous process.

Soft Tissue Flexibility

The theory of Laplace's law suggests that the length of muscle relates to the maximal force of either diaphragm or intercostal muscles, which affect ventilation in the lung.[55] Previous evidence showed that stretching the anterior deltoid and pectoralis major muscles, including the sternocleidomastoid, scalenes, upper and middle fibers of trapezius, levator scapulae, etc., can increase vital capacity.[56] In the case of a patient with COPD, the lower diaphragm is depressed horizontally in a contracted length, thus, the resting length is insufficient for contraction. Tachypnea and dyspnea is then a common sign. Muscle around the chest wall can be divided into two dimensions; anteriorly with pectoralis major and internal or external intercostal muscles; and posteriorly with erector spinae, latissumus dorsi, serratus posterior superior or serratus posterior inferior muscles, which are important for lung ventilation. Thus, retraction or spasm of these soft tissues, or muscles, limits chest expansion.[54]

Chest Mobilization

The chest wall, which is composed of spine, sternum, and ribs, moves in synchronization, no matter whether it is lateral flexion, flexion, extension, or rotation. However, the quality of movement affects individual direction because the costovertebral joint makes contact with the vertebral body, so that lateral expansion is affected more than anterior movement. Whereas, the 2nd to 8th ribs connect to the sternum anteriorly, thus expanding the chest in an anterior direction with pumping handle or anterior and superior motion, as well as bucket handle with lateral and superior motion that occur in regular breathing. The chest mobilization technique is preferred in cases of COPD or chronic lung disease, with the basic theory of mainly improving ventilation. In addition, aging, prolonged use of a ventilator and chronic illness with neuromuscular dysfunction also concern chest wall mobility. Rib torsion, passive stretching, trunk rotation, back extension, lateral flexion and thoracic mobilization are practiced to improve chest flexibility.[54]

Chest mobilization is one of many techniques and very important in conventional chest physical therapy for increasing chest wall mobility and improving ventilation. Either passive or active chest mobilizations help to increase chest wall mobility, flexibility, and thoracic compliance. The mechanism of this technique increases the length of the intercostal muscles and therefore helps in performing effective muscle contraction. The techniques of chest mobilization are composed of rib torsion, lateral stretching, back extension, lateral bending, trunk rotation, etc. This improves the biomechanics of chest movement by enhancing direction of anterior-upward of upper costal and later outward of lower costal movement, including downward of diaphragm directions. Maximal relaxed recoiling of the chest wall helps in achieving effective contraction of each intercostal muscle. Thus, chest mobilization using breathing, respiratory muscle exercise or function training allows clinical benefit in chronic lung disease, especially COPD with lung hyperinflation or barrel-shaped chest. Therefore, the technique of chest mobilization helps increase chest wall flexibility, respiratory muscle function and ventilatory pumping, and results from this relieve both dyspnea symptoms and accessory muscle use.[54]

- **Antero-posterior upper costal chest wall mobilization -** The benefits of this pattern improve both ventilation in the upper lobes and stretches the pectoralis muscle that may be tight.[57]
- **Postero-lateral chest wall mobilization -** It not only affects the ribs and tissue, but also moves the costovertebral and facet joints. This pattern is very useful to improve the ventilation around in the lower lobe of both lungs.[57]
- **Lateral chest wall mobilization -** This pattern helps to improve the chest wall flexibility around the lower thoracic and improves the ventilation in both lower lungs.[57]
- **Thoracic joint mobilization -** Vertebral joints connect to the ribs and sternum with a complex unit that promotes chest expansion. Improving mobility of this joint can prove to have positive effects on ventilation.[57]

Contra-indications for chest mobilization techniques

The contra-indications for chest mobilization are listed below[57]:

- Severe and unstable rib fracture
- Metastasis bone cancer
- Tuberculosis spondylitis
- Severe osteoporosis
- Herniation
- Severe pain
- Unstable vital signs

Nutrition

Patients with advanced COPD and a predominance of emphysema often experience progressive weight loss. The weight loss is multifactorial including a 15% to 25% increase in resting energy expenditure from elevated work of breathing and increased circulatory inflammatory cytokines, higher energy cost of daily activities and a reduced caloric intake. This leads to reduced muscle strength including weakness of respiratory muscles thus worsening the dyspnea. Improved nutrition can restore respiratory and general muscle strength and endurance.[5]

All individuals with COPD should receive educational and nutritional interventions as part of an integrated care plan that seeks to achieve a normal nutritional status, either through natural diet or supplements. Nutrition depletion occurs by multiple mechanisms including energy imbalance, disuse atrophy of the muscles, hypoxemia, systemic inflammation and oxidative stress.[1]

Decreased caloric intake leading to nutritional depletion occurs in about 20-35% of outpatients with COPD and up to 70% of patients with acute respiratory failure or waiting for lung transplantation[58]. Cachexia, defined as weight loss with disproportional fat-free mass wasting, occurs in about one-third of patients with COPD eligible for pulmonary rehabilitation and represents a cause of increased mortality independent of ventilatory limitation.[1]

Decline in nutritional status is directly related to lung function outcomes and has been proposed as a predictor of morbidity and even mortality in patients with chronic respiratory diseases independent of the ventilatory limitation[58]. Furthermore, malnutrition is accompanied by a loss of diaphragmatic and structural skeletal muscle mass, as well as humoral and cellular dysfunction[58]. Anabolic stimulation through a combination of nutritional support and exercise appears to be the best approach to improving functional status.[1]

Surgical Interventions

Bullectomy

Bullae are defined as emphysematous spaces larger than 1 cm in diameter in the inflated lung, usually demarcated from surrounding lung tissue and pathologically consists of enlarged airspaces covered by visceral pleural.[4] Removal of giant bullae has been a standard approach in selected patients for many years. Giant bullae may compress adjacent lung tissue, reducing the blood flow and ventilation to the relatively healthy lung. A bullectomy can produce subjective and objective improvement in selected patients, i.e., those who have bullae that occupy at least 30%—and preferably 50%—of the hemithorax that compress adjacent lung, with an FEV1 of less than 50% of predicted and relatively preserved lung function otherwise[14].[5] The overall success of a

bullectomy is contingent on the size of the bullae, the degree of compression and whether underlying condition of compressed lung parenchyma exists[59]. The reported mortality rates of bullectomy range from 1 to 5%[60] .[4]

Lung Volume Reduction

Lung volume reduction (LVR) is similar to bullectomy, the difference being that LVR is an extension performed for diseases that affect the entire lung. The underlying pathology of end-stage emphysema is characterized by distended airspaces that are inadequately ventilated but with continued perfusion (ventilation-perfusion mismatch) nevertheless, resection of the diseased portions would result in improved ventilation to other functional regions. LVR also serves to re-establish normal chest wall dynamics and may result in improvement in hemodynamic function from the lowering of intra-thoracic pressure throughout the respiratory cycle. Selection of candidates for LVR is dependent on the anatomic characteristics of the diseased portion of the lung with ideal candidates having heterogeneous upper-lobe involvement[61]. There is less dramatic improvement in candidates undergoing LVR with lower lobe involvement[62]. The mortality rate ranges from 0 to 7.5% with varying surgical approaches[63]. Multiple approaches have been used including median sternotomy, bilateral thoracotomies or VATS. All of these have similar results with functional improvement disappearing over a period of 3 to 5 years, but LVR patients continue to have a clinical advantage over medical treatment for those 3-5 years with substantial gains in exercise tolerance, freedom with oxygen therapy, and overall improvement in quality of life.[4]

Lung Transplant

Despite multiple difficulties and obstacles, single-lung transplant has become most common procedure of choice when transplantation is performed for COPD. Available data suggest that lung transplantation offers improved function and HRQOL to patients with advanced COPD, but it is not clear that it offers any survival benefit. Worldwide, COPD is the most common reason for lung transplantation. Current guidelines by the International Society of Heart and Lung Transplantation recommends referring individuals with COPD for transplantation in a scenario with the BODE index greater than 5, post-bronchodilator FEV1 <25 percent of predicted, resting hypoxemia (PaO2 <55 to 60 mmHg), hypercapnia, secondary pulmonary hypertension or accelerated decline in FEV1[14].[5] Lung transplant patients are usually so critically ill that the risk of death from their lung disease enables the actual lung transplant operation to appear quite equitable. The advantages of a lung transplant result in complete replacement of the diseased lung with significant improvements in symptoms. There are however, significant disadvantages to lung transplantation including higher mortality (5 to 15%), lifelong immunosuppression resulting in risks of serious infection and rejection with a cumulative survival rate of around 50%[64].[4]

Complications & Comorbidities

Pneumothorax

Pneumothorax can precipitate severe dyspnea and acute respiratory failure and may be life threatening since they have only a marginal pulmonary reserve. Presence of giant bullae as part of disease predisposes to this complication. It can be difficult to treat if accompanied by a persistent air leak between the involved lung and the pleural space (bronchopleural fistula).[5]

Pulmonary Hypertension and Cor Pulmonale

Both resting and exercise mean pulmonary arterial pressures may be elevated. Prolonged pulmonary hypertension can give rise to a chronic cor pulmonale in late stages. Alveolar hypoxia, respiratory acidosis, remodeling of the pulmonary vasculature with medial hypertrophy of muscular pulmonary arteries, increased viscosity of blood due to erythrocytosis, increased blood volume, left ventricular dysfunction and chronic pulmonary thromboembolic disease can all contribute to the pulmonary hypertension. Correction of hypoxia and acidosis by long-term oxygen therapy and pulmonary vasodilators may slow this process.[5]

Pneumonia

COPD predisposes the lungs to pneumonia as part of acute exacerbation or as discrete event.[5]

Systemic Complications and Co-morbidities

Ischemic cardiac disease is more common in COPD and cardiac events are the single largest cause of mortality in this population[13]. Arrhythmia, congestive heart failure and aortic aneurysm are more common. COPD may lead to a hypercoagulable state due to erythrocytosis and systemic inflammation posing greater risk of stroke, pulmonary embolism and deep vein thrombosis[14]. Weight loss, osteoporosis, skin wrinkling, anemia, fluid retention and depression are some of the other systemic co-morbidities commonly associated with COPD. Major chronic diseases (e.g. congestive heart failure, dementia, ischemic heart disease, stroke, diabetes, cancer, asthma, COPD, depression and hypertension) were associated with at least one of the other diseases in 60–90% of cases.[5]

Quality of Life

The chronic symptoms of COPD (cough, expectoration, wheezing, dyspnea and exercise tolerance) are the major factors responsible for altering the relationship between health and quality of life.

Studies of health-related quality of life (HRQoL) in patients with COPD with varying degrees of severity have consistently shown that patients have significant decrements in HRQoL[65]. Therefore, HRQoL is an important clinical outcome in COPD. The St

George´s Respiratory Questionnaire (SGRQ)[66] and The Chronic Respiratory Disease Questionnaire (CRQ)[67] are the primary questionnaires used to measure the quality of life in COPD patients.[6]

The St George's Respiratory Questionnaire (SGRQ)

The Saint George Respiratory Questionnaire (SGRQ) is the best-known and most frequently used disease-specific health related quality of life (HRQL) questionnaire for respiratory diseases[32]. The SGRQ is a standardized, self-administered questionnaire for measuring impaired health and perceived HRQL in airways disease. It contains 50 items, divided into three domains: Symptoms, Activity and Impacts. A score is calculated for each domain and a total score, including all items, is also calculated. Each item has an empirically derived weight. Low scores indicate a better HRQL[68].[2]

Psychosocial Implications

COPD is associated with increased risk for anxiety, depression, and other mental health disorders[69]. Psychiatric disorders are at least three times higher in COPD patients compared to the general population[70].[2]

Stigma of COPD

For healthy individuals, the habitual functions of breathing, walking and moving the body in meaningful ways are unconsciously undertaken as the body moves purposefully towards its tasks. Sputum production, uncontrolled coughing and wheezing and urinary urgency are examples of changed body behaviors that signify a loss of control, displaying the body in socially unacceptable ways. Because these behaviors draw attention to the person's unpredictable body, they detract from enjoyable participation in family and community activities. This has been described as the 'stigma' of COPD where the illness is visible, and is associated with disability and lack of control[71]. The visibility of the illness challenges the person's personal integrity and sense of effectiveness.[22]

Loss of Spontaneous Mobility

People with healthy bodies combine their movements and activities in a fluid manner. They spontaneously act in response to sensory stimuli, or to a perceived need to attend to a particular task, and this rarely requires a conscious appraisal of the body's capacity. People with COPD lose this spontaneous application of the body to its tasks; in fact, a lack of forward planning can leave the person gasping for breath. Simple activities such as walking and talking become difficult to combine[72]. Attending to day-to-day activities means pacing the body and spacing out activities that tax the body's breathing. Pacing of movement and activities with frequent breaks and aligning activities into sequential rather than combined tasks allows the person to recover their breathing along the way. Because of the daily variability in symptoms, people may need to take on a flexible

approach to assessing, on the day, outings they have planned in advance[73]. Those who adjust most effectively to their bodily restrictions listen to their body, plan, pace, prioritize and balance their activity with capacity on that day, and try hard to achieve a certain level of contribution within realistic parameters[74].[22]

Loss of Personal Productivity

Leidy & Haase noted physical effectiveness as a core component of personal integrity that is challenged in COPD[76]. Effectiveness is expressed as 'being able'; the body's predictability in doing what we expect or desire it to do. In sharp contrast, the failing body in COPD is nothing like what is presumed for, or wished of the body[75]. Physical effectiveness is just as much an interpersonal process that includes doing for others, as well as for one's self. This notion of contribution is an important one to most well socialized adults. When the ability to contribute is lost to ineffectiveness and dependence, then people feel shame, self-blame and perceive the blame of others[74].[22]

COPD symptoms often begin during a person's productive, working life. For many, there is an assumed level of physical adeptness and a physical and aesthetic appearance that has constituted their body as it is known to themselves in its predictability, and known to others in its apparent wholeness and application to visible tasks. For men, heavier household tasks such as mowing lawns and managing gardens are frequently tied to their own and their family's perception of them in their gendered roles. For men, these heavier tasks are eventually taken over by another family member, or by paid help. For women with COPD, there tends to be a sense of ownership and obligation towards housework, and they will tolerate significant symptoms to retain these duties. As the disease progresses there are often visible changes such as development of the classic 'barrel chest', significant weight loss and for some, facial and postural changes from prolonged steroid use. The net result of this changed capacity and appearance is that people lose a variety of modes of self-expression[76]. Each task is considered as to whether the reward, for themselves or others, will outweigh any distressing symptoms. If the real or anticipated discomfort is thought to be greater than the perceived benefit, that task will be avoided. Rewards include either personal pleasure and fulfilment, or a task that is to the benefit or welfare of others[76].[22]

Loss of Independence

Severe COPD sees people coming to terms with their diminishing ability to care for themselves. Early losses in independence may include difficulty with shopping or driving. As the disease progresses, people find that basic tasks such as showering and dressing may become insurmountable, making them feel almost child-like in their dependence on others[77]. This loss of independence with self-care is an enormous threat to people's sense of hope[78]. Showering causes particular problems because of the effect of steam on breathlessness. Lifting arms to wash the hair, or bending to dry the feet are movements that cause considerable restriction to breathing, and so may be avoided. For people who live alone, this loss of self-care may herald their movement

into residential care. For people with family caregivers, it may alter the existing family relationship dynamics[77].[22]

Loss of Self Image

There is an important temporal framework to the experience of COPD with the visions of past, present and future selves being held in constant comparison to each other. These gradual changes from independence to dependence lead to loss of self-esteem, loss of self-image and loss of power.[75] The disease creates an otherness where the more visible 'medical self' is separate from the real self. Their future is seen in terms of loss: loss of anticipated retirement, loss of hoped for relationships with children and grandchildren[75], and loss of 'possible selves' which are no longer conceivable[72]. This loss of independence and loss of family and community roles frequently lead to frustration, irritability and depression[79]. Those with advanced disease may see their life as meaningless. They communicate hopelessness, worthlessness and resignation and this can make death seem like an attractive option[74]. Despite the extremes of emotions, people try to contain their feelings as emotional turmoil can bring on exacerbations of breathlessness that are difficult to recover from. This has long been recognized and described as living within an 'emotional straight jacket' with both positive emotions such as laughter, and negative emotions such as anger, leading to distressing dyspnea[80]. Hypoxia may result in cognitive and personality changes that can further isolate people from family and others in the community. These may manifest as hallucinations, confusion, memory loss or unreasonable and unsociable behavior[81].[22]

Social Isolation

People with COPD and their close family members live within a shrinking life-world. The physical boundaries of their life are diminished as the sick person begins to avoid taxing outings and spends the majority of their time within their own four walls. People become socially isolated as they avoid environments and situations that may trigger breathlessness. Their consciousness of the socially unacceptable nature of their coughing and spitting makes them reluctant to enter new social situations. People reliant on home oxygen concentrators may be literally tied to an electrical power source and this increases isolation for the patient and the complexity of care for the family[81] (Boyle, 2009b). People lose shared experiences with family and friends leading to loneliness, sadness and abandonment as they not only avoid social activities but feel they are avoided by others[82].[22]

Living with Crises

COPD is often experienced as relatively quiet times interrupted by episodes of serious illness. Episodic crises create the essence of uncertainty that defines the experience of COPD. These episodes are often described by patients as near-death experiences that leave people with a constant sense of their own possible death. This has been described as 'living in the proximity of death'.[74] Crises may be the result of panic

attacks, acute chest infections, allergic reactions or acute emergencies related to comorbidities. The crisis events begin with dyspnea that does not respond to the usual self-management strategies. Initially, people may feel the need to be on their own during acute breathlessness, sensing that others can't help bring dyspnea under control and that there is a need to focus internally on breathing and maintaining calm[83]. Although the onset of exacerbation is recognized with panic and dread, people are often reluctant to seek help, hoping things will improve and hospital admission will be avoided. Professional assistance is sought only after people are convinced they can't self-manage the event.

As respiratory distress increases and panic rises, people may change in appearance, may be unable to speak and may experience choking and loss of bladder or bowel control[77]. These understandably terrifying events usually lead to emergency hospital admission. These crises are watershed events that mark a 'before' and 'after' in the person and family's life from which other events are then measured[84]. These crises underline life with COPD as uncertain and unpredictable and people fear each attack could be their last[81]. The experience reinforces the conviction of caregivers that they must closely monitor the person for early signs of deterioration, and this vigilance thereafter binds them emotionally and practically to the task of caring[77]. People will often develop emergency protocols that may define triggers for help-seeking and roles for family members that require 'understanding and trustworthiness' amongst those individuals[84].[22]

Caregivers

The confining nature of COPD extends to the family caregiver. As the physical effectiveness of the ill person declines, the workload of close family members increases. In the case of older couples, the primary caregiver may be facing their own health and ageing issues and the role of caring can seem overwhelming. The fear that something may happen to their loved one in their absence means that they become bound, physically to the home and psychologically to the role of caring due to a perceived need for increased vigilance. Their need to closely monitor their loved one leads to the use of phones and intercoms, listening to breathing during the night, watching for early signs of exacerbation and using the current level of breathlessness as a gauge of capacity for tasks[81]. The experience of caring differs between spouses and other family members. The reciprocal nature of most marital relationships places caring in a framework of the historical give-and-take between partners and is sealed with the understanding of "for better or worse". Amongst younger caregivers, caring may be challenged by the competing roles of working and parenting and a different level of perceived reciprocity[77]. Children and siblings are more likely to find the caring burdensome, and to note the lack of caring input from other family members[77]. Caregivers with a higher level of education may find it more difficult to accept the loss of independence[85]. Family enmeshment also makes adjustment to illness more difficult. When people weave their identities and

activities around each another so completely it is difficult for any one member to function independently[86].[22]

Caregivers often feel weighed down by their multiple roles and feel similar losses of shared social experiences[87]. The caring role may coincide with a time of both declining health and fitness and increasing heaviness of the work of nursing. Women caregivers in particular are prone to somatic symptoms and anxiety, and although taken for granted, the frequent interruptions to sleep can be wearing[81]. While some caregivers manage to integrate caring with employment to provide some personal time and space, others are forced into an unwelcomed, early retirement[81]. It is known that for people who are unable to leave the home for some sort of personal pursuit, there is a higher perceived burden of care[81]. These losses of social participation for caregivers may contribute to a loss of self-identity with some women becoming unable to separate a sense of themselves from their husbands. Their future hopes for meaningful pursuits and achievements, a relaxed lifestyle and personal freedom become lost in the daily grind of their present reality[81]. The majority of social interaction for caregivers is with the ill person; however, males with COPD tend to isolate themselves from conversation, have a reduced interest in things, and as a consequence, have little to talk about[87]. This loss of intimacy through conversation is paralleled with a loss of physical intimacy, including sexual interaction[77]. Where intercourse is attempted it may be frightening with distressing breathlessness distracting both partners from the romanticism or eroticism of the moment. Whilst for many couples, sex becomes less important, other forms of intimate physical contact is also avoided so that simple loving gestures such as cuddling or kissing may be lost to the caregiving spouse.[22]

Much of the caring literature on COPD focuses on female spouses. However, where both men and women are participants there appears to be a difference in caring styles and responses to caring. Women caregivers, in particular, take on a micro-management approach, arranging medical appointments and scrutinizing diet, medication and exercise compliance and this differs from the more passive and delegatory style of male caregivers.[22]

End of Life

End-of-life planning in COPD is an important concept that allows goal-setting for patients and families, and facilitates a peaceful and dignified death. Specialist referral to palliative care services, in combination with a partnership approach with patients and families, allows the person to retain control over aspects of the experience of dying in the context of an otherwise uncontrollable illness course. One of the great difficulties of planning the timing of end-of-life discussions is the uncertain disease trajectory in chronic respiratory conditions. COPD has not only an insidious onset, but also, an unchartable end-stage. We know that compared to patients with lung cancer, COPD patients have more Emergency Department admissions, more anxiety & depression,

and report a lower quality of life. Compared to cancer patients, financial support comes later in the disease process and patients feel in greater need of aids and appliances, and of information on services and benefits[88]. COPD patients are less likely to receive prognostic information, less likely to know they are dying, or know they are dying for less time and they are more likely than lung cancer patients to die in hospital. Relatives of COPD patients are less likely to be present at the time of death, although, we know most would like to be present[89]. Many clinicians may not think about COPD as a terminal disease and so may not consider a palliative management plan. They do not tend to talk about what dying may be like or how long that may take[90]. The tendency for people to bounce back has led to practitioners considering how best to define the time for end-of-life discussions and interventions. For clinicians, the final phase of life may be suggested by an FEV1 less than 30% of predicted, frequent exacerbations and admissions to hospital, and the presence of right heart failure. The need for mechanical ventilation and long-term oxygen therapy dependence also signal serious disease. However, such markers are not always reliable predictors of the terminal phase of COPD[91]. Patients may have their own interpretation of the time when treatment is no-longer worth the burden that continued life presents. Scenarios that include prolongation of inevitable death, dependence on machinery, functional and cognitive impairment, unmanageable symptoms and a burden on loved ones have been noted as unacceptable by patients[92].[22]

Another complicating factor is that what is acceptable to patients may change over time as they adjust to severe illness and this may influence discussions and the willingness of clinicians to initiate advance care planning. It is typical of people to normalize their experience of even severe day-to-day symptoms and see themselves as sick only during acute exacerbations. This may be, in part, a coping strategy, but is also a result of the long illness trajectory. Frequently, in cancer narratives, there is a definite beginning and developing plot to the ‘cancer story’, COPD is more likely to be insidious in its beginning and intertwined with the person’s ‘life story’. The unpredictability of exacerbations creates a chaotic component to the person’s experience of illness, yet they may have a sense of relative wellness between these crises[93]. Individuals may feel that each acute exacerbation may be their last, however, the threat of death recedes after a COPD crisis, or perhaps the threat of death is also normalized. The result is that death is less likely to be considered imminent and so wishes are rarely discussed with professionals, friends or family[93]. Where end-of-life discussions do occur, they may be poorly documented and so patient wishes may not be visible to family or other members of a multidisciplinary team[88]. Having end-of-life discussions with COPD patients and families constitutes significant emotional work for clinicians and requires ‘conscious emotional management’. This comes with experience as professionals learn to feel their way with an individual, and apply emotional intelligence and empathetic skills in their discussions[88]. Some ways to approach these difficult conversations include beginning discussions early in the disease course, using the uncertain disease trajectory to ease discussions and building a caring and respectful relationship with patients. It is useful to have a team approach to proactively identify and use opportunities to talk about prognosis. The aim of good end-of-life discussions is to inform without removing hope,

and to bring to the forefront the wishes of the patient and family. Discussing prognosis broadly in terms of a diagnostic population rather than directing it at the individual leaves room for hopeful possibilities.[22]

Clinicians can foster hope by giving a 'commitment to non-abandonment', by addressing people's fears, such as fear of pain at end-of-life, and by having a management plan that addresses their changing situation[94]. Helping people to identify realistic goals and discussing their concerns about day-to-day living can also be useful. The ideal is for a formal Advance Care Plan to be documented early. Again, the uncertain disease course of COPD makes this more complex, and means physicians are less comfortable with initiating such plans. The process can be simplified by creating possibilities for revision of the plan, and by trying to understand and be true to the patient's core values whilst remaining flexible around practical details such as where they would prefer to die.[22]

One marker of the end-of life stage may be the point where maximal therapy no longer provides relief of symptoms. Symptoms in the last year of life are characterized by constant breathlessness, weakness and fatigue. Pain, insomnia, depression, anxiety and panic attacks also shape the patient experience at this stage[95]. This requires a change in priorities of care, with symptom management needing the greatest focus. For example, in late-stage disease opioids may be central to dealing with dyspnea, dyspnea-related anxiety and pain. Clinician concerns around respiratory depression may lead to the underutilization of opioids[96]. This may require a change in our understanding of what is 'good' or 'safe' for patients at different stages of their illness experience. The COPD journey is a long and consuming one both for the person with the disease, and for the family. While this may set up challenges for clinicians in understanding and supporting psychosocial concerns, it also creates possibilities for true management partnerships with our patients and their families.[22]

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Chronic Obstructive Pulmonary Disease

Post-Test

1. Most of the major pathophysiologic changes associated with advanced COPD are attributed to systemic inflammation. (p. 3) A. True B. False

2. Which of the following is the most significant risk factor for COPD? (p. 4)
   A. Cigarette smoking
   B. Occupational exposure to air pollutants
   C. Low birth weight
   D. Vitamin C deficiency

3. Morbidity and mortality rates have been shown to be inversely related to socioeconomic status. (p. 5) A. True B. False

4. Most patients initially present with COPD in the ___ decade of life when they have _____. (p. 7)
   A. fourth; a chronic non-productive cough
   B. fifth or sixth; dyspnea with mild exertion
   C. third or fourth; wheezing and viscous sputum
   D. fifth; pleural pain and generalized muscle wasting

5. A spirometer measures _____. (p. 8)
   A. Inspiratory volume
   B. How quickly and efficiently the lungs can be emptied
   C. Maximum lung residual volume
   D. Oxygen saturation of pulmonary tissue

6. Chest radiography and CT-scans are required to make a diagnosis of COPD. (p. 9) A. True B. False

7. Which of the following is a self-administered dyspnea questionnaire that measures physical, mental, and social function? (p. 11-12)
   A. Baseline Dyspnea Index
   B. The Oxygen Cost Diagram
   C. The Borg Scale
   D. The Pulmonary Functional Status Scale

8. A person has a FEV1/FVC of 68% and a FEV1 of 42%. Their status would be classified as _____. (p. 13-14)
   A. GOLD Stage II
   B. GOLD Stage III
   C. BODE Index Score 4
   D. BODE Index Score 7

9. Which of the following statements is TRUE? (p. 14-16)
   A. Beta 2-agonists and anti-cholinergics are injectable corticosteroids.
   B. Inhaled glucocorticoids cause bronchodilation.
   C. Long term oxygen therapy reduces pulmonary hypertension.
   D. Systemic steroids are the single most effective therapy for the majority of COPD patients.

10. Which type of exercise would be most appropriate for an individual with severe dyspnea? (p. 19-22)
    A. Continuous aerobic
    B. Interval aerobic
    C. Continuous anaerobic
    D. Interval anaerobic

11. An individual has an anterior/lateral chest diameter ratio of less than 1. This is commonly referred to as a _____. (p. 24)
    A. Barrel chest
    B. Bucket deformity
    C. Flail chest
    D. Costal collapse

12. Weight loss with disproportional fat-free mass wasting is known as _____, and occurs in about ____ of patients with COPD. (p. 28)
    A. calciphylaxis; one half
    B. pertussis; 22%
    C. myorexia; 33%
    D. cachexia; one third

13. Which of the following statements is FALSE? (p.28-31)
    A. Bullae are small fluid filled spaces in the lung less than 1 cm in diameter.
    B. COPD predisposes the lungs to pneumonia.
    C. The St. George's Respiratory Questionnaire is used to assess quality of life.
    D. Individuals with COPD experience a higher rate of psychiatric disorders than the general population.

14. COPD related hypoxia may result in cognitive and personality changes. (p. 33) A. True B. False

15. The need for mechanical ventilation and long-term oxygen therapy are always reliable predictors of the terminal phase of COPD. (p. 36) A. True B. False

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