

Written, compiled and edited by: S.K. Jindal, D. Gupta and A.N. Aggarwal

[PREFACE]
There are over 40 different guidelines from different countries on diagnosis and management of chronic obstructive pulmonary disease (COPD). The guidelines formulated by the Global Initiative for chronic obstructive lung disease (GOLD) are perhaps the most popular and global in nature. The need to formulate a different set of guidelines for India was felt because of the differences in risk factors, disease prevalence and pattern, and above all, the different overall health-care infrastructure. Moreover a large burden of tuberculosis, which is an important cause of cough, adds to the difficulties of diagnosis and management.

These guidelines have been developed at the initiative of WHO (India) under the WHO-Government of India Biennium (2002-2003) programme. A consensus workshop was held in December 2002 with representative participation from several national professional bodies, medical colleges, general health sector, and other institutes. The recommendations were subsequently compiled and reviewed by the participants and other experts.

The guidelines essentially incorporate general GOLD recommendations. The major alterations include a greater stress on clinical criteria, exclusion of diagnosis of tuberculosis, and a three-tier approach at different levels of health care, especially the primary and secondary care levels. It is hoped that the recommendations will help the physicians of all hues to effectively manage COPD.


INTRODUCTION
Chronic obstructive pulmonary disease (COPD) is a common clinical problem. It is also known by various other names, such as chronic obstructive lung disease (COLD), chronic obstructive airway disease (COAD), chronic airflow obstruction (CAO), chronic airway (or airflow) limitation (CAL), or simply as chronic bronchitis and emphysema.

Chronic obstructive pulmonary disease, which includes chronic bronchitis and emphysema, is a progressive disease characterized by airflow limitation/obstruction, that is either not reversible at all or only partially reversible. It is generally difficult to separate out the two conditions (chronic bronchitis and emphysema), hence these are grouped together as COPD. Chronic obstructive pulmonary disease does not include asthma in which the airflow obstruction is largely reversible. The airflow obstruction in COPD is associated with abnormal inflammatory response of the lungs to chronic inhalational exposure from smokes, dusts and other air pollutants.

Representative Participation: (i) Indian Council of Medical Research, (ii) Indian Chest Society, (iii) National College of Chest Physicians (India), and (iv) American College of Chest Physicians (India).
Chronic obstructive pulmonary disease manifests as chronic cough with or without sputum production. To define COPD, the presence of these symptoms for more than three months of a year for at least two consecutive years is considered essential. It may or may not be accompanied with progressive breathlessness. The disease progresses with time ultimately leading to respiratory disability and death.

Acute exacerbations of COPD occur whenever there is an episode of infection or some other complication. There is worsening of symptoms, deterioration of clinical condition and impairment of lung function during the period of exacerbation.

**EPIDEMIOLOGY AND RISK FACTORS**

Chronic obstructive pulmonary disease is primarily a disease of the adult. The prevalence of COPD reported in different population-based studies from India is highly variable (Table 1)\(^5\)-\(^{16}\). The prevalence rates in male subjects of 2.12% to 9.4% in studies reported from North are generally higher than 1.4% to 4.08% reported from South India. The respective ranges for female subjects vary from 1.33% to 4.9% from North and from 2.55% to 2.7% from South India. For epidemiological assessment, the rounded-off median prevalence rates were assessed as 5% for male and 2.7% for female subjects of over 30 years of age.

The disease is distinctly more common in males. The male to female ratio had varied from 1.32:1 to 2.6:1 in different studies with a median ratio of 1.6:1.

Chronic obstructive pulmonary disease results from chronic inhalational exposures to various smokes, noxious particles and gases.

**Tobacco Smoke**

Tobacco smoke, which is a mixture of over 4000 chemical constituents, is the most important cause. Amongst males, tobacco smoking is responsible for more than 80% of patients\(^5\),\(^{17}\). Both cigarette and bidi smoking are equally responsible\(^18\). Pipe and hookah smoking are also important in causing COPD. There is no reliable information on smoking associated COPD in women in whom the overall prevalence of smoking is very low. Besides active tobacco smoking, exposure to smoking from others, i.e. passive smoking, better termed as environmental tobacco smoke (ETS) exposure, may also play a contributory role especially in non-smoker individuals including women\(^19\),\(^20\).

**Solid Fuel Combustion**

The smoke from combustion of solid fuels
such as dried dung, wood and crop residue used for cooking and heating, especially in villages, semi-urban and slum areas, is an important cause of pollution of the indoor air. It is responsible for a large number of COPD in the rural inhabitants in general and women in particular.

Outdoor Air Pollution

Exhausts from vehicles and industrial units; dusts, fumes and smoke from burning of crop residues in the fields constitute important sources of air pollution. Chronic exposure to polluted air is an important cause of chronic respiratory diseases such as the COPD.

PATHOGENESIS AND PATHOPHYSIOLOGY

Although cigarette smoking is the most important cause of COPD, only 10-15% of long-term smokers develop clinically significant COPD, and approximately half will never develop any symptomatic physiological deficit. Why the normal, protective inflammatory response becomes an exaggerated, harmful one in only some smokers is poorly understood, and the precise mechanisms underlying the development of this disorder remain largely unknown. Presumably the inflammation caused by cigarette smoking interacts with other host or environmental factors to produce excess decline in lung function that results in COPD.

It is believed that inhaled noxious particles and gases result in lung inflammation, induce tissue destruction, and impair defense mechanisms that serve to limit or repair this damage. This damage leads to the mucus hypersecretion, airway narrowing and fibrosis, destruction of lung parenchyma and vascular changes. In turn, these pathological changes lead to airflow limitation and other physiological abnormalities characteristic of COPD. It is characterized by an increase in neutrophils, macrophages and T-lymphocytes in various parts of the lung. These activated inflammatory cells release a variety of chemical mediators, many of which (e.g., leukotriene B4, interleukin-8, and tumour necrosis factor) are capable of damaging lung structures and/or sustaining neutrophilic inflammation. In addition to inflammation, two other processes thought to be important in the pathogenesis of COPD are an imbalance of proteinases and anti-proteinases in the lung, and oxidative stress. Although both these processes may themselves result from ongoing inflammation, they can also arise from genetic (e.g., alpha-1 antitrypsin deficiency) or environmental (e.g., oxidant compounds in cigarette smoke) factors.

The peripheral airways are the major sites of airways obstruction in patients of COPD. The structural changes in the airway wall, as well as airway edema and mucus hypersecretion contribute to airway narrowing. The irreversible component of airflow limitation is primarily due to remodelling of the smaller airways; lung parenchymal destruction may also play a role. In advanced COPD, peripheral airways obstruction, parenchymal destruction, and pulmonary vascular abnormalities reduce the lung’s capacity for gas exchange, producing hypoxemia and, later on, hypercapnia. Ventilation-perfusion mismatch is the dominant mechanism of hypoxemia in COPD.

HOW TO DIAGNOSE COPD?

Suspecting COPD

Chronic obstructive pulmonary disease can be suspected in most patients on the basis of symptoms and signs. Alternate diagnoses such as bronchial asthma, pulmonary tuberculosis, bronchiectasis, malignancies and other chronic lung diseases may require exclusion. Investigations would be required to confirm the diagnosis.

Clinical History

Diagnosis is considered in any individual who presents with characteristic symptoms and presence of one or more risk factors. The important clinical indicators are as follows:
1. **Chronic cough.** Present on most days for at least three months in a year for two or more consecutive years. Cough may be either present throughout the day or only intermittently. Cough is sometimes nocturnal in nature.

2. **Chronic sputum production.** Cough may or may not be associated with production of mucoid or mucopurulent sputum. Both cough and sputum production are characteristically more in the early morning, on waking up.

3. **Breathlessness (dyspnoea).** Dyspnoea may not be present initially, but develops later in the course. It is progressive over the time. Dyspnoea is worse on exercise and during acute exacerbations.

4. **Acute exacerbations.** There are repeated episodes of acute bronchitis causing worsening of symptoms. Most patients would seek medical help only during these episodes of worsening.

5. **Risk factors.** History of tobacco smoking is present in most male patients. Non-smoker patients (especially women) are significantly exposed to other risk factors, such as the combustion of solid fuels or occupational exposures to dusts and fumes.

**Physical Examination**

Though an important component of clinical assessment, physical examination is rarely diagnostic in COPD. Physical signs of airflow limitation are rarely present until significant impairment of lung function has occurred. However, certain findings on clinical examination point towards the diagnosis of COPD.

The chest examination may reveal signs of emphysema such as the barrel shape (increased anteroposterior diameter, more horizontally set ribs, prominent sternal angle and wide subcostal angle). Due to the elevation of sternum, the distance between the suprasternal notch and the cricoid cartilage is reduced from the normal 3-4 finger breadths. The patient may use accessory muscles of respiration.

Chest percussion will reveal findings of hyperinflation with obliteration of cardiac dullness and downward displaced upper border of liver dullness. Elsewhere, the note will be hyperresonant. Breath sounds will have a prolonged expiratory phase with a uniformly diminished intensity. Fine inspiratory crepitations and rhonchi are commonly heard. Forced expiratory time (FET) will be prolonged to more than six seconds and patient may have pursed lip breathing.

The physical findings may change in the presence of complications.

**Alternate Diagnosis**

Asthma is generally excluded on the basis of history. It is usually present from childhood and is characterized by episodes of breathlessness and wheezing with asymptomatic periods in between. Ronchi are more prominent and extensive on physical examination. More importantly, there is greater variability and irreversibility of symptoms, physical signs and tests of airway obstruction in asthma than COPD.

Diseases such as tuberculosis and bronchiectasis are common causes of chronic cough in this country. They are usually not confused with COPD. Physical findings of fibrocavitary disease support a diagnosis of tuberculosis. Sputum is purulent and greater in amount in patients with bronchiectasis. Coarse crepitations and finger clubbing are generally present.

Any chronic lung disease can occasionally pose a problem in differential diagnosis. Whenever, there is confusion, investigations will help.

**Presence of Complications**

1. **Chronic cor-pulmonale.** Almost all cases of COPD will progress to chronic cor-pulmonale in due course of time. It is detected from the presence of signs suggestive of pulmonary hypertension and right ventricular enlargement and/or failure, such as a loud second heart sound,
parasternal heave and raised jugular venous pressure (JVP).

2. **Respiratory failure.** Chronic respiratory failure results from disease progression. It is suspected from the presence of tachypnoea, cyanosis, flapping tremors, and altered sensorium.

3. **Chest infections, such as pneumonias.**

4. **Pneumothorax.**

**INVESTIGATIONS**

Investigations are required for exclusion of an alternate diagnosis, confirmation of diagnosis of COPD, assessment of severity of disease and diagnosis of complications.

**Excluding Alternate Diagnosis**

It is especially important to exclude tuberculosis in all patients having chronic cough. Examine sputum smears for acid-fast bacilli (AFB), at least thrice.

Chest radiograph will help to identify alternate diseases, such as fibrocavitary tuberculosis, bronchiectasis, lung tumours; and detect complications such as chronic cor-pulmonale, pneumothorax or bronchopneumonia.

Additional tests such as the spirometry may be carried out where physician feels the diagnosis of asthma is under consideration. Bronchodilator reversibility testing is useful to help rule out a diagnosis of asthma and to establish patient’s best attainable lung function. Peak expiratory flow rate (PEFR) with reversibility may be substituted for FEV₁ when spirometry is not available.

In situations when patient is not responding to adequate and properly prescribed therapy or if there is a doubt of an alternate diagnosis such as asthma, glucocorticoid reversibility test with two weeks of oral corticosteroids should be attempted (by a specialist at the secondary care centre with facilities for spirometry)³⁷. Criteria for reversibility are an increase in FEV₁ of 200 ml and 15% above baseline.

**Confirming the Diagnosis**

Spirometry remains the gold standard for confirmation and staging of COPD. Patients should be referred for spirometry if diagnosis is doubtful. Spirometry is used to measure the forced vital capacity (FVC), *i.e.* maximal volume of air forcibly exhaled from the point of maximal inhalation; the volume of air exhaled during the first second of this maneuver (FEV₁) and the ratio of these two measurements (FEV₁/FVC). The presence of a post-bronchodilator FEV₁ < 80% of the predicted value in combination with a FEV₁/FVC < 70% confirms the presence of airflow limitation that is not fully reversible. Predicted values of different spirometric parameters are available as normograms and tables drawn from different prediction equations.

**Staging Severity of COPD**

Assessment of severity is based on the degree of the spirometric abnormality. Based on the results of spirometry, COPD can be categorized into four stages: at risk, mild, moderate and severe (Table 2). If spirometry is not available, both staging of the disease and follow-up of patients should be done on the basis of severity of symptoms/level of disability/6-minute walk test and/or peak expiratory flow (PEF (Table 3)³⁸-⁴⁰.

**Table 2. Staging of COPD based on spirometry**

<table>
<thead>
<tr>
<th>Severity</th>
<th>Spirometry Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>At risk</td>
<td>Normal spirometry, chronic symptoms</td>
</tr>
<tr>
<td>Mild</td>
<td>FEV₁/FVC &lt; 70%</td>
</tr>
<tr>
<td></td>
<td>FEV₁ &gt;80% predicted</td>
</tr>
<tr>
<td>Moderate</td>
<td>FEV₁/FVC &lt; 70%</td>
</tr>
<tr>
<td></td>
<td>FEV₁ 30-80% predicted</td>
</tr>
<tr>
<td>Severe</td>
<td>FEV₁/FVC &lt; 70%</td>
</tr>
<tr>
<td></td>
<td>FEV₁ &lt;30% predicted</td>
</tr>
</tbody>
</table>

Although spirometry is the gold standard for staging, PEF can serve as a good substitute if spirometry is not available. Six-minute walking test is performed by measuring distance covered in six minutes when patient walks at his/her own speed (under physician super-
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This is a simple test, which can be performed at the primary care level. Measurement of arterial blood gases/pulse oximetry in patients with severe COPD is desirable although severity of respiratory failure may be assessed by symptoms of hypercapnia (bounding pulse, warm extremities, flaps and tremulousness) and hypoxia (tremors, restlessness, mental obtundation and cyanosis).

**TREATMENT OF PATIENTS WITH STABLE COPD**

The important components of managing patients with stable COPD include; (a) minimisation of risk factors, (b) pharmacotherapy appropriate to the disease severity, and (c) supportive nonpharmacological measures (such as patient education and rehabilitation).

Assess the risk factors and other complications and manage accordingly. Advise and help to quit smoking. Similar attempts should be made to minimise other risk factors (Table 4). Assess the disease severity on an individual basis by taking into account the patient’s symptoms, airflow limitation, frequency and severity of exacerbations, complications, respiratory failure, co-morbidities, and general health status. Start treatment depending upon the severity of the disease. None of the existing medication for COPD has been shown to modify the long-term decline in lung function. Therefore, pharmacotherapy for COPD is used only to decrease symptoms and complications. Patient education is necessary to improve skills, ability to cope with illness and the health status. Health education is particularly effective for sustained smoking cessation. In addition, appropriate information about the nature of the disease, instructions on how to use different medications and inhalers, and clues to recognize symptoms of exacerbation are mandatory.

**Smoking Cessation**

Smoking cessation is the most important and effective step. Follow the standard guidelines for helping patients with COPD to quit smoking (Tables 5 & 6).

**Table 3. Staging of COPD based on symptoms, signs, 6-minute walk test and peak expiratory flow rate**

<table>
<thead>
<tr>
<th>Symptoms (Cough and Sputum)</th>
<th>Signs</th>
<th>6-Minute Walk Test</th>
<th>PEFR (optional)</th>
</tr>
</thead>
<tbody>
<tr>
<td>At risk</td>
<td>No dyspnoea, hypersecretion+</td>
<td>Mild hyperinflation</td>
<td>&gt;200m</td>
</tr>
<tr>
<td>Mild</td>
<td>Dyspnoea on unaccustomed activity or climbing two flight of stairs</td>
<td>Mild hyperinflation</td>
<td>100-200m</td>
</tr>
<tr>
<td>Moderate</td>
<td>Dyspnoea on accustomed activity</td>
<td>Moderate hyperinflation</td>
<td>100-200m</td>
</tr>
<tr>
<td>Severe</td>
<td>Dyspnoea at rest</td>
<td>Near absence of breath sounds, respiratory failure, polycythemia, CCF</td>
<td>&lt; 100m</td>
</tr>
</tbody>
</table>

**Table 4. Treatment guidelines depending upon severity of COPD**

<table>
<thead>
<tr>
<th>COPD</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild COPD</td>
<td>Short acting bronchodilators, when needed.</td>
</tr>
<tr>
<td>Moderate COPD</td>
<td>Regular treatment with one/more bronchodilators; Pulmonary rehabilitation.</td>
</tr>
<tr>
<td>Severe COPD</td>
<td>As in moderate COPD, plus inhaled corticosteroids; Treatment of complications.</td>
</tr>
</tbody>
</table>
Table 5. Guidelines for physicians on tobacco cessation

Follow the 5A strategy

- ASK (about tobacco use)
- ASSESS (the status and severity of use)
- ADVISE (to stop)
- ASSIST (in smoking cessation)
- ARRANGE (follow-up programme)

Details of ADVICE for the patient

- Review your tobacco use. Accept that smoking is a problem and harmful for your health.
- Make a decision and determination to quit. Don't be over confident that you can quit any time you like.
- Share your decision with family, friends, doctor. Accept their help.
- Fix a quit date. Don't postpone.
- Remove ashtrays and other objects that are reminders to the habit.
- Keep away from trigger situations.
- Adopt healthy lifestyle such as relaxation, exercise, plenty of water, fruits, vegetables and avoid tea/coffee/alcohol.
- Take help from family, friends and doctor.

Table 6. First few steps of quitting tobacco smoking

A. To reduce quantity

(i) Change to non-preferred brand.
(ii) Keep a record of the amount and frequency of tobacco use.
(iii) Decrease the number of puffs when smoking.
(iv) Leave large stubs.
(v) Don't inhale deeply.

B. To deal with triggers when you have an urge to smoke (Trigger coping)

(i) For extra-ordinary urge to take tobacco, try alternatives (chewing gum, toffee, peppermint, cardamom).
(ii) Increase your water intake.
(iii) Breathe deeply and quietly.
(iv) Do some other work to engage your mind and to keep your mind off tobacco.
(v) Delay the act of smoking—count till 100 and think of pleasant situations.

C. Once you quit

(i) Learn to say “no” to tobacco offers from others.
(ii) Don’t take even a single puff.
(iii) Try to remain in smoke free areas.
(iv) Avoid company of smokers and even tobacco chewers.
(v) Make a group of people who have quit tobacco—share their experiences.
(vi) Collect the money saved from each pack of cigarette or “paan masaala”. Buy a gift for your loved ones with that money.
(vii) Try alternate ways to deal with mental stress and tension, such as relaxation, deep breathing, listening to music.
(viii) Remember there can be some withdrawal symptoms after quitting, such as headache, irritability, lack of concentration, etc. But bear with them. These are temporary and disappear in a few days.

Even if you fail in quitting smoking

- Don’t get disheartened—TRY AGAIN.
- Seek help of those who have quit smoking.
- Seek professional help and medical advice.
General measures aimed at reducing risk of COPD include the following: (a) avoiding open burning of crop residue, (b) use of water to suppress dust, and (c) wearing masks at work places in areas of dust generation.

Specific measures such as the use of smokeless chullahs should be aimed at reducing risk associated with solid fuel combustion and environmental tobacco smoke (ETS) exposure.

Substitution of solid fuels with LPG or electricity is the best approach. The “kitchen” at home should at least be located outside the living and sleeping areas. Kitchens should be adequately ventilated by providing ‘chimneys’, exhaust pipes and/or fans.

Exposure to products of solid fuel combustion can be minimized by the use of smokeless chullahs, reducing the duration of stay in the kitchen or place of fuel use, and by covering nose and mouth with a thin cloth near the sources of combustion.

Exposure to ETS can be reduced by stopping/minimizing indoor smoking, especially in front of children, and by adequate ventilation in the living rooms.

### DRUG TREATMENT

1. **Bronchodilators.** Bronchodilator medication is central to the symptomatic management of COPD\(^45\). Inhaled drugs are preferred to oral preparations\(^46\). However, the choice of drugs depends on the availability of medications and patient’s affordability (Table 7). Short-acting bronchodilators can be used ‘as-needed’ to relieve intermittent or worsening symptoms, and on regular basis to prevent or reduce persistent symptoms\(^47\). Stepwise treatment should be recommended. In general, nebulized therapy for stable patients is not appropriate unless it has been shown to be better than conventional dose therapy\(^48-50\).

Table 7. Commonly used bronchodilator drugs in India

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Metered Dose/Dry Powder Oral Inhalers (µg/dose)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Beta agonists</strong></td>
<td></td>
</tr>
<tr>
<td>Salbutamol</td>
<td>100-200</td>
</tr>
<tr>
<td>Terbutaline</td>
<td>250-500</td>
</tr>
<tr>
<td>Salmeterol</td>
<td>25-50</td>
</tr>
<tr>
<td>Formoterol</td>
<td>6-12</td>
</tr>
<tr>
<td>Bambuterol</td>
<td>10-12 mgday</td>
</tr>
<tr>
<td><strong>Anticholinergics</strong></td>
<td></td>
</tr>
<tr>
<td>Ipratropium</td>
<td>40-80</td>
</tr>
<tr>
<td>Tiotropium</td>
<td>18</td>
</tr>
<tr>
<td><strong>Methylxanthines</strong></td>
<td></td>
</tr>
<tr>
<td>Aminophylline</td>
<td>225-450 mgday</td>
</tr>
<tr>
<td>Theophylline</td>
<td>200-600 mgday</td>
</tr>
</tbody>
</table>

The long-acting inhaled beta-agonist salmeterol has been shown to improve health status significantly in doses of 50 µg twice daily. Similar data for short-acting beta-agonists are not available. Use of inhaled tiotropium (an anticholinergic) once daily also improves symptoms and health status\(^53\).

Combining drugs with different mechanisms and durations of action may increase the degree of bronchodilatation for equivalent or lesser side effects. A combination of a short-acting beta-agonist and the anticholinergic drug ipratropium in stable COPD produces greater and more sustained improvements in FEV\(_1\), than either alone and does not produce evidence of tachyphylaxis\(^54\).

The addition of oral theophylline should normally be considered only if inhaled treatments have failed to provide adequate relief. Anhydrous forms of theophylline should be discouraged. Sustained release preparations are better\(^55\). All studies that have shown efficacy of theophylline in COPD were done with slow-release preparations. Addition of theophylline to β\(_2\)-agonists or anticholinergics may produce additional improvements in lung function and health status\(^56, 57\). However, combination of salbutamol with theophylline in a single tablet is not recommended.

2. **Corticosteroids.** Inhaled corticosteroids do not change the rate of decline in lung function, but can increase post-bronchodilator FEV\(_1\),

Regular treatment with short-acting bronchodilators is cheaper but less convenient than treatment with long-acting bron-
reduce the number of exacerbations, and slow
the rate of decline in health status\textsuperscript{58-61}.

Regular treatment with inhaled
glucocorticosteroids should be prescribed for
symptomatic patients with COPD with a
documented spirometric response to glucocorti-
costeroids or for those with FEV\textsubscript{1} <50%
predicted and repeated exacerbations requiring
treatment with antibiotics or oral
glucocorticoids\textsuperscript{62-65}. Long-term treatment is
required in such patients; in fact, withdrawal of
inhaled corticosteroids can lead to increase in
symptoms and exacerbation rate. Chronic
treatment with systemic glucocorticosteroids
should be avoided because of unfavourable
benefit-to-risk ratio.

3. Role of other drugs. The use of antibiotics
other than treating infectious exacerbations of
COPD and other bacterial infections, is not
recommended\textsuperscript{66}. Although a few patients with
viscous sputum may benefit from mucolytic
agents (such as, ambroxol, carbocysteine,
iodinated glycerol, etc.), the overall benefit
seems to be very small\textsuperscript{67}.

Cough, although sometimes a troublesome
symptom in COPD, has a significant protective
role. Hence, the regular use of antitussives
should be discouraged in stable COPD.

The use of respiratory stimulants like
doxapram, almitrine bismesylate are not
recommended for regular use in stable patients.
Sedatives and narcotics should be avoided in
patients with COPD because of their respiratory
depressant effects and potential to worsen
hypercapnia.

Malnutrition (both under nutrition and over
nutrition) should be managed appropriately\textsuperscript{68, 69}.
Nutritional supplements can increase fat free
mass and muscle strength. A diet rich in
proteins and fats, but low in carbohydrates, is
preferred.

Currently available prophylactic vaccines for
influenza are not recommended for routine use,
as insufficient information is available on
serotypes prevalent in India. They may, however, be administered to the selected
patients (especially the elderly). Similarly,
routine administration of pneumococcal vaccine
is not recommended\textsuperscript{70}. Immuno-modulatory
drugs may also have a moderate protective role
in reducing infective exacerbations\textsuperscript{71}.

Pulmonary Rehabilitation

Pulmonary rehabilitation is a multi-dimen-
sional continuum of services directed to persons
with pulmonary disease and their families,
usually by an interdisciplinary team of
specialists, with the goal of achieving and main-
taining the individual’s maximum level of
independence and functioning in the commu-
nity.

Goals of pulmonary rehabilitation are: (a) to
reduce symptoms, disability and handicap, and
(b) to improve functional independence. It
should comprise of physical training
programme, disease education, and nutritional,
psychological, social and behavioural
intervention (including smoking cessation)\textsuperscript{72}.
The programme should be tailored to individual
functional needs and capacity, and should be
targeted especially to patients with co-existing
locomotor or cognitive impairment, and to those
with associated cardiac disease. Clinicians,
physiotherapists, dieticians, occupational
therapists, social workers, nurses and
pulmonary function technicians should be
involved in rehabilitation programmes.
Optimum medical management should
continue along with the rehabilitation process.

MANAGEMENT OF ACUTE
EXACERBATIONS

Exacerbation of COPD is defined as “a
sustained worsening of the patient’s condition,
from the stable state, and beyond normal day-to
day variations, that is acute in onset and
necessitates a change in regular medication”\textsuperscript{73}.

Patient Assessment

The symptoms of an exacerbation are
increased breathlessness often accompanied by
wheezing, increased cough and sputum, change
of the colour or tenacity of sputum, and fever.
The common causes of an exacerbation are infection of the tracheobronchial tree and air pollution\textsuperscript{74-76}. The cause of approximately one-third of severe exacerbations cannot be identified. Conditions that may mimic an acute exacerbation include pneumonia, congestive heart failure, pneumothorax, pleural effusion, pulmonary embolism, and arrhythmias. These conditions should be ruled out by clinical examination and investigations.

The assessment of severity of acute worsening is based on the patient's medical history before the exacerbation, symptoms, physical examination, lung function tests, arterial blood gas measurements, and other laboratory tests. The medical history should cover the period of worsening since the new symptoms have been present, the frequency and severity of breathlessness and coughing attacks, sputum volume and colour, limitation of daily activities, any previous episodes/exacerbations, hospitalisation, and the present treatment regimen.

\textit{Treatment of Acute Exacerbations}

Bronchodilators are the cornerstone of managing exacerbations of COPD. Patients need to increase the dose and/or frequency of existing bronchodilator therapy. New drugs, which patient is not taking at the time of worsening, may be added. Short-acting bronchodilators should ideally be administered using inhalers (preferably with spacers). In a severe case, nebulizers may be used for drug administration. In situations where these drugs are not available, parenteral aminophylline can be used with due attention to its toxicity. Aminophylline dose should appropriately be modified in elderly patients, those in congestive cardiac failure or having liver cirrhosis, and those already taking oral methylxanthines, cimetidine, ciprofloxacin or erythromycin.

Antibiotics should be used when symptoms of breathlessness and cough are increased and sputum is purulent and increased in volume\textsuperscript{77,78}. The choice of antibiotic depends on the affordability of the patient, the severity of exacerbation and the bacterial spectrum\textsuperscript{79}. Amoxycillin, doxycycline, cotrimoxazole, fluoroquinolones or a second generation macrolide/cephalosporin are used as the first choice. For severe exacerbations higher-grade antibiotics, such as coamoxiclav or a fourth generation cephalosporin can be used.

Systemic glucocorticoids should be used in acute exacerbations. They shorten recovery time and help to restore lung function more quickly\textsuperscript{80-82}. A dose of 40 mg oral prednisolone per day (or equivalent) for 5-10 days is recommended. Carefully look for tuberculosis by sputum examination and chest radiograph before starting corticosteroids.

Controlled oxygen therapy can be administered at low flow rates (preferably with a Venturi mask) with monitoring for features of CO\textsubscript{2} retention\textsuperscript{83}. Chest physiotherapy, inhaled corticosteroids and mucolytic agents are generally not useful in the management of acute exacerbations.

Patients with the following features should be hospitalised for further management:

1. Marked increase in intensity of symptoms, such as sudden development of resting dyspnoea;
2. Onset of new physical signs (\textit{e.g.}, cyanosis, drowsiness, confusion, flaps, peripheral oedema);
3. Failure of exacerbation to respond to initial medical management;
4. Significant co-morbidities, such as diabetes or associated cardiac disease;
5. Newly occurring arrhythmias; and
6. Diagnostic uncertainty.

\textbf{DISEASE PROGRESSION AND PROGNOSIS}

Physiological changes characteristic of the disease include mucus hypersecretion, ciliary dysfunction, airflow limitation, pulmonary hyperinflation, gas exchange abnormalities, pulmonary hypertension and cor-pulmonale, and they usually develop in this order over the course of disease. Pulmonary hypertension
A PRACTICAL APPROACH AT DIFFERENT LEVELS OF CARE

Primary Care Level (Primary health centres, Dispensaries, General practice clinics)

Facilities for diagnosis at the primary healthcare facilities are generally few. Diagnosis can be made with the help of a good history and physical examination following the algorithm shown in the figure given below. Sputum examination for AFB should be done as per RNTCP guidelines. However, isolated positive result may not mean pulmonary tuberculosis. Further investigations should be done to rule out other conditions.

The best guide to the progression of COPD is the change in FEV1 over time. FEV1 declines with normal ageing at about 30 ml/year and this increases to an average of 45 ml/year in smokers. However, the individual susceptibility to cigarette smoking is very wide. Smoking produces only small improvement in patients with COPD at a rate of about 30 ml/year. Depending on disease severity, the five-year mortality of patients with COPD varies from 40% to 70%.

The three major causes of death have been pulmonary tuberculosis, lung cancer, and cardiovascular disease. The age corrected py FEV1 the most commonly recognized prognostic factor.

Figure: Algorithm for diagnosis and management at different levels of health care.
If the sputum is negative, a provisional diagnosis of COPD can be made and treatment given depending on the disease severity, classified as per table 2.

**Mild COPD**

(a) Advice on smoking cessation (Tables 5 & 6) and reduction of exposures to other risk factors (for all stages).

(b) Drug therapy. Salbutamol or terbutaline (inhalational): 2-4 inhalations/day on “as and when needed” basis.

**Moderate COPD**

(a) Start with oral theophylline: 300-600 mg per day.

(b) Inhalational ipratropium or tiotropium on regular basis.

(c) Inhalational salmeterol or formoterol: twice daily.

(d) Salbutamol or terbutaline on “as and when needed” basis.

**Severe COPD**

(a) Treatment steps (a to d) as above.

(b) In the presence of infective complications: A short course of oral antibiotics amoxycillin, quinolones (levofloxacin or gatifloxacin), macrolides (azithromycin/clarithromycin/roxithromycin) or oral first/second generation cephalosporin (cephalexin, cefadroxil). If response is not good, refer to a secondary care level centre.

**Secondary Care Level (District level hospitals and clinics)**

(a) Chest radiograph and sputum examination should be done to look for complications, such as pneumonias, pneumothorax, chronic cor-pulmonale, etc.

(b) Treat infective exacerbation with a course of antibiotic (as above). Higher grade antibiotics may be required.

(c) Confirm diagnosis and severity of COPD with the help of spirometry.

(d) Institute drug treatment as at primary care level.

(e) Consider addition of inhaled corticosteroids (beclomethasone, fluticasone or budesonide), if COPD is severe. Add long term inhaled corticosteroid therapy, only if the patient shows good response to a trial of inhaled corticosteroids administered for about six weeks. A patient who shows frequent exacerbations can also be advised long term inhaled steroid treatment.

If the patient does not show good response to treatment, refer to a tertiary care level centre.

Faulty technique is perhaps the important cause of failure of response to inhalational therapy. It is, therefore, important to properly explain and let the patient practise inhalation technique in your presence.

**Tertiary Care Level (Medical colleges, large corporate, institutional and speciality hospitals)**

It is important for a tertiary care centre to establish facilities for speciality advice and intensive respiratory care. This should include assisted ventilation and all other steps of acute care such as the monitoring of vital parameters, blood gas assessment, maintenance of blood pressure, fluids, electrolytes, nutrition and general organ functions.

At a tertiary care centre, acute exacerbation should be handled followed by stabilisation and rehabilitation therapy.

Respiratory rehabilitation. Advice on respiratory rehabilitation is important at all levels of care. Advice on smoking cessation and avoidance of risk factors is an essential component of respiratory rehabilitation. Guidelines on advice to quit smoking are listed in tables 5 and 6.

Rehabilitation at secondary and tertiary care level centres should include advice on nutrition, maintenance bronchodilators and inhalational corticosteroids, prophylactic vaccines and domiciliary oxygen.

Once the patient is stabilised, he should be sent back to the primary care doctor with appropriate briefing and advice on follow up management.
REFERENCES


24. Becklake MR. Occupational exposures:


47. Ram FSF, Sestini P. Regular inhaled short acting...


Wilson DO, Rogers SM, Sanders MH, Pennock BE, Reilly JJ. Nutritional intervention in


Programme Convener

S.K. Jindal

Participants

Ashutosh Nath Aggarwal, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; VK Arora, Director, Lala Ram Swaroop Institute of Respiratory Diseases, New Delhi; Pradeep Bambery, Department of Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; Madhu Sudan Barthwal, Classified Specialist and Pulmonologist, Military Hospital, Pune; Rajinder Singh Bedi, Chest Consultant, Patiala; Digamber Behera, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; Dhruv Chaudhry, Department of Medicine, BD Sharma Postgraduate Institute of Medical Sciences, Rohtak; Kishore Chaudhry, Senior Deputy Director General, Indian Council of Medical Research, New Delhi; SK Chhabra, Department of Cardiorespiratory Physiology, Vallabhbhai Patel Chest Institute, Delhi; Vishal Chopra, Medical Officer, Punjab Civil Medical Services; Rohini Chowgule, Secretary, Indian Chest Society, and Consultant Pulmonologist, Bombay Hospital, Mumbai; RS Dhaliwal, Department of Cardiothoracic Surgery, Postgraduate Institute of Medical Education and Research, Chandigarh; George A D'Souza, Department of Medicine, St. John
Medical Ganguly, Department of Medicine, Vivekanand Institute of Medical Sciences, Kolkata; SN Gaur, Department of Respiratory Medicine, Vallabhbhai Patel Chest Institute, Delhi, and Secretary, National College of Chest Physicians (India); Girdhari Lal Goyal, Director Health Services (ESI), Punjab; Randeep Guleria, Department of Medicine, All India Institute of Medical Sciences, New Delhi; Dheeraj Gupta, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; KB Gupta, Department of Chest Diseases and Tuberculosis, BD Sharma Postgraduate Institute of Medical Sciences, Rohtak; Prahlad Rai Gupta, Department of Respiratory Diseases and Tuberculosis, Swai Man Singh Medical College, Jaipur; HS Hira, Department of Medicine, Maulana Azad Medical College, New Delhi; Nirmal Kumar Jain, Department of Respiratory Diseases and Tuberculosis, Swai Man Singh Medical College, Jaipur; Ashok Janmeja, Department of Chest and Tuberculosis, Govt. Medical College Hospital, Chandigarh; LR Jena, GC, CRPF, Pinjore; Surinder Kumar Jindal, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; S Kashyap, Department of Pulmonary Medicine, Indira Gandhi Medical College, Shimla; SK Katiyar, Department of Chest Diseases and Tuberculosis, GSV Medical College, Kanpur; GC Khilnani, Department of Medicine, All India Institute of Medical Sciences, New Delhi; Jai Kishan, Department of Chest and Tuberculosis, Medical College Hospital, Patiala; DDS Kulpati, Consultant Pulmonologist, Sir Ganga Ram Hospital, New Delhi; Rajesh Kumar, Department of Community Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; SS Lehal, Department of Medicine, Government Medical College Hospital, Chandigarh; AA Mahashur, Consultant Chest Physician, PD Hinduja Hospital, Mumbai; Savita Malhotra, Department of Psychiatry, Postgraduate Institute of Medical Education and Research, Chandigarh; TM Mohankumar, Senior Consultant Pulmonologist, Institute of Pulmonary Medicine and Research, Sri Rama Krishna Hospital, Coimbatore; JN Pande, Department of Medicine, All India Institute of Medical Sciences, New Delhi; BNBM Prasad, Classified Specialist and Pulmonologist, Military Hospital, New Delhi; Rajendra Prasad, Department of Respiratory Diseases and Tuberculosis, King George Medical College, Lucknow; P Ravindran, Department of Respiratory Medicine, Medical College and Hospital, Trivandrum; M Sabir, Department of Medicine, and Head of Respiratory Division, SP Medical College, Bikaner; RM Sarnaik, Chest Physician, Nagpur; Meenu Singh, Department of Pediatrics, Postgraduate Institute of Medical Education and Research, Chandigarh; Virendra Singh, Department of Medicine, Swai Man Singh Medical College, Jaipur; SK Sharma, Department of Medicine, All India Institute of Medical Sciences, New Delhi; UPS Sidhu, Department of Chest Medicine, Dayanand Medical College, Ludhiana; Sudha Suri, Department of Radiodiagnosis, Postgraduate Institute of Medical Education and Research, Chandigarh; PS Tampi, Classified Specialist and Pulmonologist, INHS Asvini, Colaba, Mumbai; Cherian Varghese, National Professional Officer, World Health Organization, New Delhi; S Varma, Department of Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; K Venu, Department of Chest Medicine, Osmania Medical College, Secunderabad; Ajit Vigg, Consultant Chest Physician, Apollo Hospital, Hyderabad; VK Vijayan, Director, Vallabhbhai Patel Chest Institute, Delhi, and Regent (India), American College of Chest Physicians; Jagdeep Whig, Department of Medicine, Dayanand Medical College Hospital, Ludhiana.

Consultants and Reviewers

Sydney Parker, Vice President (Health and Science Policy), American College of Chest Physicians, USA; UBS Prakash, President, American College of Chest Physicians, USA; Bela Shah, Senior Deputy Director General, Indian Council of Medical Research, New Delhi; PS Shankar, Director, Mediciti (MIMS), Hyderabad; Farukh E Udwadia, Consultant Physician, Breach Candy Hospital, Mumbai; Cherian Varghese, National Professional Officer, World Health Organization, New Delhi.

Rapporteurs

Ritesh Aggarwal, T Balamugesh, Dinesh Goyal, Rajesh Gupta, Surinder Kumar Mahajan, Javed Malik, Pralay Sarkar, and Chetty Mahendran, Postgraduate Institute of Medical Education and Research, Chandigarh.