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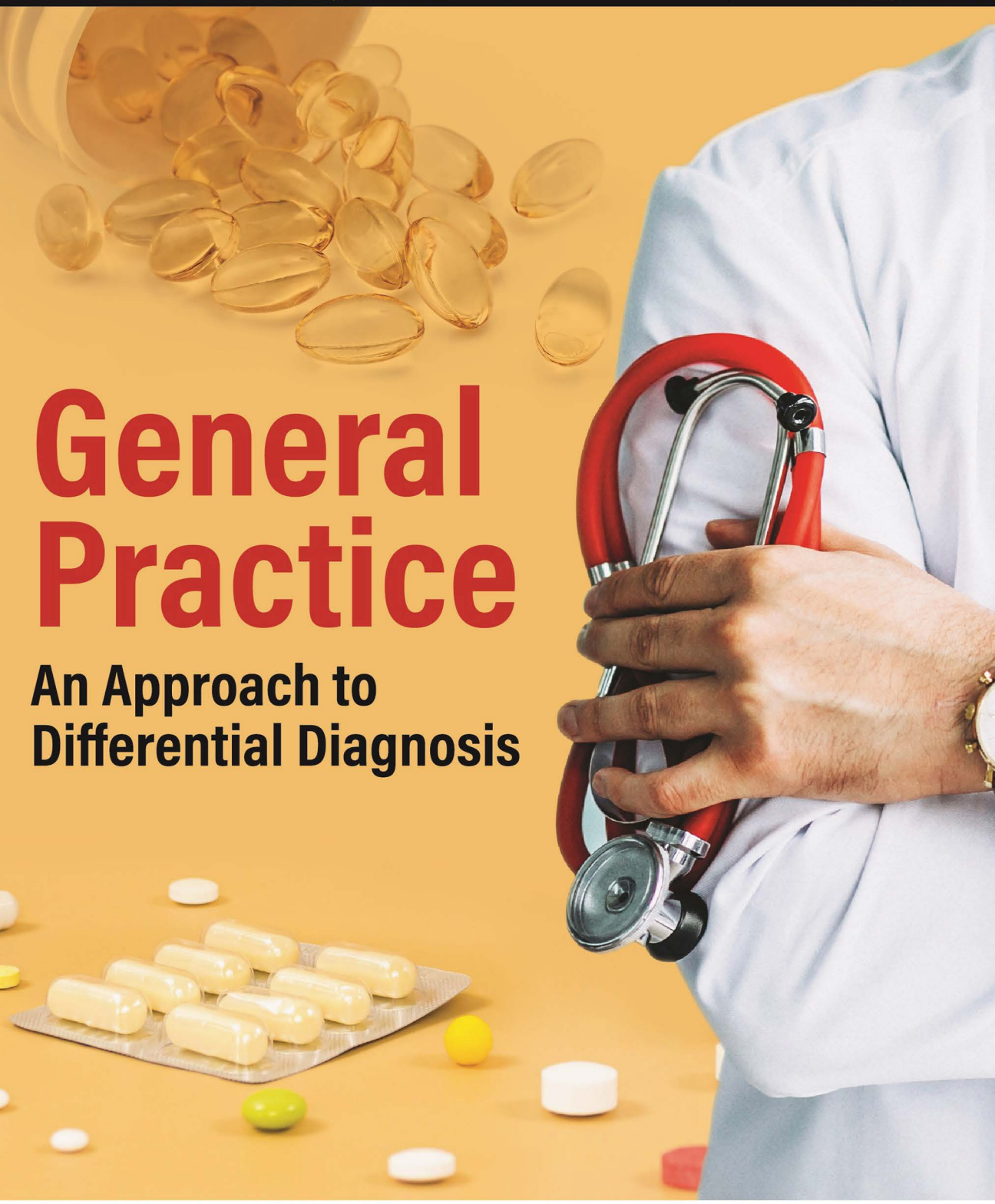

General Practice

An Approach to Differential Diagnosis

Jyotirmoy Pal
Nandini Chatterjee

JAYPEE

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CHAPTER 2

Dyspnea

Supriya Sarkar, Nandini Chatterjee

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INTRODUCTION

Unlike cardiac activities, respiratory activities can be controlled voluntarily. Yet, we are not aware of our breathing as we take breathing for granted. But when we cannot breathe, nothing else matters. Dyspnea is classically described as unusually uncomfortable awareness of breathing. It may be described as chest tightness or constriction, increased work or effort of breathing, air hunger, need to breathe, urge to breathe, cannot get a deep breath, unsatisfying breath, heavy breathing, rapid breathing, or breathing more. We must be convinced about the symptom of dyspnea. For example, catch of breath may be due to pleuritic chest pain or heaviness of chest may be due to accumulation of pleural fluid, and these should be differentiated from dyspnea. It is always useful to understand what a patient means rather than what the patient describes.

WHAT IS DYSPNEA?

The American Thoracic Society defines dyspnea as a "subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. The experience derives from interactions among multiple physiological, psychological, social, and environmental factors and may induce secondary physiological and behavioral responses."

Dyspnea is a symptom and must be distinguished from the signs of increased work of breathing. Signs of increased work of breathing include increased rate and depth of respiration and use of accessory muscles of respiration—intercostal, suprasternal, and supraclavicular suction. As dyspnea is a subjective symptom, some patients may malinger, some may complain disproportionately of increased dyspnea as compared with his disease, and some patients may not complain even when they have signs of increased work of breathing (probably they ignore it or may be habituated with it or may accommodate dyspnea with lifestyle modification).

MECHANISM OF DYSPNEA

Receptors

As with other sensations, the sensation of dyspnea starts from receptors. These receptors are present in the airways, lung parenchyma, respiratory muscles, and other areas.

Chemoreceptors (both central and peripheral) can sense the changes in partial pressure of CO_2, O_2, and pH and ensure that alveolar ventilation can adapt to the metabolic changes. Hypoxia can cause dyspnea indirectly by increasing ventilatory drive. The mechanism of direct effect of hypoxia is poorly understood. It may be due to perceiving the sensation as it reaches directly to the level of consciousness. The direct effect may explain the beneficial role of oxygen on dyspnea. Hypoxic hyperventilation may cause air trapping and dynamic hyperinflation, which may produce dyspnea.

Pulmonary receptors are slowly adapting stretch receptors (SASRs), rapidly adapting receptors (RARs), and juxtapulmonary (J) receptors. SASRs present in large airways respond to increase in lung volume. RARs present in airway epithelium respond to particulate irritations, direct stimulations of the airways, and pulmonary congestion. Juxtapulmonary receptors present in the bronchial and laryngeal mucosa and pulmonary capillaries are nonmyelinated fibers. They are stimulated by different mechanical and chemical stimuli. Sensations from those pulmonary receptors are carried to the central respiratory centers via the vagus nerve. Pulmonary receptors, particularly juxtapulmonary receptors, are implicated as the generator of dyspnea.

Mechanoreceptors are mainly present in the respiratory muscles and have a negative effect on dyspnea. Stimulation of mechanoreceptors, contrary to our belief, generates a negative impact on the genesis of dyspnea and reduces its sensation. Mechanoreceptors for volume, flow, muscle shortening, muscle tension, and chest wall displacement provide peripheral sensory feedback. Ultimately, the intensity of central motor output at the central sensory level is modulated. Inspiratory chest wall vibration reduces the sense of dyspnea, whereas expiratory vibration has an opposite effect.

Respiratory Effort and Central Corollary Discharge

Dyspnea is said to be arising from greater respiratory muscle activity or effort. Sense of effort is a central sensation related to increased central motor command, muscle load, or when muscles are weakened, fatigued, or paralyzed. When ventilation is increased, the increased motor command is sent to ventilatory muscles and the motor cortex. Simultaneously, a copy signal is sent to sensory cortex, which is termed central corollary discharge. A corollary discharge is also sent from brainstem centers, but they are weak, and they are usually sensed as air hunger. The sense of muscle effort and corollary discharge is dependent on the absolute magnitude and duration of load. Relative magnitude of load, compared with the maximum capacity of muscles, is also an important determinant of corollary discharge.

Neuroventilatory Dissociation

We get distress when we achieve less than what we deserve. Similarly, dyspnea results from interaction between efferent and afferent signals, the neuroventilatory dissociation. Dyspnea occurs when more than expected respiratory effort is necessary to achieve a given amount of ventilation. The phenomenon is also described as length–tension inappropriateness, where length represents change in lung volume and in other way the ventilation whereas tension indicates respiratory muscle tension and in other way respiratory effort. Misbalance between them produces the sensation of dyspnea.

Role of Central Nervous System

Dyspnea results from simultaneous and multiple sensory inputs. The role of the central nervous system is important in the integration of those complex sensations. Dyspnea arises from three distinct brain areas: Right anterior

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insula, cerebellar vermis, and medial pons. The sensation of dyspnea results from the sensorimotor integration and the modulation of the perceived intensity.

WHAT ARE THE CAUSES OF DYSPNEA?

Dyspnea may occur due to increased work of breathing as a result of airway obstruction, stiff lung or hyperinflated lung, increased respiratory activity as a result of increased need of ventilation, and stimulation of receptors within lungs, pulmonary vessels, and extravascular structures. Cardiovascular diseases may cause dyspnea by accumulation of fluid in lungs. Some noncardiac/nonrespiratory conditions may also cause dyspnea. Finally, dyspnea may be psychological in origin.

Dyspnea in Airway Diseases

Both asthma and chronic obstructive pulmonary disease (COPD) can cause dyspnea by expiratory airflow obstruction and dynamic hyperinflation of the lungs and chest wall, thereby increasing the work of breathing. Patients with acute bronchoconstriction may complain of a sense of tightness of chest. A ventilation–perfusion mismatch in obstructive airway diseases may cause hypoxemia, thereby increasing respiratory effort.

Classically, dyspnea in COPD is persistent (without a symptomless period) and progressive (increases its intensity relentlessly) with periods of exacerbations (periods of increased symptoms). On the other hand, dyspnea in asthma is usually episodic in nature with symptom-free intervals. Dyspnea of chronic asthma may have the same characteristics as those of COPD. Dyspnea of airway diseases is usually associated with wheeze (a musical wheezy sound heard by naked ears).

Dyspnea of Lung Parenchymal Diseases

Diseases involving interstitium and lung parenchyma due to several mechanisms (infections, occupational dust exposures, autoimmune disorders, or by unknown mechanisms) cause stiffness of lung. As a result, lung compliance (distensibility of lungs) decreases and thereby work of breath increases. Hypoxemia due to ventilation–perfusion mismatch increases ventilatory drive. Stimulation of pulmonary receptors is also responsible for the pathogenesis of dyspnea in mild-to-moderate interstitial lung diseases.

Diseases of the Chest Wall and Pleura

Diseases of the chest wall (kyphoscoliosis), weakness of respiratory muscles (myasthenia gravis or Guillain–Barré syndrome), and stiffness of chest wall (scleroderma) increase the effort of breathing. Similarly, pleural diseases (effusion or pneumothorax) cause dyspnea by increasing the work of breathing and by stimulating pulmonary receptors (due to underlying atelectasis).

Dyspnea due to Diseases of Cardiovascular System

Left ventricular diseases resulting from coronary artery diseases, hypertensive heart diseases, and nonischemic cardiomyopathies cause pulmonary interstitial edema and thereby stimulate pulmonary receptors. Dyspnea may be augmented by hypoxemia (as a result of ventilation–perfusion mismatch) and increased ventilatory drive. Constrictive pericarditis and cardiac tamponade increase intracardiac pressure and pulmonary vascular pressures, and thereby cause dyspnea.

Pulmonary thromboembolic diseases and primary diseases of the pulmonary circulation

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(primary pulmonary hypertension and pulmonary vasculitis) can cause dyspnea. The underlying mechanism is thought to be due to stimulation of receptors in pulmonary vessels. Hypoxemia and hyperventilation may augment that process.

Other Causes of Dyspnea

Anemia is associated with dyspnea, which may be due to stimulation of metaboreceptors. Dyspnea may be observed in hyperthyroidism as a result of increased metabolic activities and in hypothyroidism as a result of pleural or pericardial effusion.

HOW TO APPROACH DYSPNEA?

First Step

Firstly, we should identify different expressions of dyspnea as dyspnea may be expressed in different words according to a patient's understanding. It is also important to be sure whether the patient is actually complaining of dyspnea. Expressions of other symptoms may mimic those of dyspnea. For example, a catch of breath may be due to pleuritic chest pain. It is also important to recognize dyspnea even when a patient is not complaining about it. A patient with arthritis may have limited activities, and thereby he/she cannot recognize his/her symptoms. A patient with chronic dyspnea may adapt that problem by modifying his/her lifestyle.

Second Step

Secondly, respiratory origin of dyspnea should be characterized by presence of seasonality and wheezing, be to differentiate from dyspnea arising from other diseases. Anemia, hyperthyroidism, or hypothyroidism can be easily identified clinically as well as with simple laboratory tests.

Psychological Dyspnea

Psychological dyspnea is usually identified by detailed history, a patient's background, and meticulous observation of the patient's behavioral pattern. But in some cases, it is difficult to recognize, and then it is important to rule out organic diseases before diagnosing it as functional. It is also important to remember that psychological factors may precipitate attacks of dyspnea in organic diseases. For example, attacks of asthma may be precipitated by psychological stress.

Distinguishing Cardiovascular Dyspnea from Respiratory Dyspnea

The differentiation is often difficult and sometimes may be impossible. Yet, an endeavor in this regard is always beneficial in clinical medicine. Differentiating dyspnea from respiratory diseases and cardiac diseases is an important step. The differentiation is often difficult as cardiac diseases cause dyspnea mainly by accumulation of fluid in lungs. Cardiac origin of dyspnea should be suspected if there is a history suggestive of cardiac disease, history of hypertension, ischemic heart disease, presence of palpitation, precordial chest pain, and history suggestive of paroxysmal nocturnal dyspnea. On the other hand, the presence of wheeze, cough with expectoration, hemoptysis, and nonprecordial chest pain is suggestive of respiratory origin of dyspnea. Wheeze may be found in left ventricular failure due to accumulation of fluid in the peribronchovascular spaces. Cough may be found in both conditions, but expectoration (mucoid and mucopurulent) is suggestive of respiratory disease and pink frothy expectoration is suggestive of left ventricular failure. Frank hemoptysis is always suggestive of respiratory diseases except mitral valvular diseases.

High blood pressure may suggest cardiac origin, but it is nonspecific. Physical findings such as gallop rhythm, murmur, abnormal heart sounds, and fine basal crackles are suggestive of cardiac origin of dyspnea. Right ventricular gallop and murmur arising from tricuspid and pulmonary valves may occur in cor pulmonale and are indicative of a respiratory cause of dyspnea. Basal crackles

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are also found in respiratory diseases, particularly in diffuse parenchymal lung diseases.

Cardiopulmonary exercise test may be an effective tool in differentiating respiratory from cardiac origin of dyspnea. Respiratory origin is evident when at peak exercise the patient demonstrates hypoxemia or the patient develops bronchospasm. Cardiac origin is suggested if the heart rate becomes >85% of predicted maximum, anaerobic threshold occurs early, blood pressure is extremely high or low, O_2 consumption per heart rate falls, or there is appearance of ischemic changes in electrocardiogram (ECG).

Third Step

A detailed history taking has no substitute in the analysis of dyspnea. The onset of dyspnea is important as sudden onset usually indicates a vascular phenomenon (acute myocardial infarction and pulmonary thromboembolism) or pneumothorax. In children, sudden onset of dyspnea may occur in foreign body inhalation. Longer duration of dyspnea usually suggests obstructive airway diseases, interstitial lung diseases, chronic heart failure, or cardiomyopathy. Shorter duration of dyspnea may occur in infective diseases, pleural diseases, or acute cardiac diseases. Dyspnea may increase rapidly in tension pneumothorax, acute myocardial infarction, or gradually in pleural effusion or cardiomyopathy. Episodic dyspnea (with symptom-free intervals) usually suggests asthma, bronchospasm, intermittent myocardial ischemia, pulmonary embolism, etc. Acute increase in dyspnea in patients with relentlessly progressive dyspnea is suggestive of exacerbations of COPDs or interstitial lung diseases.

Dyspnea with wheeze usually suggests obstructive airway diseases. Nocturnal dyspnea can occur in left ventricular failure (due to increased venous return) and asthma (due to presence of allergens in bed). Exertional dyspnea is nonspecific and it only gives an early indication of respiratory or cardiac diseases. Orthopnea occurs in conditions such as congestive cardiac failure, gastroesophageal reflux diseases, and obesity. Platypnea (dyspnea in an upright position that relieve in supine position) occurs in left atrial myxoma or hepatopulmonary syndrome. Smoking history may be associated with both COPD and ischemic heart disease.

Fourth Step

A meticulous physical examination may give important clues to the diagnosis. General survey should focus on pallor, cyanosis, jaundice, clubbing, neck vein, edema, etc. Signs of dyspnea such as tachypnea, use of accessory muscles of respiration, supraclavicular and intercostal retractions, and tripod position (sitting with one's hands braced on the knees) are important to note. Different breathing patterns (Cheyne–Stokes breathing, Biot's breathing, Kussmaul's breathing, etc.) should be appreciated. Pulsus paradoxus may be found in COPD or asthma. Examination of thorax (symmetry of hemithorax, movement of thorax, mediastinal position, percussion notes, breath sounds, added sounds, and vocal resonance) should be carefully done to diagnose the pathological changes of lungs, pleura, or thoracic cage. Engorgement of neck veins and their pulsatility are important and reflect right-sided cardiac events and diseases of superior vena cava. Cardiac examination should include examination of blood pressure, pulses, jugular veins, abnormalities of heart sounds, and murmurs. Abdomen should be carefully examined for paradoxical movement. Raynaud's phenomenon may suggest collagen vascular diseases.

Fifth Step

Investigations should depend on clinical clues and clinical settings. They depend on the suspected origin of dyspnea. Routine blood examination, thyroid function tests, liver function tests, and lipid profile will eliminate

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anemia, thyroid disorders, hepatopulmonary syndrome, and risk of cardiovascular diseases.

When the cardiac cause of dyspnea is suspected, then the investigation should start with ECG and echocardiography. Special cardiac investigations such as 24-hour Holter monitoring or cardiac angiography should be done on a case-to-case basis. When respiratory causes are suspected, then investigations should start with chest X-ray posteroanterior (PA) view, spirometry, and sputum examination. High-resolution computed tomography (HRCT) of thorax should be done if preliminary investigations suggest interstitial lung disease, parenchymal lung disease, or airway disease. Contrast-enhanced computed tomography (CECT) of thorax should be done for pleural diseases, lung malignancies, and mediastinal diseases. Pleural fluid examination is an essential step for pleural effusion. It should be remembered that the most common cause of bilateral pleural effusion is congestive cardiac failure. Measurement of diffusion capacity of the lungs for carbon monoxide (DLCO) should be done for interstitial lung diseases and obstructive airway diseases. Bronchoprovocation test is rarely required for the diagnosis of asthma when symptoms are intermittent and spirometry is normal.

When no clue is available from history and clinical examination, then apart from routine blood biochemistry, chest X-ray PA view, ECG, echocardiography, and HRCT thorax should be done to start with. The next step will be determined by the results of preliminary investigation results.

Sixth Step

It is important to assess the severity of dyspnea. Generally, dyspnea is classified as stage I (dyspnea occurring with more than habitual exercise), stage II (with habitual exercise), stage III (with less than habitual exercise), and stage IV (dyspnea occurring at rest). The Modified Medical Research Council (mMRC) dyspnea scale **(Table 1)** is widely used and standardized. A patient's descriptions of dyspnea are graded as "I only get breathlessness with strenuous exercise" (mMRC grade 0); "I get short of breath when hurrying on the level

Table 1: Modified Medical Research Council (mMRC) scale for dyspnea.

Please tick in the box that applied to you (one box only) (Grades 0–4)		
mMRC grade 0	I only get breathless with strenuous exercise	☐
mMRC grade 1	I get short of breath when hurrying on the level or walking up in a slight hill	☐
mMRC grade 2	I walk slower than people of same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level	☐
mMRC grade 3	I stop for breath after walking 100 m or after few minutes on the level	☐
mMRC grade 4	I am too breathless to leave the house or I am breathless when dressing or undressing	☐

Continued

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Continued

CAT assessment		
For each item below, place a mark (×) in the box that best describes you currently. Be sure to only select one response for each question		Score
I never cough	⓪①②③④⑤	I cough all the time
I have no phlegm (mucus) in the chest at all	⓪①②③④⑤	My chest is completely full of phlegm (mucus)
My chest does not feel tight at all	⓪①②③④⑤	My chest feels very tight
When I walk up a hill or one flight of stairs, I am not breathless	⓪①②③④⑤	When I walk up a hill or one flight of stairs, I am very breathless
I am not limited doing any activity at home	⓪①②③④⑤	I am very limited doing activity at home
I am confident leaving my home despite my lung condition	⓪①②③④⑤	I am not at all confident leaving my home because of my lung condition
I sleep sound	⓪①②③④⑤	I do not sleep sound because of my lung condition
I have lots of energy	⓪①②③④⑤	I have no energy at all

(CAT: chronic obstructive pulmonary disease assessment test)

or walking up in a slight hill" (mMRC grade 1); "I walk slower than people of my age on the level because of breathlessness, or I have to stop for breath when walking on my own space on the level" (mMRC grade 2); "I stop for breath after walking 100 meters or after few minutes on the level" (mMRC grade 3); and "I am too breathless to leave the house or I am breathless when dressing or undressing" (mMRC Grade 4). COPD assessment score (CAT score) is an important tool for assessing dyspnea and other symptoms.

MANAGEMENT OF DYSPNEA

Management of dyspnea depends on identification of cause and treatment of specific disease. Supplemental O_2 should be administered for resting or exercise-induced desaturation [oxygen saturation (SpO_2) <90%]. Pleural fluid aspiration may temporarily relieve dyspnea. When the disease is not curable or treatable, then symptomatic management may be an essential step. Pulmonary rehabilitation program may be helpful in patients with chronic dyspnea. Dyspnea due to malignancies may respond to opioids, particularly morphine. Assurance and sympathetic interventions are helpful in terminally ill patients.

CONCLUSION

Dyspnea may arise from a wide variety of respiratory, cardiac, hematological, metabolic, and psychological diseases. Identification of the causes of dyspnea is an essential step. Detailed history, meticulous clinical examination, and routine investigations will identify the etiology in most of the cases. Sometimes, special investigations are required. Symptomatic management should go along with specific management of etiological disease. In terminally ill patients, assurance and opioids may alleviate symptoms.

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CLINICAL PEARLS

- Dyspnea can occur due to respiratory cardiac renal hematological metabolic and psychological causes.
- Each etiology may be recognized by following a systematic clinical approach of exclusion.
- The therapy is individualized according to etiology.

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General Practice

An Approach to Differential Diagnosis

Salient Features

A clinical approach to diverse symptomatology
Analysis of differential diagnosis
Focused and individualized management strategies

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