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Review in Clinical Anesthesiology





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Forewords
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CHAPTER 2

COPD AND ANESTHESIA

Chapter Outline

- Definition of COPD
- Risk factors
- Clinical features
- Pathophysiology
- Preoperative evaluation
- Assessment of pulmonary function
- Staging/Classification of severity of COPD
- Management of COPD
- Preoperative optimization strategies
- Management of anesthesia
- Postoperative care
- Role of chest physiotherapy
- Postoperative pulmonary complications
- Controversies
- Smoking and anesthesia

Obstructive airways disease in all its forms represents the commonest pulmonary disorder that the anesthetist encounters. Pulmonary complications after surgery are the leading cause of postoperative morbidity and mortality. Screening for pulmonary risk factors proceeds on the basis of clinical history and physical examination, the results of which can then be combined with the knowledge of the proposed surgical procedure to dictate an appropriate preoperative management.

WORKING DEFINITION

Chronic Obstructive Pulmonary Disease (COPD) has been defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD -2013) as a common preventable and treatable disease, characterized by presistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients.

According to American Thoracic Society, COPD is a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema; the airflow obstruction is generally progressive, may be accompanied by airway hyperreactivity, and may be partially reversible.

COPD includes:

i. Emphysema, (defined pathologically as dilatation of air spaces beyond the terminal bronchioles with destruction of their walls, but without obvious fibrosis);

ii. Chronic bronchitis, (defined clinically as chronic cough with sputum production for more than 3 months each year over the previous 2 years); and

iii. Small airways disease, a condition in which small bronchioles are narrowed.

COPD exacerbation is defined as an event in the natural history of the disease characterized by a change in the patient's baseline dyspnea, cough and/or sputum that is beyond normal day-to-day variations, is acute in onset, and may warrant a change in regular medication in a patient with underlying COPD. During exacerbations there is increased hyperinflation and gas trapping, with reduced expiratory flow, thus accounting for the increased dyspnoea. There is also worsening of V/Q abnormalities (ventilation-perfusing mismatch), which can result in hypoxemia.

Risk Factors

1. *Cigarette smoking:* Although the causal relationship between cigarette smoking and the development of COPD has been absolutely proved, there is considerable variability in the response to smoking. Although pack-years of cigarette smoking is the most highly significant

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predictor of FEV_1, only 15% of the variability in FEV_1 is explained by pack-years. This finding suggests that additional environmental and/or genetic factors contribute to the impact of smoking on the development of airflow obstruction.

2. *Occupational exposures:* Several specific occupational exposures, including coal mining, gold mining, and cotton textile dust, have been suggested as risk factors for chronic airflow obstruction.
3. *Ambient air pollution*
4. *Passive, or second hand, smoking exposure*
5. *Respiratory infections:* Although respiratory infections are important causes of exacerbations of COPD, the association of both adult and childhood respiratory infections to the development and progression of COPD remains to be proven.
6. *Genetic considerations:* α_1-antitrypsin (AT) deficiency—Although only 1—2% of COPD patients are found to have severe α_1-AT deficiency as a contributing cause of COPD, these patients demonstrate that genetic factors can have a profound influence on the susceptibility for developing COPD.

Clinical Features

The key features of COPD are given in Table 2.1.

Patients with COPD characteristically present in the fifth or sixth decade of life complaining of excessive cough, sputum production, and shortness of breath. Symptoms have often been present for 10 years or more. Dyspnea is noted initially only on heavy exertion, but as the condition progresses it occurs with mild activity. In severe disease, dyspnea occurs at rest. Pneumonia, pulmonary hypertension, cor pulmonale, and chronic respiratory failure characterize the late stage of COPD. As the disease progresses, two symptom patterns tend to emerge, historically referred to as "pink puffers" and "blue bloaters" (Table 2.2). These patterns were once thought to represent pure forms of emphysema and bronchitis, respectively, but this is a simplification of the anatomy and pathophysiology. Most COPD patients have pathologic evidence of both disorders, and their clinical course may reflect other factors such as central control of ventilation and concomitant sleep-disordered breathing.

Table 2.1: Features of COPD

- Progressive dyspnea
- Cough with/without sputum production, purulent in exacerbation
- Evidence of expiratory airflow limitation
- Poor reversibility with bronchodilators
- History of smoking
- Hyperinflation of the chest

Pink puffers, more obviously breathless, combine mild arterial hypoxia with low or normal $PaCO_2$. They have a normal resting pulmonary artery pressure, and are not centrally cyanosed.

Blue bloaters, not characteristically breathless at rest, have marked arterial hypoxia with hypercapnia. Cor pulmonale develops, defined clinically as right heart failure due to chronic lung disease in the absence of pre-existing left ventricular failure. The end-stage condition includes central cyanosis, right heart failure and secondary polycythemia. Pulmonary pressures are elevated at rest. They may show alarming fall in arterial oxygen saturation during sleep, and after surgery this feature is potentiated by the use of opioid analgesia.

One of the underlying differences between the two patterns is in the control of breathing. Pink puffers have a normal ventilatory drive and maintain a normal PCO2. While blue bloaters have a reduced ventilatory drive and retain carbon dioxide.

Pathophysiology of COPD

Expiration is normally a passive phenomenon, driven by the elastic recoil of the lungs. Airways closure, limiting expiration, is determined by the transmural pressure gradient between the pressure inside the conducting airways (generated by the elastic recoil) and the external pleural pressure.

The causes of expiratory airflow limitation in these patients are multifactorial and the following features occur in varying proportions (Fig. 2.1):

a. Chronic mucosal inflammation caused by repeated exposure to foreign material (e.g. smoking)
b. Active bronchospasm and excessive bronchial secretions with mucosal plugging
c. Destruction of normal alveolar architecture
d. Increased transmural pressure gradient across the wall of bronchiole favouring collapse during exhalation. (Increasing intrathoracic pressure due to forced exhalation exerting pressure on the bronchiolar walls via the neighboring lung parenchyma).
e. Loss of elasticity of lung parenchyma causing less opening traction on the airways
f. Loss of rigidity of bronchiolus wall due to chronic inflammation
g. Increase in gas velocity in the narrowed bronchiolus, which lowers the pressure inside the bronchiolus via Bernoulli's law.

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Table 2.2: Patterns of disease in COPD

	Pink puffer (Emphysema predominant)	*Blue bloater (Bronchitis predominant)*
Clinical	Breathless	Not breathless at rest
	No central cyanosis	Severe central cyanosis
	Chest hyperinflation	Cor pulmonale – ankle edema, hepatomegaly, raised JVP
	Patients usually thin	Patients frequently overweight
Blood gases	PaO_2 normal to slightly reduced (65–75 mm Hg) but SaO_2 normal at rest.	PaO_2 reduced (45–60 mm Hg)
	$PaCO_2$normal to slightly reduced (35–40 mm Hg)	$PaCO_2$ slightly to markedly elevated (50–60 mm Hg)
Chest X-ray	Hyperinflation – flat diaphragms, horizontal posterior ribs; Small heart shadow (“tubular heart”)	Increased interstitial markings ("dirty lungs"), especially at bases. Diaphragms are not flattened. Cardiomegaly may be present.
Red cell count	Normal	Secondary polycythemia
Hemodynamics	Normal resting PA pressure Normal cardiac output	Pulmonary hypertension Cardiac output slightly elevated at rest
Control of breathing	Normal ventilatory drive	Reduced ventilatory drive, retain carbon dioxide
Diffusing capacity	Normal	Reduced
Prognosis	Poor	Good

Abbreviations: JVP, Jugular venous pulse; SaO_2, Arterial oxygen saturation; PA, Pulmonary artery

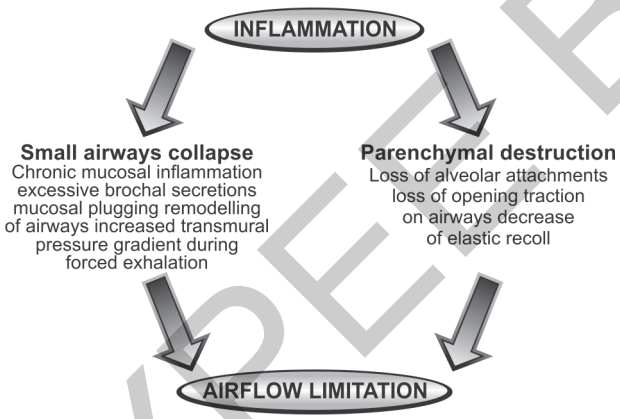

Fig. 2.1: Mechanisms underlying airflow limitation

In advanced stage, the limitation of expiratory flow occurs even during resting expiration. The inspiratory muscles work at a mechanical disadvantage, in part due to changes in the shape of the diaphragm and chest wall. Other features of the abnormal pulmonary mechanics in COPD patients include a greatly increased work of breathing, which in severe disease may account for more than 20% of the patient’s total oxygen consumption.

The consequences of these changes is incomplete expiration and gas trapping within the lungs, with a positive alveolar pressure at the end of expiration: the auto-PEEP (positive end-expiratory pressure). In patients breathing spontaneously auto-PEEP must be overcome before inspiratory flow can begin, further adding to the work of breathing. In mechanically ventilated patients auto-PEEP may promote unsuspected increases in intrathoracic pressure, reducing venous return and cardiac output. Auto-PEEP and the work of breathing can be reduced, and the mechanical efficiency of the respiratory muscles improved, by therapies that reduce airflow limitation and reduce gas trapping.

PREOPERATIVE EVALUATION FOR PATIENT WITH PULMONARY DISEASE

History

The most important symptoms of respiratory disease are: cough, sputum, breathlessness, hemoptysis, wheeze and chest pain.

Cough

Chemical irritants, including smoke and increased secretions can stimulate cough. Cough may be dry (irritating) or productive (sputum/blood). After a rapid increase in intrathoracic pressure caused by contraction

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of respiratory muscles against a closed glottis, the glottis opens with an explosive release of air into the upper airway. The sound of the cough and the circumstances in which it occurs may be helpful in making the diagnosis Nocturnal cough may indicate asthma or pulmonary edema. Recurrent laryngeal nerve palsy can cause bovine cough; intrathoracic neoplasia can cause brassy cough.

Sputum

Expectorated respiratory secretions are known as sputum. Normal lung produces about 100 ml of clear sputum each day, which is transported to the oropharynx and swallowed. Bronchorrhea refers to production of excessive secretions. An estimate should be made of the daily volume of sputum produced. Smokers often falsely consider their morning cough and sputum production to be normal. Some of the differential diagnosis with the types of sputum are given in Table 2.3.

Hemoptysis

It is the expectoration (coughing up) of blood or of blood-stained sputum from the bronchi, larynx, trachea, or lungs. Important causes are neoplastic and infective pulmonary disorders, and cardiac disease. Acute respiratory infection may be responsible for some blood streaking of the sputum but catastrophic bleeding can occur with causes including bronchiectasis, aspergillloma and tuberculosis.

Massive hemoptysis is variably defined as the expectoration of >100–600 mL over a 24-h period, although the patient's estimation of the amount of blood is notoriously unreliable. Massive hemoptysis can be usefully defined as any amount that is hemodynamically significant or threatens ventilation, in which case the initial management goal is not diagnostic but therapeutic.

Breathlessness

Breathlessness is the subjective (undue) awareness of breathing.

Breathlessness inappropriate to the level of physical exertion, or even occurring at rest is termed dyspnea. Dyspnea has been more specifically defined by the American Thoracic Society as the "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."[2]

Objective measurement of dyspnea is therefore difficult. It may be quantified by asking how many stairs can be climbed without stopping. The classification of Roizen has 5 grades of dyspnea (Table 2.4). Undiagnosed dyspnea of grade II or worse may require further investigation.

The classification given by Medical Research Council is given in Table 2.5.

The grades of dyspnea as described by American Thoracic Society is given in Table 2.6.

Orthopnea, or dyspnea on lying flat is a feature of left heart failure, severe airflow limitation and bilateral diaphragmatic weakness.

Breathlessness in COPD is characteristically persistent and progressive. In patients with established COPD, the severity of dyspnea is superior to forced expiratory volume in 1 second (FEV_1) in predicting quality of life and 5-year mortality.

Table 2.3: Types of sputum and common cause

Type of sputum		*Common cause*
Mucoid	Clear, gray	Chronic bronchitis
	White, viscid	Asthma
Serous	Clear, watery, frothy, pink	Acute pulmonary edema, Alveolar cancer
Purulent	Yellow, Green	Bronchopulmonary infection (pneumonia, bronchiectasis, lung abscess, cystic fibrosis)
Rusty	Golden, yellow	Pneumococcal pneumonia
Sputum with black parts		Bronchopulmonary aspergillosis
Foul-smelling sputum		Lung abscess, bronchiectasis

Note: The term "Phlegm" is referred to the mucus produced by the respiratory system, excluding that from the nasal passages, and particularly that which is expelled by coughing (sputum). Phlegm is in essence a water-based gel consisting of glycoproteins, immunoglobulins, lipids and other substances.

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Table 2.4: Roizen's classification of dyspnea

Grade	*Description*
Grade 0	No dyspnoea while walking on the level at normal pace
Grade I	"I am able to walk as far as I like provided i take my time"
Grade II	Specific street block limitation-"I have to stop for a while after one or two blocks"
Grade III	Dyspnoea on mild exertion-"I have to stop and rest going from the kitchen to the bathroom"
Grade IV	Dyspnoea at rest

Table 2.5: Severity of breathlessness: Medical Research Council classification

Grade 1	Breathless when hurrying on the level or walking up a slight hill
Grade 2	Breathless when walking with people of own age or on level ground
Grade 3	Has to stop because of breathlessness when walking on level ground at own pace

Table 2.6: American Thoracic Society grades of dyspnea

Grade	*Degree*	*Description*
0	None	No shortness of breath while hurrying on the level/walking up slight hill
1	Mild	Shortness of breath while hurrying on the level/walking up slight hill
2	Moderate	Walks more slowly than people of the same age on the level because of breathlessness, or has to stop for breath walking at own pace on level
3	Severe	Stops for breath after walking about 100 yards/after a few minutes on the level
4	Very severe	Too breathless to leave the house, to dress and undress

Wheeze

Wheeze is a high-pitched whistling sound produced by air passing through narrowed small airways. Typically wheeze is limited to, and louder during, expiration. In COPD wheezing may be caused by the presence of mucus accumulation in the airways and may mimic asthma. Wheeze indicates airflow limitation.

Chest Pain /Chest Tightness

Chest pain can originate from the pleura, the chest wall, and mediastinal structures. The lungs are not a source of pain because of their exclusive autonomic innervation. A careful history of chest pain should include site, radiation, mode of onset, duration, severity, and aggravating/relieving factors including the effects of breathing and movement. In COPD, chest tightness often follows exertion; poorly localized; muscular in character; and may arise from isometric contraction of the intercostal muscles.

Other Details in Medical History

- Pattern of symptom development
- History of exacerbations or previous hospitalizations for respiratory disorder: Patient may be aware of periodic worsening of symptoms even if these episodes have not been identified as exacerbations of COPD.
- Current medications and their appropriateness. Usually patients will be in a position to say which of his medications work well in case of exacerbations. (Patient may be instructed to carry these medications to the operating room as well).

Additional Features in Severe Disease

- Weight loss and anorexia
- Cough syncope: due to rapid increases in intrathoracic pressure during attack of coughing
- Ankle swelling: may be the only symptomatic pointer to the development of cor pulmonale
- Psychiatric morbidity: symptoms of depression and/or anxiety.

Personal History

Smoking: Cigarette smoking is the single most important risk factor.

Occupational exposure to toxic substances/allergens.

Family History

α_1-antitrypsin (AT) deficiency; cystic fibrosis; pulmonary hypertension; pulmonary fibrosis.

PHYSICAL EXAMINATION

While taking the clinical history observe the patient's appearance.

- Is the patient breathless at rest or in conversation?
- Is there audible wheeze?
- What is the resting respiratory rate?

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- Does the patient look anemic, polycythemic or cyanosed?
- Clubbing/nicotine stain in fingers?

Inspection

- Central cyanosis, or bluish discoloration of the mucosal membranes, may be present but is difficult to detect in artificial light and in many racial groups.
- Common chest wall abnormalities, which reflect the hyperinflation of the lung in COPD, include relatively horizontal ribs, "barrel-shaped" chest, and protruding abdomen.
- Flattening of the hemidiaphragms may be associated with paradoxical in-drawing of the lower rib cage on inspiration, and widening of the xiphisternal angle.
- Resting respiratory rate is often increased to more than 20 breaths per minute and breathing can be relatively shallow.
- Patients commonly show pursed-lip breathing, which may serve to slow expiratory flow and permit more efficient lung emptying.
- COPD patients often have resting muscle activation while lying supine. The use of scalene and sternocleidomastoid muscles is a further indicator of respiratory distress.
- Ankle or lower leg edema can be a sign of right heart failure.

Measurements of the Chest

- Rate of respiration
- Rhythm of respiration
- Chest expansion
- Anteroposterior and lateral dimensions.

Palpation

- Tenderness and local rise of temperature
- Tracheal position
- Cardiac impulse detection may be difficult due to lung hyperinflation
- Symmetry of expansion of chest
- Tactile vocal fremitus.

Percussion

- Resonance/dullness.

Auscultation

- Breath sounds – Vesicular/bronchial; often reduced in COPD due to pulmonary hyperinflation.
- Vocal resonance
  - Bronchophony – Vocal resonance seems to be near the earpiece of the stethoscope
  - Aegophony – Heard over consolidation; above the level of pleural effusion
  - Whispered pectoriloquy – It refers to an increased loudness of whispering noted during auscultation with a stethoscope on the lung fields on a patient's back.
- Added sounds: Pleural rub/wheezes/crackles.

ASSESSMENT OF PULMONARY FUNCTION

Pulmonary Function Tests

Pulmonary function tests refer to a group of tests used to diagnose and quantify the presence of respiratory disease. The classification and detailed description of pulmonary function tests are given elsewhere in this chapter.

Unlike asthma, COPD does increase the risk of postoperative pulmonary complications—PPC (odds ratio = 1.79). The more severe the COPD, the greater the risk, but there is no prohibitive degree of severity that precludes surgery. Although COPD increases PPC, surprisingly the risk is less than that associated with other patient-related risk factors including congestive heart failure (CHF), advanced age, and poor general overall health status. Patients with COPD also have higher risk for nonpulmonary complications such as wound infections and atrial arrhythmias.

In patients with COPD, preoperative spirometry may be useful to assess the disease severity and adequacy of bronchodilator therapy, but only in patients in whom it is difficult to determine this from the history and physical examination. Even before high-risk surgery, there is no role for routine PFTs. The rationale for this recommendation is that spirometry usually adds no information beyond that obtained by a careful history and physical examination. Patients with severe COPD as determined by spirometric values are unlikely to escape clinical detection.

Bedside Tests of Pulmonary Function

1. *Snider's Match Blowing Test*
   This test is based on individual's ability to blow out a match (with mouth open, no lip pursing) from three, six or nine inches away. The subjects with normal ventilatory function would be able to blow out the match at nine inches distance. The patients who were unable to blow it out at six inches distance were found to have some ventilatory defect, usually obstructive, and most

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of the patients who could not blow it out at three inches had combined defects.

Modified Snider's test

3 inches [Maximum Breathing Capacity (MBC) > 40 l/min]

6 inches [MBC > 60 l/min]

9 inches [MBC > 150 l/min].

2. *Forced Expiratory Time (FET)*

The subject should be asked to take a deep breath in and then breath out as quickly as he can. He should be encouraged to keep on breathing out during the procedure. Three consistent readings should be obtained. An FET of more than 6 seconds suggests airway obstruction.

3. *Saberazes Single Breath Count*

Patient is asked to take a deep breath followed by counting 1,2,3...till the time he cannot hold breath.

Shows trend of deteriorating or improving pulmonary function in preoperative and postoperatuve patients.

4. *Saberazes Breath Holding Test*

The breath holding test is all the more valuable because it represents in its ultimate analysis the response of the body to the oxygen-carbon dioxide balance in the alveolar air, blood, and tissues under any and all conditions of health and disease. According to the apneic pause, split-seconds watch method of Sabrasez, the following intervals represent the same degrees of normality and depletion of alkaline reserve (Table 2.7): A thorough understanding and proper evaluation of the breath holding test, as a diagnostic and prognostic indication in abnormal conditions, depends on the realization that the breath holding capacity and apneic pause involve the oxygen absorption and carbon dioxide elimination phases of pulmonary respiration as well as the balance of blood and tissue reserve alkalinity.

5. *Cough Test*

Observe for ability to cough, strength and effectiveness. Wet productive cough candidate for pulmonary complications. Inadequate cough indicates reduced FVC (Forced Vital Capacity).

6. *De Bono's Whistle Test*

De Bono's whistle (Fig. 2.2) is used to measure the maximum respiratory flow rate which is affected by changes in the airway resistance. The whistle consists of a plastic tube with a slot in the side and a double-orifice type of whistle at the end. The slot in the side is a leak-hole, the size of which can be altered by sliding the plastic tube in and out of a disposable cardboard mouthpiece. For any given size of the leak-hole, a certain airflow rate is required for the whistle to sound.

The patient places his lips around the cardboard mouthpiece and blows out through it as forcibly as possible. It is best to start with a small leak-hole, which is gradually made larger (by sliding the cardboard mouthpiece) until a whistle can no longer be effected. At the last position at which the whistle can be obtained, the peak expiratory flow rate can be read off the scale (calibrated in hundreds of liters per minute) at the edge of the mouthpiece.

7. *Wright's Peak Flowmeter*

The peak flowmeter, developed by Martin Wright in 1959, has been used to monitor serial lung function in asthmatics because it is inexpensive, portable, easy to use, and it is an objective measure of lung function (Fig. 2.3). In addition, patients can be taught to use the meter and monitor their own lung function to determine when to use different medication as guided by an asthma action plan. The only weakness of the peak flowmeter as with most pulmonary function measures is that it does require patient cooperation and effort.

Normal value for males is 450 – 700 l/min, that for females is 300 – 500 l/min.

Table 2.7: Breath holding time and inferences

Breath holding (seconds)	*Condition*	*Vital capacity*
30 – 50	Exceptional	
25 – 30	Average normal	3.5 L
20 – 25	Acidemia	3.0 L
15 – 20	Mild acidosis	2.5 L
10 – 15	Acidosis	2.0 L
5 – 10	Severe acidosis	1.5 L

Chest X-ray Findings in COPD

Emphysema

Increased lucency of the lung fields. (Smokers – more prominent in the upper zone, α_1 antitrypsin deficiency – more in basal zones).

Bullae – It refers to localized rediolucency more than 1 cm in diameter and surrounded by hairline arcuate shadows. Look for features of pneumothorax – rupture of bullae.

Chest – Vertically elongated with low flattened diaphragm (hyperinflation).

Heart shadow – Vertical and narrow.

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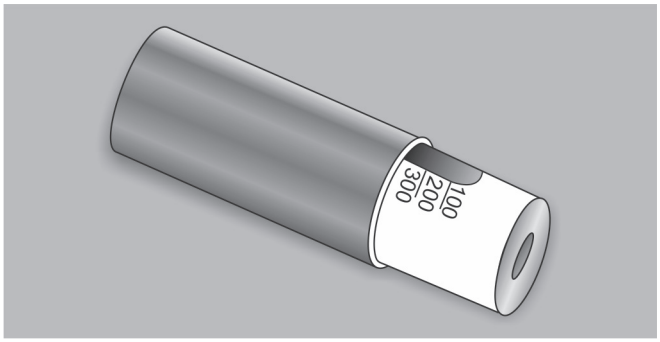

Fig. 2.2: De Bono's whistle

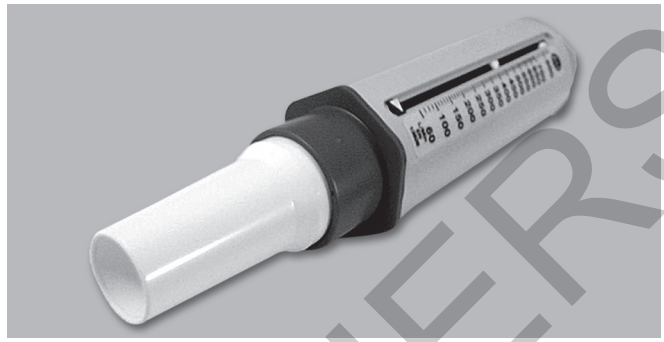

Fig. 2.3: Wright's peak flow meter

Features of pulmonary hypertension – pulmonary arteries become enlarged and taper rapidly ("pruning" – prominent hilar markings with loss of peripheral vascular shadows). Right heart border – more prominent and impinge on retrosternal air space).

Chronic Bronchitis:

Increased interstitial markings – "dirty lung fields".

Computed Tomography Scanning

- High-resolution CT (HRCT) scanning is more sensitive than standard chest radiography.
- HRCT scanning is highly specific for diagnosing emphysema, and the outlined bullae are not always visible on a radiograph. HRCT scanning may provide an adjunct to diagnosing various forms of COPD (i.e., lower lobe disease may suggest α_1 antitrypsin deficiency) and may help determine if surgical intervention would be of benefit to the patient.

Electrocardiogram in COPD

Look for evidence of right atrial and ventricular hypertrophy (cor pulmonale).

Strain pattern – ST depression and inverted T waves in leads V1 – V4.

P—pulmonale (tall P wave, >3mm). Right axis deviation. Prominent R waves and T inversion in right chest leads.

STAGING/CLASSIFICATION OF SEVERITY OF COPD

Until recently, the severity of airflow obstruction has been the mainstay of staging chronic obstructive pulmonary disease (COPD). The American Thoracic Society (ATS) has provided criteria for staging COPD based on the presence of obstruction (ratio of FEV_1 to forced vital capacity [FEV_1/FVC] <70%) and its severity as measured by percent of predicted FEV_1. The GOLD guidelines have taken staging one step further by incorporating the presence or absence of signs/symptoms in its most severe stage.

American Thoracic Society (ATS) Classification

ATS criteria for assessing the severity of airflow obstruction (based on the percent predicted postbronchodilator FEV_1 when the FEV_1/FVC is <70%). The staging is as given in Table 2.8.

Note: Stage 0: At risk COPD category which appeared in the GOLD 2001 report is no longer included as a stage of COPD, as there is incomplete evidence that the individuals who meet the definition of "at risk COPD" (chronic cough and sputum production, normal spirometry) necessarily progress on to Stage I. However, the importance of public health message that chronic cough and sputum are not normal is unchanged in the latest GOLD report.

GOLD – Global Initiative of Obstructive Lung Disease classification of COPD

Table 2.9 gives the classification suggested in GOLD guidelines. It is based on post-bronchodilator FEV_1.

Table 2.8: American Thoracic Society classification of severity of COPD

Stage	*Classification*	*Description*
I	Mild	FEV_1 greater than or equal to 80% of predicted
II	Moderate	FEV_1 less than 80% and greater than or equal to 50% of predicted
III	Severe	FEV_1 less than 50% and greater than or equal to 30% of predicted
IV	Very severe	FEV_1 less than 30% of predicted or FEV_1 less than 50% and chronic respiratory failure

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Table 2.9: COPD classification by GOLD

	In patients with $FEV_1/FVC < 0.70$
GOLD 1: Mild	$FEV_1 \geq 80\%$ of predicted
GOLD 2: Moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3: Severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4: Very severe	$FEV_1 < 30\%$ predicted

BODE Index

The BODE index is a composite marker of disease taking into consideration the systemic nature of COPD (Celli et al 2004). It predicts all cause mortality and respiratory related mortality with better accuracy than the FEV_1 alove.

The indices are given in Table 2.10.

The approximate 4-year survival, as per BODE index is estimated as:

- 0-2 points = 80%
- 3-4 points = 67%
- 5-6 points = 57%
- 7-10 points = 18%

MANAGEMENT OF COPD

An effective COPD management strategy includes four components:

1. Assess and monitor disease
2. Reduce risk factors
3. Manage stable COPD
4. Manage exacerbations.

1. Assess and Monitor Disease

   Consider COPD, and perform spirometry if any of the following indicators are present in any individual above the age of 40 (Table 2.12).

   These indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of a diagnosis of COPD. Spirometry is needed to establish a diagnosis of COPD. The presence of a post-bronchodilator $FEV_1/FVC < 70\%$ predicted and $FEV_1 < 80\%$ predicted confirms the presence of airflow limitation that is not fully reversible.

   Stage I: Mild COPD or stage II: Moderate COPD may not come to medical facility until their airflow limitation becomes more severe or their lung functions are acutely worsened by a respiratory infection. As airflow limitation worsens, the patient enters stage III: Severe COPD, the symptoms of cough and sputum production typically continue, dyspnea worsens, and additional symptoms heralding complications (such as respiratory failure, right heart failure, weight loss and arterial hypoxemia) may develop.

2. Reduce Risk Factors

   Smoking cessation is the single most effective – and cost effective – intervention in most people to reduce the risk of developing COPD or stop its progression. Many occupationally induced respiratory disorders can be reduced or controlled through a variety of strategies aimed at reducing the burden of inhaled particles and gases.

3. Manage Stable COPD

   None of the existing medications for COPD have been shown to modify the long-term decline in the lung function that is the hallmark of this disease. Therefore, pharmacotherapy for COPD aims at reducing the symptoms and/or complications. Bronchodilator medications are central to the symptomatic management of COPD. They are given on *as needed basis* or *on regular basis* to prevent or reduce symptoms and exacerbations.

   The principal bronchodilator treatments are β_2 agonists, anticholinergics and methylxanthines used singly or in combination. Regular treatment with long-acting bronchodilators is more effective and convenient than short-acting bronchodilators.

Table 2.10: BODE index

	Score			
	0	*1*	*2*	*3*
Body Mass Index (kg/m^2)	< 21	≥ 21	-	-
Obstruction -FEV1 (postbronchodilator percent predicted)	> 65%	50 – 64%	35 – 49%	< 35%
Dyspnoea scale (MRC) – see table 2-11	0 -1	2	3	4
Exercise capacity (6 minute walk distance in meters)	> 350	250 – 350	150 - 250	< 150

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Table 2.11: MRC dyspnea scale

Score	*Description*
0	Dyspneic on strenuous exercise
1	Dyspneic on walking a slight hill
2	Dyspneic on walking level ground; must stop occasionally due to breathlessness
3	Dyspneic after walking 100 yards or a few minutes
4	Cannot leave house; dyspneic doing activities of daily living

Table 2.12: The key indicators for considering a diagnosis of COPD

Dyspnea that is	Progressive (worsens over time) Usually worse with exercise Persistent (present every day) Described by the patient as " increased effort to breathe", "heaviness", "air-hunger" or "gasping"
Chronic cough	May be intermittent and may be nonproductive
Chronic sputum production	Any pattern
History of exposure to risk factors, especially:	Tobacco smoke Occupational dusts and chemicals Smoke from home cooking and heating fuels
Family history of COPD	

The addition of regular treatment with inhaled glucocorticosteroids to bronchodilator treatment is appropriate for symptomatic COPD patients with FEV_1<50% predicted (stage III: Severe COPD and stage IV: very severe COPD) and repeated exacerbations. Chronic treatment with systemic glucocorticosteroids should be avoided because of an unfavorable benefit-to risk ratio.

All COPD patients benefit from exercise training programs, improving with respect to both exercise tolerance and symptom of dyspnea and fatigue.

A concise description of the drugs used for the management of stable COPD is given in Table 2.13.

A proposed model for initial pharmacological management of COPD according to the individualized assessment of symptoms and exacerbations risk is shown in Figure 2.4.

Note: Surgical Treatments Include: Bullectomy, lung volume reduction surgery (LVRS), lung transplantation

4. Management of Exacerbations

The most common causes of exacerbation are infection of the tracheobronchial tree and air pollution, but the cause of about one-third of exacerbations cannot be identified. Table 2.14 enumerates common precipitating factors for acute respiratory crisis in COPD. The common signs of severity are listed in Table 2.15

Patients experiencing COPD exacerbations with clinical signs of infection (e.g. sputum purulence) may benefit from antibiotics. Inhaled bronchodilators (particularly inhaled β_2-agonists with or without anticholinergics) and oral glucocorticosteroids are effective treatments.

The key points in the emergency room management of COPD exacerbation are given in Table 2.16.

The indications for intensive care unit (ICU admission are enumerated in Table 2.17.

Noninvasive Ventilation (NIV)

Noninvasive mechanical ventilation in exacerbations improves respiratory acidosis, increases pH, decreases the need for endotracheal intubation, and reduces $PaCO_2$, respiratory rate, severity of breathlessness, the length of hospital stay and mortality.

The common indications for noninvasive ventilation are:

- Moderate to severe dyspnea with use of accessory muscles of respiration and paradoxical abdominal breathing, or retraction of the intercostal spaces.
- Moderate to severe acidosis (pH ≤ 7.35) and/or hypercapnia (PCO_2 ≥ 45 mm Hg)

The presence of any of the following is considered as a contraindication to noninvasive ventilation:

- Respiratory arrest
- Cardiovascular instability (hypotension, arrhythmias, myocardial infarction)
- Change in mental status; uncooperative patient
- High aspiration risk
- Viscous or copious secretions
- Recent facial or gastroesophageal surgery
- Craniofacial trauma
- Fixed nasopharyngeal abnormalities
- Burns
- Extreme obesity.

Invasive Mechanical Ventilation

The indications for invasive mechanical ventilation are:

- Unable to tolerate NIV or NIV failure
- Respiratory or cardiac arrest

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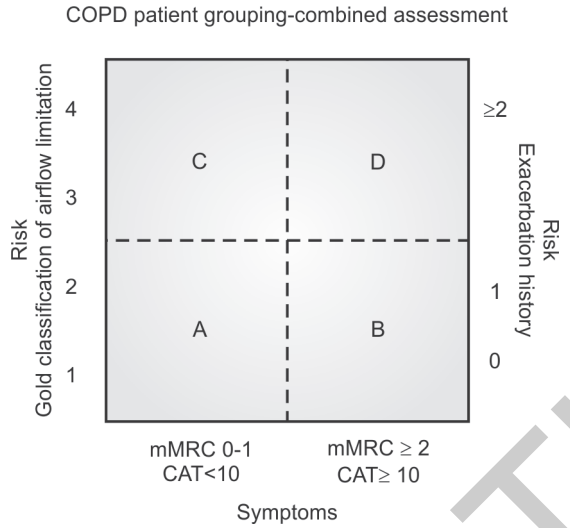

Initial Pharmacological Management of COPD

Patient group	Recommended first choice	Alternative choice	Other possible treatments
A	Short-acting anticholinergic (when required) or Short-acting β_2 agonist (when required)	Long-acting anticholinergic or Long-acting β_2 agonist or Short-acting β_2 agonist and Short-acting anticholinergic	Theophylline
B	Long-acting anticholinergic or Long-acting β_2 agonist	Long-acting anticholinergic and long-acting β_2 agonist	Short-acting β_2 agonist and/or Short-acting anticholinergic Theophylline
C	Inhaled corticosteroid + long-acting β_2 agonist or Long-acting anticholinergic	Long-acting anticholinergic and long-acting β_2 agonist or Long-acting anticholinergic and Phosphodiesterase 4 inhibitor or Long-acting β_2 agonist and Phosphodiesterase 4 inhibitor	Short-acting β_2 agonist and/or Short-acting anticholinergic Theophylline
D	Inhaled corticosteroid + long-acting β_2 agonist and/or Long-acting anticholinergic	Inhaled corticosteroid + Long-acting β_2 agonist and long-acting anticholinergic or Inhaled corticosteroid + long-acting β_2 agonist and Phosphodiesterase 4 inhibitor or Long-acting anticholinergic and long-acting β_2 agonist or Long-acting anticholinergic and Phosphodiesterase 4 inhibitor	Carbocysteine Short-acting β_2 agonist and/or Short-acting anticholinergic Theophylline

Fig. 2.4: Combined assessment and initial pharmacological management of COPD

Note: Group A - few symptoms and a low risk of exacerbations; Group B - more significant symptoms but still a low risk of exacerbations; Group C - few symptoms but a high risk of exacerbations; Group D - many symptoms and a high risk of exacerbations.

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Table 2.13: Concise description of drugs used for stable COPD

β_2 agonists	
Mechanism	Relax airway smooth muscle by stimulating β_2-adrenergic receptors, which increases cyclic AMP and produces functional antagonism to bronchoconstriction.
Salient features	Relatively rapid onset of bronchodilator effect (probably slower in COPD than asthma).Oral therapy is slower in onset and has more side effects than inhaled treatment. Duration of action: short-acting – 4 to 6 hours; long-acting – 12 hours or more with no loss of effectiveness overnight or with regular use.
Adverse effects	Resting sinus tachycardia, cardiac arrhythmias in susceptible patients (rare with inhaled therapy). Exaggerated somatic tremor. Hypokalemia (especially when combined with thiazide diuretics) Increased oxygen consumption (mild fall in PaO_2 - ?clinical significance)
Examples	Short-Acting: Salbutamol (Albuterol) – 100, 200μg (MDI, DPI); 5mg PO Terbutaline – 400, 500μg (DPI; 2.5, 5mg PO; 0.2, 0.25mg SC inj Long-acting: Salmeterol – 25, 50 μg (MDI, DPI) Famoterol – 4.5,12 μg (MDI, DPI)
Anticholinergics	
Mechanism	Blockage of acetylcholine's effect on M3 receptors. Current short-acting drugs also block M2 receptors and modify transmission at the preganglionic junction (less important in COPD).
Salient features	Duration of action of short-acting agents – upto 8 hours; while long-acting ones – more than 24 hours. Meaningful increases in lung function can be achieved following administration of inhaled anticholinergic plus sympathomimetic bronchodilators even in patients with moderate to severe COPD. Mucociliary clearance is unaffected by these drugs, and respiratory infection rates are not increased.
Adverse effects	Dryness of mouth. Occasional prostatic symptoms. Bitter, metallic taste (ipratropium) Nebulization via facemask can precipitate acute glaucoma in susceptible patients.
Examples	Short-acting: Ipratropium bromide – 20, 40 μg (MDI) Oxitropium bromide – 100 μg (MDI) Long-acting: Tiotropium – 18 μg (DPI)
Methylxanthines	
Mechanism	Non-selective phosphodiesterase inhibitors. Other proposed mechanisms are: blockade of adenosisne receptors and release of endogenous catecholamines.
Salient features	Changes in respiratory muscle function have been reported – improves the contractility of the diaphragm. Narrow therapeutic range: 5-20 μg/ml. Low dose theophylline reduces exacerbations in patients with COPD but does not increase post-bronchodilator lung function.
Advantages	Improve diaphragmatic contractility Direct smooth muscle relaxation Synergistic with β_2 agonists Reduce steroid requirement
Adverse effects	Toxicity is dose related Atrial and ventricular arrhythmias Grandmal convulsions Headache, insomnia, nausea, heartburn
Examples	Aminophylline: 200-600mg PO (for acute exacerbation – loading dose 5mg/kg over 30 minutes, followed by 0.5mg/kg/hour continuous infusion) Theophylline (SR): 100-600 mg PO

Contd...

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