Chronic Obstructive Pulmonary Disease (COPD)

COPD is a chronic progressive lung condition, with 85-90% of all cases being associated with cigarette smoking. The only intervention that has been proven to slow COPD progression is smoking cessation. Nonetheless drug therapy can play an important role to relieve symptoms, increase exercise tolerance, improve quality of life, and reduce exacerbation and hospitalisation rates.

Learning objectives:

After reading this article pharmacists should be able to:

- Outline the etiology and pathophysiology of COPD
- Describe the typical presenting features of COPD
- Discuss the approach to acute and chronic COPD treatment
- Identify opportunities to optimise the management of COPD

This activity has been accredited for 1 hour of Group One CPD (1 CPD Credit) that may be converted to 2 Group Two CPD Credits upon successful completion of the corresponding assessment for inclusion on an individual pharmacist’s CPD Record. Accreditation number: A1302AP0.

Under the auspices of the Australian Pharmacy Council, the Australian College of Pharmacy may accredit continuing professional development for pharmacists that is eligible to be used as supporting evidence of continuing competence.

The competency standards addressed by this activity include (but may not be limited to) 4.2.2, 4.2.3, 7.1.2, 7.1.3, 7.2.2, 7.2.3.

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Angus graduated from the School of Pharmacy at Bath University in the UK in 1989 and undertook pre-registration training in the South Warwickshire Hospitals. He then worked for three years as a Pharmacist at Dorset County Hospital during which time he completed his MSc in Clinical Pharmacy at Portsmouth University with a thesis looking at Antimicrobial Prescribing.

In 1993 he took up a new role as Pharmacy Manager at the BMI Droitwich Private Hospital, where he became involved in oncology and was a member of the Drug Purchasing group for BMI. Six years later, saw a move back to the South-West of England and appointment as the Pharmaceutical Advisor to South Somerset Primary Care Group/Trust (PCT), with responsibilities including provision of advice on evidence-based and cost-effective prescribing to GPs; it was during this time that he completed his Prescribing Sciences diploma at Liverpool University.

In 2002, he moved to develop a portfolio career which included PCT work, project management, pharmaceutical writing, community pharmacy locums; and education and training for nurses, GP trainees and pharmaceutical industry personnel. Angus’s publications have appeared in the Pharmaceutical Journal, Prescriber and Independent Nurse and include research into patient satisfaction with medication changes.

He moved from the UK to Tasmania with his family and joined UMORE in July 2008.

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Competency standards addressed:

4.2.2, 4.2.3, 7.1.2, 7.1.3, 7.2.2, 7.2.3

Introduction

COPD is a chronic progressive lung condition, with 85-90% of all cases being associated with cigarette smoking. COPD has a huge impact on those individuals it affects and it is predicted to become the third largest cause of death in the world within the next decade. The only intervention that has been proven to slow COPD progression is smoking cessation. Nonetheless drug therapy can play an important role to relieve symptoms, increase exercise tolerance, improve quality of life, and reduce exacerbation and hospitalisation rates.

COPD – emphysema and chronic bronchitis

COPD is an umbrella term that is now used to describe the conditions traditionally referred to as emphysema and chronic bronchitis. Whilst these two conditions do have some different features, they also have similarities and the management of those with COPD is not significantly influenced by these historic definitions.

It has taken time for some clinicians and patients to embrace the term COPD and there is evidence from Australian research that some GPs may continue to have a nihilistic attitude to the condition. It has been suggested that this may reflect the fact that until recently understanding of the disease and its management was poor, together with the fact that the disease is predominantly smoking related.

Prevalence

It has been estimated that around 1.2 million Australians have some form of COPD and that almost 700,000 of these have symptoms that affect them on a daily basis.

COPD is rare in those under 40 years of age, but beyond that age it is estimated that around 1 in 7 people may be affected. As with other common chronic diseases, such as Type 2 diabetes, many people with COPD have not been medically diagnosed and are not actively managing their condition. Data from the UK suggests that for every one person with COPD known to the health system, there are around another two yet to be diagnosed.

COPD is thought to account for around 50,000 hospital admissions per year in Australia with an average length of stay of around 7 days per admission, the condition is therefore responsible for a significant burden on the secondary healthcare system.
**Etiology and Pathophysiology**

COPD is characterised by airflow limitation that is not fully reversible. This limitation occurs as a result of an inflammatory response by the lungs to noxious stimuli, the most significant of which is cigarette smoking.

The risk of developing COPD is related to the cumulative consumption of cigarettes. COPD is most often seen in those with 20 or more pack-years of smoking history. It has been estimated that a long-term smoker who consumes one pack a day has around a 15% chance of developing COPD and this rises to approximately 25% for someone smoking two packs a day long term.

The remaining causes of COPD relate to exposure to other pulmonary irritants, for example in developing countries exposure to smoke from cooking indoors over open fires is a significant cause. Other irritants include a range of dusts and fumes, some of which may be encountered through occupational exposure, such as amongst miners, timber workers etc.

Another well recognised cause of COPD is alpha-1-antitrypsin (AAT) deficiency. AAT is a potent inhibitor of the enzyme neutrophil elastase, which reduces the normal elasticity of tissues in the lungs and other organs. It is estimated that around 1% of all cases of COPD are due to AAT deficiency. It is important to recognise that the degree of deficiency varies between individuals and consequently not all people with AAT deficiency develop COPD, but the risk of doing so is increased by other COPD risk factors, such as smoking, male gender and asthma.

The pulmonary inflammatory response that occurs in COPD is often described as neutrophilic and whilst these cells do play an important role, a range of mediators are also involved such as tumour necrosis factor-alpha (TNF-α), leukotriene B₄ (LTB₄) and interleukin 8 (IL-8). Another factor is the imbalance between aggressive factors (such as oxidative stress and release of proteases including matrix metallo-proteinases, neutrophil elastase, cathepsins) and protective factors (such as anti-proteases, including AAT).

The pathological changes that occur in the lung are widespread and affect both small and large airways. Structural changes occur, including destruction and enlargement of the alveoli, reducing the surface area for gaseous exchange. Smooth muscle in the walls of the airways becomes thickened and over time the response to chronic inflammation sees the development of scarring, fibrosis and less elastic airways.

It is important to note that the structural changes are however not limited to the airways, they may also affect the pulmonary blood vessels and in late stage COPD this may lead to secondary pulmonary hypertension and right sided heart failure.

**Signs, Symptoms and Diagnosis**

In the early stages, COPD is often asymptomatic. However when it does present clinically, the symptoms that are typically reported are breathlessness, a chronic cough and sputum production.

Whilst the disease is progressive and the standard timeline is for gradual worsening of signs and symptoms, acute exacerbations are a common feature of the disease. These exacerbations are usually characterised by a more rapid deterioration in breathlessness and changes in sputum, which may be in terms of volume, purulence or colour. Some people with the disease may present to their health practitioner for the first time during an exacerbation.
COPD is just one of many diseases that gives rise to these symptoms and so further information is required to confirm the diagnosis. In particular, there is a clear need to differentiate COPD from asthma. Whilst the onset of asthma is typically in younger patients than those with COPD, asthma may present later in life, in some cases this may be a representation after a period of disease quiescence that began in adolescence or early adulthood. Several aspects of the presenting feature help with this differentiation and these are summarised in Table 1.

**Table 1. Comparison of the typical presenting features of COPD and Asthma**
(Adapted from reference 5)

<table>
<thead>
<tr>
<th>Feature</th>
<th>COPD</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking (or ex-smoker)</td>
<td>Nearly all</td>
<td>Possibly</td>
</tr>
<tr>
<td>Age &lt;40 years at onset of symptoms</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Chronic productive cough</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Breathlessness</td>
<td>Persistent and progressive</td>
<td>Variable</td>
</tr>
<tr>
<td>Night-time waking with breathlessness/wheeze</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>Significant diurnal/day to day symptom variability</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>Atopy</td>
<td>Possibly</td>
<td>Common</td>
</tr>
</tbody>
</table>

Whilst the features in Table 1 assist clinicians, spirometry is widely regarded as a gold standard tool to help clarify the diagnosis. One of the most significant features that helps to differentiate COPD from asthma is the degree of reversibility of airways obstruction. The obstruction in asthma is typically reversible, whereas that in COPD is not, or at least is significantly less so. It is therefore essential that spirometry with reversibility testing is done to ensure an appropriate diagnosis is made.

Airflow limitation is considered not fully-reversible (and therefore consistent with COPD) when, after administration of bronchodilator medication, the ratio of forced expiratory volume in 1 second (FEV₁) to forced vital capacity (FVC) is <70% and the FEV₁ is <80% of the predicted value.⁶

There are however cases where an equivocal result may be obtained, in some cases repeating the test may be beneficial, however in other cases these findings may suggest the co-existence of asthma with COPD.
The severity of COPD is classified based on the level of FEV$_1$ as summarised in Table 2.

**Table 2. Severity classification of COPD** (Adapted from reference 7)

<table>
<thead>
<tr>
<th>Severity</th>
<th>FEV$_1$</th>
<th>Typical symptoms and functional limitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>60-80% of predicted</td>
<td>Few symptoms&lt;br&gt;Little effect on daily activity&lt;br&gt;Breathless on moderate exertion</td>
</tr>
<tr>
<td>Moderate</td>
<td>40-59% of predicted</td>
<td>Cough and sputum&lt;br&gt;Increasing limitation of daily activity&lt;br&gt;Breathless walking on level ground</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt; 40% of predicted</td>
<td>Chronic cough&lt;br&gt;Severe limitation of daily activity&lt;br&gt;Breathless on minimal exertion</td>
</tr>
</tbody>
</table>

In the past asthma has tended to receive more attention than COPD, however with growing recognition that early diagnosis and appropriate management of COPD can make a difference to those with the condition, it is now becoming a priority health area.

**Management**

The Thoracic Society of Australia and New Zealand and the Australian Lung Foundation have developed management guidelines for COPD, generally referred to as the COPD-X plan. These comprehensive guidelines support health professionals in optimising delivery of services and care to those with COPD.

**Smoking cessation**

Smoking cessation is the single most important intervention for people with COPD, as it is associated with slowing the rate of lung function decline. This is seen irrespective of age, thus supporting the adage that it is ‘never too late to quit’, for example:

- a smoker aged 45 years who is susceptible to COPD would typically present with disabling COPD symptoms at around the age of 65 if they continued to smoke. However by quitting at the age of 45, they can expect to gain 20 years of life without disabling symptoms
- a smoker who presents with disabling symptoms aged 65 years would typically have a life expectancy of around 5 years if they continued to smoke. If they were to quit at 65, they can expect to gain between 5 and 10 years of life.

The benefits of smoking cessation are of course not limited to COPD and those who quit also stand to gain health benefits in terms of a lower risk of other diseases commonly affecting smokers such as cardiovascular disease and a range of cancers.

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Brief interventions by health professionals, such as simply asking about smoking status during consultations, can have beneficial effects in terms of quit attempts and pharmacists are ideally placed to deliver these and provide information on the benefits of smoking cessation. Smokers who make the decision to quit should be signposted to the range of support services available such as Quitline. Simple non-pharmacological strategies can be important and include avoidance of environments that encourage smoking, quitting with a partner and motivational support. Pharmacological options typically double the chance of a successful quit attempt and include nicotine replacement therapy, varenicline, bupropion or nortriptyline. Whilst there is evidence supporting the use of nortriptyline, it should be noted that smoking cessation is outside of TGA approved indications and that it may increase the risk of adverse effects from inhaled antimuscarinics where they are used in COPD.

**Chronic therapy for stable disease**

A stepped approach is recommended for chronic therapy of stable COPD. In one regard this stepped approach is similar to that employed for asthma, in that patients should be commenced on therapy at a level appropriate to the severity of their disease. However, whereas in asthma treatment is stepped-up and stepped-down, in COPD the more persistent and progressive nature of the condition dictates that therapy is usually only stepped-up, once a patient is stable on treatment it is generally maintained at that intensity.

The stepwise approach to managing stable COPD is summarised clearly in an excellent document readily accessible on the Australian Lung Foundation website.

Short-acting inhaled bronchodilators fulfil a valuable role at all severity levels in COPD. For those patients who present with mild COPD, these agents may be all that is required initially. Whereas for those with more severe forms of the condition, they may be part of a more complex treatment regimen. The choice lies between a short-acting beta-agonist (SABA, e.g. salbutamol or terbutaline) or a short-acting antimuscarinic (SAMA, e.g. ipratropium) and decisions regarding which to use are likely to be based on a variety of factors, including adverse effects. Whilst these are not usually a major problem when inhaled therapy is used at standard doses, some patients may find adverse effects bothersome and this may influence the option chosen. For example a dry mouth and throat irritation with ipratropium; or tremor, tachycardia and agitation with a beta-agonist. Overall however, there are several advantages of SABAs, namely a faster onset of action (typically 5 minutes rather than 15 minutes), options regarding inhaler devices for delivery and the fact that a SABA can be continued in combination with any of the other classes of agent that may be added as the disease progresses. These short-acting agents are typically prescribed for use on demand and although they may be used more regularly (and in some cases SABA and SAMA together) in those with more significant symptoms, the advent of the long-acting bronchodilators has tended to reduce the need for this. SABAs and SAMAs may also be used at high doses during exacerbations.

Inhaled long-acting bronchodilators now play a fundamental role in the management of all but the mildest forms of COPD and once again the choice lies between a beta-agonist and antimuscarinic. Both the long-acting beta-agonists (LABAs, namely eformoterol, salmeterol and indacaterol) and long-acting antimuscarinic (LAMA, tiotropium) have been shown to have benefits in terms of symptoms, as well as reducing exacerbations and hospitalisations. Many of the same factors influencing the choice parallel those that apply to the short-acting agents. With the recent launch of indacaterol, there are now both LABA and LAMA options that can be given in a single daily dose. Furthermore, in terms of the relative clinical effectiveness of LABA and LAMA therapy in COPD, a recent Cochrane review concluded that the evidence is equivocal regarding improvements in quality of life. Both LABAs and LAMAs have been subject to some concerns regarding their
safety, in particular cardiovascular safety, however the current evidence provides reassurance in this regard for both drug groups.\textsuperscript{10,11} It is important to recognise that in contrast to the situation with asthma, a LABA can be used with or without an inhaled corticosteroid (ICS) in COPD. Also, whilst short and long-acting beta-agonists may be used together, the same does not apply with the antimuscarinics. Ipratropium and tiotropium should not be combined due to concerns about additive antimuscarinic adverse effects, which may include problems such as urinary retention.

The inhaled corticosteroids (ICS) are the other class of inhaled therapy used in COPD. Traditionally it was considered that the neutrophilic inflammation typical of COPD meant these agents were of limited value, however in recent years the role of these drugs has been more clearly defined. They are now considered potentially valuable, added in to existing therapy for those people with a FEV\textsubscript{1} <50% of predicted who have had 2 or more exacerbations in a year; i.e. those with more severe and unstable disease. The typical ICS adverse effects such oral soreness, candidiasis and hoarse voice are particularly common amongst those with COPD who are also using an antimuscarinic. It is noteworthy that the trials which showed benefit from ICS used doses at the high end of the dose range and this is reflected in both the regulatory approval and PBS listing. This is an important point with regard to the potential adverse consequences of ICS therapy in COPD.

Firstly, the TORCH trial which showed benefits for ICS in COPD, also reported that those patients who received ICS (fluticasone 500mcg twice daily) had an increased risk of pneumonia.\textsuperscript{12} Secondly a study published in 2010 reported a 34% increase in the risk of diabetes amongst those using ICS, a greater progression from oral hypoglycaemic therapy to insulin and these problems were particularly apparent when high doses of ICS (e.g. ≥1000 mcg fluticasone/day) were used.\textsuperscript{13} Thirdly, whilst the data remains contradictory, there is some evidence that there may be an association between high dose ICS and fracture risk.\textsuperscript{14} These adverse effects have understandably led some to suggest that the dose of ICS used in COPD should be reduced to the lowest effective dose, mirroring the approach in asthma. However it should be acknowledged that we cannot be sure that the beneficial effects reported by the trials will be maintained with this approach. Irrespective of uncertainties regarding the dose-response relationship for ICS in COPD, there is a clear need to evaluate the effectiveness of therapy and to only continue ICS where there is a sound rationale to do so, in order to ensure a favourable balance of benefit and risk.
### Reviewing Therapy

#### Inhaler technique

- **Evidence suggests that 59% of people with COPD make critical errors with their inhaler technique**\(^{15}\)
- **Many people with COPD will be using a range of different inhaler devices and there may be little opportunity to standardise these due to the formulations available**
- **Opportunities for review and education regarding inhaler technique should be acted upon, particularly when control is sub-optimal and consideration is being given to intensifying therapy**
- **Patients using MDIs should be encouraged to use a spacer to optimise delivery of inhaled therapy and be educated around spacer care**

#### Effectiveness of therapy

- **Lung function tests have a limited role in evaluating effectiveness of treatments in COPD**
- **Patient (and carer) reports of the effects on symptoms e.g. cough, and functional status e.g. exercise tolerance; should be a major influence on treatment decisions made jointly between clinicians and patients**
- **Frequency and volume of short-acting bronchodilator use may serve a valuable purpose as a proxy for effectiveness of other therapies**

### Other chronic therapies

Recent years have seen a steady decline in the use of theophylline. As improvements have been made in inhaled therapies, the problems of using a drug with a narrow therapeutic range and numerous drug interactions have made it a less attractive choice. Nonetheless, it remains an option for those who remain symptomatic despite optimised inhaled therapy. Of particular interest is the contemporary evidence that suggests low dose theophylline (100mg twice daily) may be beneficial, a regimen which would be expected to reduce the risk of toxic effects.\(^{16}\)

After many years in development, the phosphodiesterase-4 inhibitor roflumilast (*Daxas*) has now reached the Australian market and whilst there is some evidence that it may have benefits on lung function tests and exacerbations, the strength of this evidence is modest. At the time of writing, it is not listed on the Pharmaceutical Benefits Scheme.

Given the problems of excessive mucus production in those with COPD, it is not surprising that mucolytics have been evaluated as part of COPD therapy. Whilst there is some evidence that mucolytics may be effective in reducing exacerbations,\(^ {17}\) the agents studied are not marketed in Australia and it is unknown if the same effects will be provided by mucolytics that are available such as bromhexine.

Long-term oxygen therapy plays an important role for those with severe COPD complicated by hypoxaemia. There is evidence of benefit for reducing mortality where oxygen is used for > 15 hours per day, which is most practically delivered overnight. Patients who continue to smoke should be clearly informed of the fire risks associated with this at the time of arranging oxygen therapy.
Managing CVD in COPD

Whilst beta-blockers have fallen from favour for the management of uncomplicated hypertension, they remain key therapies for many patients with ischaemic heart disease, heart failure and certain tachyarrhythmias.

Whereas in asthma the hyper-responsiveness of the airways usually means that beta-blockers remain inappropriate, the situation is somewhat different in COPD. Indeed several studies have now shown that beta-blockers do not necessarily make COPD worse and they may actually be beneficial. For those people with COPD who stand to gain from a beta-blocker e.g. in the post-myocardial infarction setting, a cautious trial of a cardioselective agent may be entirely justified. In some cases it is possible that there may be a detrimental effect and in such situations the beta-blocker should be withdrawn, but the days of routinely avoiding these potentially beneficial agents in those with COPD should now be behind us.

Whilst systemic corticosteroids play an important role in managing acute exacerbations of COPD, they should not be considered for routine use in chronic therapy due to the unfavourable balance of risk and benefit.

People with COPD are in a high-risk category and should be prioritised for pneumococcal and annual influenza vaccination in line with the immunisation handbook.

Nutritional status is increasingly recognised as playing an important role and some patients may benefit from dietetic assessment and advice on nutritional support.

Many people will have a range of co-morbidities alongside their COPD. Some of these may be pre-date the airways disease and be wholly unrelated to it. Nonetheless, there are some comorbidities that are especially common amongst those with COPD, for example cardiovascular disease. It has been estimated that around 40% of people with COPD have heart disease, an overlap partly related to the two conditions sharing smoking as a major risk factor. Others include depression, anxiety; and osteoporosis, the baseline risk of which is increased in those with COPD and then magnified by exposure to steroid therapy.

It is therefore important that people with COPD are managed holistically and ongoing care involves screening for comorbidities and implementation of management strategies where appropriate.

Exacerbation therapy

The management of acute exacerbations of COPD may occur in the community or in hospital depending on the severity. Patients experiencing exacerbations will usually report deterioration in their symptoms, particularly cough, breathlessness and sputum changes; they may also have fever.

Early commencement of treatment for exacerbations is associated with better outcomes. In line with this, patients may have a COPD action plan which guides self-directed modification of treatment.

The first step is usually to administer short-acting bronchodilator therapy. It is generally considered that delivery of these via an MDI with spacer device can be just as effective as nebulised therapy, consequently this route is preferred. However, nebulised therapy may be an option in some cases where the MDI plus spacer option is not appropriate. A SABA is usually preferred due to the faster onset of action, but a SAMA may be used, as can a combination of the two if necessary. The doses that are required to provide relief in an acute exacerbation are typically higher than the standard ‘reliever’ dose, for example 8 to 10 actuations of a...
SABA MDI can be required and repeated doses may need to be given. With these high doses adverse effects such as tachycardia, tremor, anxiety and hypokalaemia are more likely to occur, but these risks need to be balanced against the requirement for timely bronchodilation.

Another key intervention is to provide a course of systemic corticosteroids, as these have been shown to reduce the duration of exacerbations. For the majority of patients, these can be given orally, typically prednisolone 30 to 50mg daily for 7 to 14 days is used. With courses of this length there is no need for tapering and where possible the dose should be given as a single daily dose in the morning to more closely mimic physiological cortisol secretion peaks and to minimise sleep disturbance. In more severe cases, initial therapy may be with parenteral agents e.g. hydrocortisone 100mg IV, with a switch to oral prednisolone being made once the patient is starting to improve and/or able to tolerate oral therapy.

Between 60 and 80% of acute exacerbations are due to infection and whilst many of these have a viral cause the standard bacterial pathogens affecting the respiratory tract e.g. *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis*, are often implicated. It is important to recognise that the isolation of bacteria from any sputum samples provided does not necessarily reflect overt infection, many people with COPD are chronically colonised with these organisms. Nonetheless, antibacterial therapy has a role to play where there are indications of infection, such as worsening breathlessness, increased sputum volume and viscosity, leucocytosis and fever. The currently recommended Australian treatment options are a 5 day course of either amoxycillin 500mg tds or doxycycline 100mg bd. Whilst a five day course is standard, the duration of antibiotic treatment may be guided by the response in a previous exacerbation, and in some cases a 7 or 10 day course may justified. In severe cases of AECOPD, it is recommended to use the same antimicrobial agents that are advised for community acquired pneumonia.

Indiscriminate use of oxygen is inappropriate, but where there is hypoxaemia, oxygen therapy (28%, or 0.5-2 litres per minute) is indicated. Non-invasive positive pressure ventilation is effective where there is acute hypercapnic ventilatory failure.

There is increasing interest in the role of pulmonary rehabilitation for those with COPD. The aims of this are to improve functional status and quality of life and also to facilitate early discharge from hospital where appropriate.

The occurrence of an exacerbation should also be seen as an opportunity to re-evaluate management strategies, such as putting a COPD action plan into place, optimising chronic therapy and revisiting the issue of smoking cessation where appropriate.

**Summary**

COPD is a common progressive condition that relies heavily on pharmacotherapy in both the acute and chronic phases and pharmacists have a key role to play in optimising patient care, through areas such as disease state education, smoking cessation advice, medication counselling and review.
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MCQs

Questions based on this article:

Select ONE alternative that best represents the correct answer to each of the following multiple choice questions (only one answer per question is correct)

Q1. Which ONE of the following is understood to be the main reason why the response to inhaled corticosteroids is less in COPD than it is in asthma?
   a) Neutrophil mediated inflammation dominates in COPD and is less ICS responsive
   b) The benefits of ICS are offset by the concurrent use of inhaled antimuscarinics
   c) High volumes of mucus secretion in COPD prevent the ICS from reaching its target
   d) People with COPD lack the enzyme required to activate ICS in-vivo
   e) CYP induction from tobacco smoking accelerates metabolism of ICS

Q2. Which ONE of the following inhaled drug combinations is NOT appropriate at any severity level of COPD? (SABA = short-acting beta-agonist; SAMA = short-acting muscarinic-antagonist, LABA = long-acting beta-agonist, LAMA = long-acting muscarinic-antagonist, ICS = inhaled corticosteroid)
   a) SABA plus LABA plus LAMA
   b) SAMA plus LABA plus ICS
   c) SAMA plus LAMA plus ICS
   d) SABA plus LABA plus ICS
   e) SABA plus LAMA plus LABA plus ICS

Q3. Bob is a 68 year old ex-miner with moderately severe COPD, currently managed with salbutamol MDI 100mcg 2 puffs when required, Salmeterol/fluticasone Accuhaler 500/50 1 puff twice daily and tiotropium Handihaler 18mcg once daily. Which ONE of the following would be the LEAST appropriate to periodically assess related to his COPD management?
   a) Bone mineral density scan
   b) Full lipid profile
   c) Respiratory symptom assessment
   d) Depression screening
   e) Fasting blood glucose
Q4. What proportion of people with COPD make significant errors when using their inhalers?
   a) Around 1 in 100
   b) Around 1 in 10
   c) Around 3 in 10
   d) Around 6 in 10
   e) Around 9 in 10

Q5. Mavis is a 76 year old woman with COPD who is diagnosed with her second acute infective exacerbation this year. Mavis’ medical history is significant for dyslipidaemia and atrial fibrillation, she has an allergy to penicillin and her drug profile shows the following:
   - Terbutaline DPI 500mcg 2 puffs PRN
   - Eformoterol/budesonide DPI 400/12mcg 2 puffs BD
   - Atorvastatin 40mg daily
   - Warfarin 3mg/4mg alternate days
   - Verapamil CR 160mg daily

Which ONE of the following is the MOST appropriate combination of antibiotic and steroid to treat her exacerbation with?
   a) Cephalexin 500mg TDS and prednisolone 25mg TDS, both for 5 days
   b) Clarithromycin 250mg BD and prednisone 10mg daily, both for 7 days
   c) Amoxycillin 250mg TDS and prednisolone 30mg daily, both for 14 days
   d) Moxifloxacin 400mg OD and prednisone 75mg daily, both for 5 days
   e) Doxycycline 100mg BD and prednisolone 50mg daily, both for 7 days