Management of Chronic Stable COPD

Sujeet Rajan*, Ratna Balakrishnan**

This chapter will focus on managing COPD based on treatment (largely pharmacological). It is our endeavour to ensure that the reader is left with the basic knowledge of various pharmacological options and why they need to be used as the disease progresses.

The successful management of COPD rests on the following key practice areas which we will elucidate as we go ahead:

1. Arriving at a diagnosis (already referred to in a previous chapter)
2. Communicating the diagnosis
3. Explaining and commencing a treatment plan
4. Monitoring the patient
5. Not missing the forest for the trees – reviewing the patient as a whole

1. Arriving at a diagnosis

COPD diagnosis is arrived at by a good history and clinical examination, almost always to be supported by a spirometry test.

Though progressive exertional dyspnoea and cough remain some of the most frequent symptoms of this disease, it is not uncommon for patients to be mislabelled as asthma.

The simple and key practical pointers for differentiation from asthma include:

i. COPD does not usually begin in childhood. Asthma often does.
ii. COPD is often associated with tobacco smoke exposure, but need not. Smokers can also develop asthma, but the link is weak for causation.
iii. COPD usually commences after age 40
iv. COPD dyspnoea is usually slowly progressive over months to years. Asthma dyspnoea is more often paroxysmal.
v. Inhaled, and especially oral steroid ‘treatment trials’ have a dramatic positive response for asthma symptoms and lung function. In COPD the response is rarely dramatic.

Once you are suspecting COPD in a patient, the need to order a spirometry test is essential, both to confirm the diagnosis, and grade the severity. Based on spirometry results as detailed below, the COPD is graded, and a conversation with the patient needs to begin.

2. Communicating the Diagnosis

Remember when a patient with COPD enters a physician’s clinic, he comes with a lot of expectations, so do not destroy the hope with which he has come to you for help. In this regard, we would strongly advise not to use the following statements:

i. Your disease is not curable
ii. You have a badly damaged lung (perceived as a death sentence by some patients)
iii. Persistently stressing that he has self-inflicted this disease by smoking tobacco. (the patient has not come to hear about why his problem has occurred, but mainly how to solve it)
iv. Drugs will not help you much now (pessimistic and untrue statement)
v. You are becoming a burden on your family (the patient knows this and doesn’t need to be reminded often about it)
vi. You will respond dramatically to my treatment (unlike asthma, the response to treatment is rarely dramatic, but happens positively over time.)

Most patients have no clue about how their lungs look like. Let alone patients, most doctors when entering medical school often don’t have a clue of what to expect when they have a first look at cadaver lungs. I would stress a simple approach to explaining anatomy of the lungs and airways to simplify the disease for the patient. This will make the patient understand. Understanding the disease is likely to make the patient commence the treatment plan, and adhere better, both vital components to the successful out-patient management of COPD.

Step 1: Draw the lung structure on a piece of paper, and show where the lower airways begin. Compare the lungs to two balloons in either part of the thoracic cavity, which inflate and deflate during breathing. Explain that these lungs are too inflated in COPD, and almost about to burst, especially in severe disease. This hyperinflation in turn makes it more difficult to get more air in, and so the patient has to stop after short exertions itself, especially in severe disease.

Step 2: Draw a simple cross-section of the airways now (see figure 1), and explain to the patient that the airways are like a tree turned upside-down, with the trachea being the trunk, and the various branches being the bronchi. The trachea never closes down, otherwise he would drop dead (if that happened for a while!), but the bronchi do. Now draw a cross-section of the airway and explain to the patient what a narrowed airway looks like on cross-section. (Using Breathing-tubes can often help – Figure 2).

Step 3: Finally, the patient needs to understand that his airway is unlikely to return to normal caliber, (unlike an asthma patient’s airways) with inhaled steroids. The airway will be dilated with appropriate treatment (see treatment of COPD), but never to normal. The dilated airway permits air to be let out more easily, allowing better deflation of the lungs (balloons), and therefore more easier inflation in the next breath. Thereby the patient breathes more comfortably, and is able to do more and walk more.

Fig. 1 : Normal, severely narrowed and partly narrowed airways
It is essential that the patient is made to understand the above, both as simply as possible, and with adequate time for the message to get across. Time invested in clinical practice on this will significantly boost patient understanding, confidence and trust in the doctor to deliver correctly.

3. Explaining and commencing a treatment plan
If the diagnosis of COPD is explained well, commencing a treatment plan is not difficult. Everything falls into place as you and your patient have understood where he stands. Spirometry becomes essential in grading the severity of disease, and as you will see from the diagram below, the FEV1 serves as your marker for disease severity grading. You diagnosis is written down after obtaining the FEV1 % predicted from the spirometry report.

At every stage of disease however, there are certain important interventions that are critical to the successful management of COPD. These include the following:

MEASURES APPLICABLE TO ALL STAGES

1. Avoidance of active and passive tobacco smoke exposure:
   a. Write it down on the prescription.
   b. Spend a few minutes explaining to the patient how continued cigarette/beedi smoking affects his lung function, and the passive smoke effects on his family and friends. Just a few minutes of counselling can achieve a quit rate of 3 %!
   c. Ensure a close relative or friend is also present. This allows you to have an anchor/mediator for the process to commence and be monitored well.
   d. Use nicotine 2 and 4 mg chewing gum. Nicotine replacement therapy helps to reduce cravings. As should be known, cravings are short-lived, and hence gum can help. The gum should be chewed by the patient, and then parked between the buccal mucosa and teeth/gums. It can be rolled out to chew again after some time. For patients smoking 10 or less cigarettes/day, the 2 mg chewing gum should do. For those smoking more, the 4 mg gum may be more effective.
   e. If counseling and nicotine replacements don’t help, a drug may be required. Choose from bupropion hydrochloride (to be avoided in heavy alcohol consumers due to lower seizure threshold) or varenicline (avoid in severely depressed individuals due to higher suicide risk) as explained in chapter on “Smoking Cessation Programs and Other Preventive Strategies for Chronic Obstructive Pulmonary Disease” by Raj Kumar and VK Vijayan.

2. Avoidance of non-tobacco exposures and other environmental measures:
Smoking is not the only cause of COPD. A variety of non-tobacco exposures ranging from wood smoke (chullahs), biomass fuels like kerosene, nitrogen dioxide from cooking gas in poorly ventilated kitchens, sulphur dioxide from industrial gases etc. have been implicated in the causation of COPD. Due diligence to an accurate history looking for these exposures is important. Occupational exposures like cadmium too have a significant impact on the pathogenesis of COPD.

In relatively warmer countries, indoor temperatures need to be higher in cold weather in both living rooms and bedrooms; patients with COPD need to be able to afford heating, and houses may need to be designed with warmer bedrooms.

3. Checking the patient is using his inhaler both correctly and regularly.
Inhaler therapy is the mainstay of COPD pharmacotherapy. So whatever stage the patient presents with, never forget to check that the patient has been, or will be, using his inhaler correctly. If the patient has been using his inhaler correctly, then do confirm that use has also been regular, especially with anything more than mild COPD.

This applies to any stage of the disease.

4. Vaccination against pneumococcal and influenza infection.
The influenza vaccine can reduce serious illness and death in COPD by close to 50 %. Vaccines containing live inactivated viruses are recommended. The strains are adjusted each year for appropriate effectiveness and should be given once a year, prior to the flu season.

In studies in patients with COPD, a 52 % decrease in hospitalizations for all episodes of pneumonia and influenza were noted, and a 70 % reduction in deaths from all causes.

Pneumococcal polysaccharide vaccine is also recommended for COPD patients 65 and older. This vaccine has also been shown to reduce the incidence of community-acquired pneumonia in COPD patients below 65 years, but with an FEV1 < 40 % predicted.
Important and never to be forgotten.

6. **Action plan for acute exacerbations of COPD:**

Just as in asthma, a relief inhaler is essential to be prescribed, regardless of the stage of COPD. This could be salbutamol, levosalbutamol, ipratropium bromide or a combination of salbutamol and ipratropium bromide. However correct your prescription for the long-term is, never forget the short-acting bronchodilator. Just like you wouldn’t forget a sub-lingual nitrate prescription for a patient with ischaemic heart disease.

5. **Prescribing a short-acting inhaled bronchodilator/s for quick relief of symptoms.**

Just as in asthma, a relief inhaler is essential to be prescribed, regardless of the stage of COPD. This could be salbutamol, levosalbutamol, ipratropium bromide or a combination of salbutamol and ipratropium bromide. However correct your prescription for the long-term is, never forget the short-acting bronchodilator. Just like you wouldn’t forget a sub-lingual nitrate prescription for a patient with ischaemic heart disease.

7. **Patient education.**

As explained in “communicating a diagnosis”, this is essential at every stage for patients to understand their disease better, and adhere better to treatment strategies. It would include disseminating booklets on the disease, checking and correcting inhaler technique at the first and every visit, helping family members to understand the disease better, and guiding patients to appropriate websites to understand their disease better.

PHARMACOTHERAPY ACCORDING TO STAGE

**Long-acting beta-agonists (LABA):**

Bronchodilators are the cornerstone in treatment of COPD. They are prescribed on an as needed (as detailed above), or on a regular basis to prevent or reduce symptoms. The most common drugs used, beta-agonists, basically consist of the short-acting (SABA) and the long-acting (LABA) types. They act by stimulating the beta 2-receptors on airway smooth muscle, increase cyclic AMP and cause smooth muscle relaxation, with subsequent bronchodilatation. The inhaled route is by far the most efficient way to deliver these drugs with greatest bronchodilation and the highest fraction of the drug reaching the lungs and airways.

Oral therapy is slower in onset and has more side effects than inhaled treatment. Most of the inhaled beta2-agonists have an onset of action within 5 minutes, and that can last up to 4 to 6 hours. The LABAs have much longer duration of action up to 12 hours, and with no loss of effect after overnight use and with regular use. Formoterol is unique among the LABAs in that it has both a fast (like a SABA) and a long duration of action. SABAs in COPD management today should be restricted to quick-relief of symptoms only, allowing for more long-acting drugs to form the mainstay of maintenance therapy. The main side effects of beta-agonists include tremors and tachycardia.

Formoterol 12 to 24 mcg twice daily maximum
Salmeterol 50 mcg twice daily, up to 100 mcg twice daily maximum
Indacaterol 150 to 300 mcg once daily
Nebulised Aformoterol 15 mcg twice daily

**Long-acting anti-cholinergics (LAMA):**

Anticholinergics include ipratropium, oxitropium and tiotropium bromide. The first two are short-acting anticholinergics and the last one is long-acting. These drugs act by blocking the effects of acetylcholine on the M3 receptors. The short-acting drugs also act on the pre-ganglionic M2 receptors and these effects are less desirable. Tiotropium, the only long-acting antimuscarinic drug (LAMA) has specific effects on the M1 and M 3 receptors only and has a duration of action of up to 24 hours. Though ipratropium acts for longer than most short-acting beta2-agonists, tiotropium’s long duration of action makes it the preferred inhaled bronchodilator in the initiation of long-term maintenance bronchodilation for COPD. Narrow-angle glaucoma is an absolute contra-indication for the use of tiotropium. Acute glaucoma being precipitated by nebulised ipratropium through facemask has been reported. In patients with an enlarged prostate, symptoms of prostatism may increase with tiotropium. Anther common side effect of inhaled anticholinergics is dryness of mouth.

Tiotropium 18 mcg once daily
Combination of LABA + LAMA

As COPD severity increases to a moderate type, (FEV1 < 80 % predicted), most patients will benefit with a combination of at least 2 classes of long-acting bronchodilators. This will help maximize bronchodilation by different mechanisms, and reduce hyperinflation to the maximum possible degree.

Use any one of these combinations:
Salmeterol + Tiotropium
Indacaterol + Tiotropium or
Formoterol + Tiotropium (the only one available in a single inhaler)

**Methylxanthines**

Though controversy remains about the exact role of these drugs, they remain the most commonly used drugs for COPD in India. The oral, sustained release formulations are usually used as all studies that have shown efficacy in COPD have been done with these formulations. The therapeutic ratio of theophylline is low with levels > 5-10 mg/ml associated with significant toxicity. Hence it is rare for any patient to need more than 300 to 400 mg of theophylline in a day. Higher doses may occasionally be required in young smokers with a high BMI, who are likely...
Drugs | Mode of delivery | Dose in COPD | Drugs | Mode of delivery | Dose in COPD
---|---|---|---|---|---
1. **Short acting bronchodilators**
   a. Salbutamol
      i. Inhaler (100 mcg)
      ii. Rotacaps (200 mcg)
      iii. Respule (2.5 mg/2.5 ml)
      i. ii. 1-2 Inhalations every 4-6 hour, need-based
      iii. 2.5 mg every 4-6 hours need-based
   b. Ipratropium bromide
      i. Rotacaps (40 mcg)
      ii. Inhaler (20 mcg)
      iii. Respule (500 mcg/2 ml)
      i. ii. 2 inhalations 4 times daily max 10-12 per day
      iii. 0.5 mg every 6-8 hours
   c. Salbutamol + Ipratropium
      i. Rotacaps (Ipratropium 40 mcg + levosalbutamol 100 mcg)
      ii. Inhaler (Ipratropium 20 mcg + levosalbutamol 50 mcg)
      iii. Respules (Ipratropium 500 mcg + levosalbutamol 1.25 mg/2.5 ml)
      i. ii. 2 inhalations 4 times daily max 10-12 per day
      iii. 1 Respule every 4-6 hours
2. **Long Acting Bronchodilators**
   d. Salmeterol
      i. Rotacaps (50 mcg)
      ii. Inhaler (25 mcg)
      i. 1 Rotacap twice daily
      ii. 2 – 4 puffs twice daily
   e. Formeterol
      i. Rotacaps (12 mcg)
      i. 1-2 Rotacaps twice daily
   f. Aformeterol
      i. Respules (15 mcg/2ml)
      i. 1 Respule twice daily
   g. Tiotropium
      i. Rotacaps (18 mcg)
      ii. Inhaler (9 mcg)
      i. 1 Rotacap once daily
      ii. 2 puffs once daily
3. **Combination LABA + Inhaled Corticosteroids**
   h. Fluticasone + Salmeterol
      i. Rotacaps (250) Fluticasone 250 mcg + Salmeterol 50 mcg
      ii. Rotacaps (500) Fluticasone 500 mcg + Salmeterol 50 mcg
      i. 1 Rotacap twice daily
      ii. 2 puffs twice daily

4. **Other Important Combinations**
   j. Tiotropium + Formeterol
      i. Rotacaps (Tiotropium 18 mcg + Formeterol 12 mcg)
      i. 1 Rotacap once daily
      ii. 2 puffs once daily
   k. Tiotropium + Formeterol + Ciclesonide (Triple combination inhaler)
      i. Inhaler (Tiotropium 9 mcg + Formoterol 6 mcg + Ciclesonide 200 mcg)
      i. 2 puffs once daily
      ii. 1 inhalation once daily
   l. Multihaler (Tiotropium 18 mcg + Formoterol 12 mcg + Ciclesonide 400 mcg)
      i. 1 Rotacap twice daily
      ii. 2 puffs once daily

---

to metabolise theophylline much faster. Inhaled long-acting bronchodilators are always to be preferred over theophylline in the regular management of COPD.

The adverse effects are mainly on the cardiovascular and neurological system. Tachycardia and CNS irritability with higher serum levels are known, often aggravating any tremors already present due to beta-agonist therapy. Seizures may occur even in the absence of a prior history of epilepsy. Atrial and ventricular tachyarrhythmias can occur too. Headache, nausea, insomnia and gastritis are some of the other common side effects. It is also important to remember that the drug is largely metabolized by the cytochrome p450 enzyme mixed function oxidases in the liver, and therefore advancing age, liver disease and chronic passive congestion of the liver, especially in COPD patients with cor pulmonale will warrant a reduction in dose. The low cost, anti-inflammatory effects, and possible benefits on diaphragmatic contractility makes it a good choice for adjuvant therapy in severe disease (FEV1 usually below 50 %, and occasionally above that). Reduction in COPD exacerbations with theophylline have been small, although further studies are required.

**Glucocorticosteroids:**
The effect of oral and inhaled glucocorticoids in COPD are
Most studies clearly indicate that they do not alter the Inhaled Steroid (For FEV1 below 50% predicted or in dual combination inhalers). Budesonide 200 to 400 mcg twice daily or Fluticasone 250 to 500 mcg twice daily. As with inhaled steroids in general, side effects like oral thrush and dysphonia are the most common. The former can be reduced by mouth rinsing after inhaler use, or the use of a spacer.

Fluticasone 250 to 500 mcg twice daily
Budesonide 200 to 400 mcg twice daily
or in dual combination inhalers with LABA, as outlined in the Table 1.

Oxygen Therapy

Oxygen is one of the earliest and few interventions to have shown to reduce mortality in COPD. Oxygen therapy also reduces anxiety, cardiac arrhythmias and improves sleep quality in COPD patients who fit the criteria for regular domiciliary use. Two studies have shown that patients with hypoxemia, and not treated with LTOT have a higher risk for hospital admissions and mortality.

The flow rate of oxygen prescribed should be sufficient to maintain the patient’s oxygen saturations above 88% at sea level, and for at least 17 hours a day. Alternatively, oxygen can be administered during exercise, sleep or strenuous activity whenever the need arises, even if the oxygen saturations at rest remain above 90%.

The following criteria on blood gases should be used to prescribe long-term oxygen therapy:

i. \( \text{pO}_2 < 55 \text{ mmHg} \)

ii. \( \text{pO}_2 55 – 59 \text{ mmHg with pulmonary hypertension, cor pulmonale, polycythaemia, edema from right heart failure or impaired mental state.} \)

iii. Desaturation during sleep, exercise and high altitude

When oxygen is prescribed, always write out the prescription the following way:

Oxygen at ___ ltrs/min for ___ hours/day, with nasal prongs/facemask and delivered by an oxygen cylinder/oxygen concentrator.

Mucolytics

The use of regular mucolytics in the treatment of stable COPD has little evidence to support it. In fact, the GOLD guidelines for the disease stress the fact that these drugs are unlikely to help. A meta-analysis suggested that there is reasonable evidence for some treatment effect, but most of this is related to the use of N-acetylcysteine (NAC).

NAC has also been shown to have significant anti-oxidant effects in vivo.

Mucolytics continue to be used in large parts of the world. Also, given the low risk of adverse effects, oral effervescent NAC at a dose of 600 mg up to twice daily may be a reasonable choice for patients with recurrent exacerbations despite the use of other drugs currently recommended in the guidelines.

Antibiotics

Prophylactic and continuous use of antibiotics has been shown to have no effect on frequency of exacerbation. There is no current evidence for use of antibiotics other than in infectious exacerbations of COPD and other bacterial infections.

A recent study of a five day course of moxifloxacin every 8 weeks for a total of 6 courses versus placebo, has shown to help reduce the frequency of exacerbations by 25%. On further analysis it was shown that a larger reduction (45%) was seen in those patients with mucopurulent sputum at baseline.
Immunomodulators

Studies using immunomodulators in COPD show a decrease in severity and frequency of exacerbations. However, further studies are required before its regular use can be routinely recommended.

Antitussives:

Regular use not recommended in stable COPD.

The pharmacotherapy is simplified and summarized below with good examples:

- **Mild COPD:** LABA (salmeterol alone) or LAMA (tiotropium) alone
- SABA (salbutamol or ipratropium + salbutamol) need-based only
- **Moderate COPD:** LABA + LAMA (formoterol + tiotropium) combination inhaler
- SABA need-based only
- **Severe COPD:** LABA + LAMA combination inhaler
- Inhaled Steroid +/- low dose theophylline
- SABA need-based only
- **Very severe COPD:** LABA + LAMA combination inhaler
- Inhaled steroid + low dose theophylline
- Evaluate for home oxygen therapy
- N-Acetylcysteine 600-1200mg/day for frequent exacerbators
- SABA Need-based only

4. Monitoring the Patient

Accurate diagnosis, and a good prescription is not enough for a patient with COPD. The COPD patient is on his own after he consults you, and if not given an adequate monitoring plan, is likely to be lost soon to follow-up. Give your patient a monitoring plan, and be clear on what you need to look at on each follow-up. Having a checklist with you will always helps. Here’s what should be on a 10-point checklist.

- i. Symptom check
- ii. Weight
- iii. Adherence to regimen check
- iv. Frequency of SABA use check
- v. Need for antibiotic/oral steroid check
- vi. Spirometry
- vii. 6 minute walk test
- viii. Echocardiography/Right heart catheter/Arterial blood gases
- ix. Patient queries/concerns
- x. Influenza vaccination

To elucidate more on some of the points above:

1. **Symptoms**
   - Has your treatment made a difference to you?
   - Are you able to do more?
   - Are you able to walk more?

2. **Diet, Weight and Haemoglobin**
   - Body mass index is inversely proportional to survival.
   - It is essential to aim for a BMI of over 22 in patients who are underweight. These patients tend to do much worse with higher mortality than others.

Importantly most patients with severe COPD with resting hypoxemia, tend to develop a secondary polycythemia. A baseline Hb value is always helpful in this regard. A drop in Hb in a patient with severe COPD is a worrying sign, and warrants speedy diagnosis and correction, as the anemia (and the consequent drop in oxygen-carrying capacity of the blood) itself, is likely to make the patient more fatigued and dyspnoeic.

3. **Spirometry**

Spirometric values like FEV1 and FVC rarely improve markedly in COPD, and hence constantly repeating these may not be cost-effective or useful. A remarkable improvement in FEV1 on a follow-up study may need you to revise your diagnosis to asthma.

Lung volumes are usually elevated and improvements in inspiratory capacity signify reductions in hyperinflation and correlate with clinical improvements.

A low diffusion is usually suggestive of emphysema, but has more significance from a prognostic point of view and sometimes pre-operatively.

4. **6 minute walk test**

This test is simple to perform and is of far more utility from a monitoring point of view, than standard spirometry. Since most activities of daily living are done at sub-maximal exertion, this test is a very useful predictor of overall cardio-pulmonary and musculoskeletal status, as well as mortality.

6MWD distances drop much more significantly with worsening disease than FEV1, and correspondingly rise significantly with interventions like pulmonary rehabilitation. Hence this test becomes more reliable and indicative of deteriorations and improvements in functional status over time.

5. **Right heart echocardiography and catheterisation**

The estimated pulmonary artery systolic and diastolic pressures can now be fairly accurately assessed on echocardiography, as are the right heart chamber dimensions. Left ventricular function is equally important as a prognostic indicator. Treatments for pulmonary hypertension, however, must be carefully introduced. Studies have shown that pulmonary hypertension in COPD is often over-diagnosed and does not correlate well with invasive right heart measurements. In severe COPD patients with co-existing heart disease, it may be wise to do right heart haemodynamic studies before instituting newer PAH treatments.

A baseline echocardiography followed by annual monitoring should suffice in all patients with severe COPD.

6. **Arterial blood gases**

In all patients with severe COPD, especially the very severe ones, and those hospitalized with an exacerbation, it is advisable to obtain resting arterial blood gases. This is not just to assess the need for home oxygen treatment, but to serve as a baseline for future management decisions, especially with regard to invasive and non-invasive ventilation, the latter especially when instituted at home.
5. Don’t Miss the Forest for the Trees (the systemic disease)
With all our focus on the lungs, it is not uncommon for physicians to miss a few of the smaller, but nevertheless important aspects of COPD treatment. But the bigger error arises when respiratory physicians miss the forest for the trees. COPD is more and more understood as a systemic disease, and failure to recognize its systemic consequences often results in poor patient satisfaction and treatment outcomes. Not just the fact that the patient is elderly, but also by the fact that systemic inflammation has been implicated in disease pathogenesis of late.

Suffice it to say here that we need to treat the patient as a whole, and not just as another patient with lung disease. The term chronic systemic inflammatory syndrome (CSIS) was recently coined in an editorial, and remains an important aspect of COPD as a disease.

The Way Forward
It is evident from all the above that comprehensive management of COPD can have a great impact on the natural course of the disease. Frequent exacerbations can adversely affect a patient’s quality of life, and short as well as long-term pulmonary function. At present most randomized controlled trials have limited themselves to pharmaceutical interventions. There is, however, a need for a more wide-ranging approach. In relatively warmer countries for instance, indoor temperatures need to be higher in cold weather in both living rooms and bedrooms; patients with COPD need to be able to afford heating, and houses may need to be designed with warmer bedrooms.

Regular exercise outdoors seems beneficial, helped by pulmonary rehabilitation. However cold stress is detrimental. Health-care workers have been reluctant to have annual influenza vaccination themselves.

We are lucky in India to have a pharmaceutical industry that has all the relevant combinations we need to optimise pharmacotherapy, including a formoterol-budesonide combination that has double (12 mcg) the regular dose of formoterol specifically for the long-term treatment of COPD. A triple inhaler is also available recently with once daily ciclesonide, tiotropium and formoterol, something the developed countries may require.

There is, however, a need for a more wide-ranging approach. In relatively warmer countries for instance, indoor temperatures need to be higher in cold weather in both living rooms and bedrooms; patients with COPD need to be able to afford heating, and houses may need to be designed with warmer bedrooms.

Finally, we doctors and other health care professionals need to have a much more positive approach to COPD. Sadly, COPD patients are often blamed for a self-inflicted illness - this attitude of health care professionals must change. Smoking is a disease in itself and must be appropriately discussed with the patient and effectively treated with counseling, nicotine replacement therapy, and specific medication (bupropion and varenicline) if needed. Integrated long-term care that combines many of the above approaches is the need of hour to give our COPD patients not just longer lives, but better lives.

References
17. Improving the care of COPD patients - suggested action points by the COPD exacerbations task force for reducing the burden of exacerbations of COPD. Editorial, Primary Care Respiratory Journal 2006;15:139-42.