

# COPD Challenge

Chronic obstructive pulmonary disease (COPD) exacerbations may make up more than 10% of acute medical admissions [1], and they are increasingly recognised as a cause of significant morbidity and mortality for sufferers. Indeed, 90-day mortality may be as high as 15% [2]. The aim of the following case histories is to provide clinicians with an understanding of COPD exacerbations and their management.

L. Willis

Royal Brompton Hospital  
Sydney Street  
London  
SW3 6NP  
UK  
E-mail: l.willis@  
rbht.nhs.uk



## Case 1

A 57-year-old male has arrived at the accident and emergency department with a letter from his general practitioner (GP), which reads as follows:

*Thank you for seeing Mr A. He has recently undergone spirometry by our practice nurse, which showed a forced expiratory volume in one second (FEV<sub>1</sub>)/forced vital capacity ratio of 60%. His exercise tolerance is normally 800 m on flat ground. However, he now becomes breathless walking to the bathroom and is productive of green phlegm. He feels that his salbutamol inhaler is no longer of benefit. I think he may require admission and would be grateful for your assessment.*

### Task 1

Based on the above information, which of the following statements is true?

1. The patient definitely has a diagnosis of COPD and should be admitted for treatment immediately.
2. The patient has mild COPD and an exacerbation that may be suitable for management at home.
3. The patient has airflow obstruction and symptoms suggestive of acute deterioration. Further information and assessment are required to confirm a diagnosis of COPD and determine the need for admission.
4. The patient has features of acute severe asthma and the intensive treatment unit (ITU) should be involved in his management.

**Answer 1**

This answer is incorrect. A reasonable definition for COPD includes airflow obstruction with minimal (<15%) reversibility to bronchodilators. Other features include minimal day-to-day variability in symptoms and progressive decline. In total, 90–95% of cases in the developed world are smoking-related. The minority that are not smoking-related may be related to environmental exposures (such as fumes from cooking fires) or occupational exposure to dusts.

Although it is likely that this patient has COPD, ideally further information should be obtained with regard to smoking history, previous airways disease (if any) and reversibility before a diagnosis is given.

Many exacerbations of COPD can be successfully treated at home, provided the patient is not too unwell and has adequate community support [3].

**Answer 2**

This answer is incorrect, for the diagnostic reasons above and others.

The severity of COPD can be defined by the degree of impairment of FEV<sub>1</sub> compared with that predicted for the patient's age and height. Mild COPD is defined by an FEV<sub>1</sub> of 50–80% predicted, moderate COPD by an FEV<sub>1</sub> of 30–50% pred and severe COPD by an FEV<sub>1</sub> <30% pred. FEV<sub>1</sub> does correlate with mortality in COPD, but not always with the degree of disability. In the example given, even once the diagnosis of COPD was confirmed, it would not be possible to comment on the severity without a figure for the FEV<sub>1</sub>.

The definition of an exacerbation of COPD varies, but a worsening of usual symptoms – which may be associated with a change in sputum colour and/or volume and requires healthcare intervention and a change in management – would be a reasonable starting place. If, with additional information, the patient is felt to have COPD, he also has symptoms suggestive of an exacerbation. There is growing evidence to suggest that he may be managed at home with appropriate support [3].

**Answer 3**

This is the correct answer. The patient's spirometry results confirm airflow obstruction, although further history is required to establish a diagnosis of COPD (see answers 1 and 2).

The severity of COPD can be defined by the degree of impairment of FEV<sub>1</sub> compared with that predicted for the patient's age and height (see answers 1 and 2).

Using the above criteria defining an exacerbation of COPD as a worsening of usual symptoms, the patient in the example has an exacerbation of his underlying airways disease.

There is now evidence to support the treatment of COPD exacerbations at home, provided the patient is sufficiently stable and there is adequate community support [3].

**Answer 4**

This answer is incorrect. There is nothing in the letter to suggest features of acute severe asthma.

While the ITU may be appropriate, again there is insufficient information with which to make that assessment. Consultation with the patient in order to take a history and perform an examination is required.



## Case 2

As the trainee respiratory physician on call, a 69-year-old male has been referred to you by the accident and emergency department. He has been diagnosed with COPD by his GP, and is prescribed salmeterol and salbutamol inhalers. He is now complaining of increased shortness of breath, pleuritic chest pain and a cough productive of yellow phlegm. While he can normally walk to the shops, he is now breathless on crossing the room. His arterial oxygen saturation ( $S_{a,O_2}$ ) is 90% on room air. Blood tests (results pending) and chest radiography have been performed, but the patient has been moved to the medical assessment unit without further investigation owing to pressure on accident and emergency beds.

### Task 1

Which of the following options best describes your next step?

1. Ask the emergency department to assess the patient fully and initiate treatment.
2. Review the patient's history, perform an examination and look at the chest radiography before deciding what further investigations and treatment may be necessary.
3. Ask for an electrocardiogram (ECG) and arterial blood gases (ABGs), then admit the patient directly to the respiratory ward without further review.
4. Ask the nursing staff to administer high-flow oxygen immediately.

**Answer 1**

This answer is incorrect. While ideally all initial investigations and treatment should have been initiated in accident and emergency, the patient's care has now been transferred to the medical team. As such, responsibility for the patient's care is now yours.

**Answer 2**

This is the correct answer. Initial triage has been carried out by Accident and Emergency, but further history and examination are required to determine the cause of this patient's deterioration. Points in the history and examination should help to guide decisions with regard to further investigation and required treatment.

**Answer 3**

This answer is incorrect. While ABCs and ECG are likely to be a part of further investigations, an assessment to ascertain the patient's clinical status, and the priority of such tests *versus* immediate treatment, is needed. Direct admission to the respiratory ward without further assessment may further delay the diagnosis of any potentially life-threatening complications.

**Answer 4**

This answer is incorrect. Although  $S_{a,O_2}$  of 90% on room air is of concern, in the context of known COPD, rapid further assessment of the patient, particularly with respect to how well he is tolerating hypoxia (this may be normal for him) and any features of  $CO_2$  retention should be undertaken before requesting uncontrolled oxygen therapy.

**Task 2**

The results of investigations are now available. The patient's chest radiography is on the right. What does it show?

1. Normal chest radiography.
2. Hyperexpansion, flattened diaphragms but otherwise normal.
3. Right lower lobe pneumonia on a background of changes consistent with COPD.
4. Hyperexpansion, flattened diaphragms and prominent pulmonary arteries.



**Answer 1**

This answer is incorrect. While there are no focal lung field abnormalities, the chest radiograph is not completely normal. It shows evidence of hyperexpansion and flattening of the diaphragms, both of which are consistent with COPD, although not a required feature for the diagnosis.

**Answer 2**

This is the correct answer. There is indeed hyperexpansion and flattening of the diaphragms consistent with COPD. Chest radiography may be normal in patients with COPD, particularly those with mild disease. It is important to examine the radiograph for pneumonia or pneumothorax, which are important causes of deterioration in patients with COPD. Neither of these are present on this film.



*This radiograph shows a patient with a COPD exacerbation complicated by right-sided pneumothorax. Reproduced from [4].*

**Answer 3**

This answer is incorrect. While the radiograph does show hyperexpansion and flattened diaphragms consistent with COPD, there is no evidence of lobar consolidation.

**Answer 4**

This answer is incorrect. The radiograph does show hyperexpansion and flattened diaphragms, which would be consistent with COPD. While some patients with severe disease may have large pulmonary arteries (possibly representing the development of pulmonary artery hypertension), that is not the case here.





### Case 3

A 67-year-old female has had a diagnosis of COPD for 3 years, although this is her first emergency hospital attendance. You have seen her in the Accident and Emergency Department with a 3-day history of increasing shortness of breath and cough productive of purulent phlegm. She has become increasingly short of breath in the past hour. She has no other significant comorbidities and is usually independent with an exercise tolerance of ~1 km on the flat.

On examination, following 2.5 mg nebulised salbutamol by the Accident and Emergency Department, she was short of breath at rest, but completing sentences with a respiratory rate of 28 breaths per min and using accessory muscles of respiration. Heart rate was 105 beats per min, with a blood pressure of 150/90 mmHg. Oxygen saturations were 89% on room air and auscultation of the patient's chest revealed decreased air entry throughout, with widespread expiratory wheeze. Physical examination was otherwise unremarkable.

Her bloods revealed a white cell count of 12.4 g per dL (with a neutrophilia) and a C-reactive protein level of 92. Full blood count, urea and electrolytes, and liver function tests are otherwise normal. Chest radiography shows changes consistent with COPD, and 12-lead ECG shows sinus tachycardia as the only abnormality. Blood gas analysis shows pH 7.32, carbon dioxide partial pressure ( $P_{a,CO_2}$ ) 7.9 kPa, oxygen partial pressure ( $P_{a,O_2}$ ) 6.0 kPa, bicarbonate ( $HCO_3^-$ ) level of 30 mEq per L and base excess (BE) +5 mEq per L.

#### Task 1

##### What is your immediate management plan?

1. Nebulise 2.5 mg salbutamol with the addition of 500 µg ipratropium over air, repeating the salbutamol if necessary, followed by oxygen 24% *via* venturi mask to target  $S_{a,O_2}$  of 90%, antibiotics, prednisolone and reassessment by repeat ABGs after 30 min provided there is no deterioration.
2. Nebulise 5 mg salbutamol with the addition of 500 µg ipratropium over oxygen, followed by oxygen *via* a mask with a reservoir bag to target  $S_{a,O_2}$  of 98%, *i.v.* antibiotics and hydrocortisone and admit to the medical assessment unit.
3. Nebulise 2.5 mg salbutamol with the addition of 500 µg ipratropium over air, followed by 2 L of oxygen *via* nasal specs, antibiotics and prednisolone, and send to the ward, asking the doctor to reassess in 2 hours.
4. Transfer for immediate commencement of noninvasive ventilation and request ITU review.

**Answer 1**

This is the correct answer. The first line of treatment in COPD exacerbations is nebulised bronchodilation with both  $\beta$ -agonists such as salbutamol and cholinergic agents such as ipratropium. While salbutamol can be repeated as necessary (as long as it does not precipitate a marked tachycardia) there is no benefit to giving ipratropium more frequently than every 6 hours. A 2.5 mg dose of salbutamol is as effective as 5 mg in terms of recovery of peak expiratory flow and length of hospital stay in patients with COPD exacerbations [5].

The patient's blood gases and oximetry indicate hypoxia, which should be corrected. The elevated bicarbonate and base excess at presentation to accident and emergency suggest the possibility of chronic  $\text{CO}_2$  retention, as compensation for raised  $\text{CO}_2$  with retention of bicarbonate at a renal level takes hours to develop. In the case of chronic  $\text{CO}_2$  retention, the body can become tolerant to an elevated level and may come to rely on hypoxia to drive respiration. In such circumstances, correction of the  $P_{a,O_2}$  to so-called normal levels (10–14 kPa) may result in a decrease in respiratory drive and worsening  $\text{CO}_2$  narcosis, and as such should be avoided. Use of the venturi mask system allows accurate control of the inspiratory oxygen fraction ( $F_{i,O_2}$ ) in the acute setting to target pulse oximetry readings of ~92%. This value correlates with a  $P_{a,O_2}$  of ~8 kPa [6]. The  $F_{i,O_2}$  may need to be altered in order to achieve the target saturations. For the same reason, nebulisation should take place using air rather than oxygen.

As the patient is able to complete sentences, she should be able to take oral medication, in which case there is no evidence that *i.v.* hydrocortisone confers additional benefit over oral prednisolone. The purulent phlegm and raised inflammatory markers suggest the possibility of an infective cause for this patient's deterioration, which should be treated with antibiotics.

As the patient has a respiratory acidosis, she needs close monitoring and reassessment with repeat ABGs to ensure she is responding to medical therapy.

**Answer 2**

This answer is incorrect. While nebulised bronchodilation is an important first line of treatment, this patient is hypercapnic, and the elevated  $\text{HCO}_3^-$  levels and BE at presentation suggest that this may be acute-on-chronic. Under these circumstances, the patient may be reliant upon a hypoxic drive to breathe and uncontrolled oxygen therapy, be it in the form of an oxygen-driven nebuliser or high-flow oxygen through a mask with a reservoir bag, may result in decreased respiratory drive and worsening  $\text{CO}_2$  narcosis.

As the patient is completing sentences, she is likely to be able to tolerate oral medication, in which case there is no evidence of benefit for *i.v.* hydrocortisone over oral prednisolone.

This patient also has a respiratory acidosis, therefore, a plan should be made to reassess after initial therapy in order to ensure she has responded.

**Answer 3**

This answer is incorrect. While nebulised bronchodilation is the correct first line of treatment, this patient is hypercapnic, and the elevated  $\text{HCO}_3^-$  and BE at presentation suggest that this may be acute-on-chronic. Under these circumstances the patient may be reliant upon a hypoxic drive to breathe and uncontrolled oxygen therapy may result in decreased respiratory drive and worsening  $\text{CO}_2$  narcosis. Although oxygen delivery *via* nasal specs may be more convenient for patients, it is impossible to control the  $F_{i,O_2}$  using this method, making it unsuitable in this case because of the need not to over-correct  $P_{a,O_2}$ .

In view of the patient's respiratory acidosis, reassessment with repeat ABGs is needed in order to ensure she is responding to optimal medical therapy. While she could be transferred out of the accident and emergency department, she needs reassessment within the next hour to ensure improvement. She should be seen by someone who is able to take ABGs and interpret the results correctly.

**Answer 4**

This answer is incorrect. Although the patient has a respiratory acidosis, with a pH that fits the criteria for noninvasive ventilation, she should be given optimal medical therapy before commencing noninvasive ventilation. She has only received one nebuliser from accident and emergency, so there is further medical treatment that could be given. From the information above, she is currently in single-organ failure so ITU review could be postponed pending a review of her response to optimal medical therapy.

## Part 2

Despite repeated nebulisation, prednisolone, antibiotics and controlled oxygen, the patient does not improve. When you reassess her, her respiratory rate is now 32 breaths per min, her heart rate is 120 beats per min and she is struggling to complete sentences. On 24% oxygen, her repeat ABGs show pH 7.27,  $P_{a,CO_2}$  8.9 kPa,  $P_{a,O_2}$  8.0 kPa,  $HCO_3^-$  31 mEq per L and BE 5.5 mEq per L.

### Task 2

#### What do you do now?

1. Remove the oxygen as  $CO_2$  narcosis has occurred as a result of removal of the hypoxic stimulus to respiratory drive, repeat the salbutamol nebuliser and reassess in 30 minutes.
2. Arrange for the patient to be set-up on bilevel noninvasive ventilation (NIV), consider the addition of *i.v.* aminophylline and reassess with repeat ABGs in 60 minutes, provided there is no further deterioration.
3. Increase the oxygen to 28% as the patient remains hypoxic, repeat the nebulisers, load with *i.v.* aminophylline and reassess in 2 hours.
4. Arrange for the patient to be set up on continuous positive airway pressure (CPAP), consider the addition of *i.v.* aminophylline and reassess with repeat ABGs in 60 minutes, provided there is no further deterioration.



**Answer 1**

This answer is incorrect. Although the CO<sub>2</sub> level has increased, removal of the oxygen is likely to result in unacceptable hypoxia. Below a Pa<sub>a</sub>O<sub>2</sub> of 8 kPa, oxygen delivery to the tissues is at risk of being compromised. Additionally, her increase in respiratory distress suggests that her respiratory function is becoming more compromised, which is the likely cause of her increasing CO<sub>2</sub> level. Therefore, removing the oxygen is unlikely to improve the respiratory acidosis and may worsen tissue oxygen delivery.

**Answer 2**

This is the correct answer. The patient has not improved with optimum medical therapy. A trial of *i.v.* aminophylline could be considered in addition to previous therapy; however, there is little evidence to support its use in either acute or stable COPD. It should certainly not delay organising bilevel NIV, which should be commenced as soon as possible, provided there are no contraindications. This should be undertaken in an appropriate environment. This will vary between hospitals, as will the person who physically starts the patient on the treatment. The nursing staff should be familiar with the treatment and there should be the facility for continuous monitoring of oxygen saturations as a minimum.

It is also important to consider whether NIV is the ceiling of care or whether the patient is suitable for ITU. In the absence of an advanced directive to the contrary, this patient should be considered for invasive ventilation should NIV not be successful; she was independent at home prior to admission with a reasonable exercise tolerance, and has evidence to suggest a reversible cause for her deterioration.

As with any treatment, it is important to assess its success or failure. In the case of NIV, this should be with an assessment of the patient and a repeat ABGs after ~1 hour (provided there is no further deterioration) to ensure that the respiratory acidosis is improving [7].

**Answer 3**

This answer is incorrect. Increasing the Pa<sub>a</sub>O<sub>2</sub> above 8 kPa is unlikely substantially to improve oxygen delivery to the tissues and may blunt respiratory drive, as discussed in answer 1. While *i.v.* aminophylline and nebulisers may play a role in this patient's further management, she has deteriorated despite medical therapy. She has significant respiratory acidosis which requires urgent treatment.

**Answer 4**

This answer is incorrect. A trial of *i.v.* aminophylline could be considered in addition to previous therapy; however, there is little evidence to support its use in either acute or stable COPD. It should not delay commencing the patient on noninvasive respiratory support. In the case of patients with COPD, CPAP is not the ideal mode of support. It provides only single-level support, which may decrease the work of breathing and improve oxygenation but in this situation would do little, if anything, to improve CO<sub>2</sub> elimination.

**Further reading**

American Thoracic Society, European Respiratory Society. *Standards for the diagnosis and management of patients with COPD.*

<http://www.ersnet.org/InPresentations/copd/files/main/index.html>. Date last updated: 2004. Date last accessed: May 30, 2007.

National Institute for Health and Clinical Excellence. *Chronic obstructive pulmonary disease: management of chronic obstructive pulmonary disease in adults in primary and secondary care.* [http://www.nice.org.uk/pdf/CG012\\_niceguideline.pdf](http://www.nice.org.uk/pdf/CG012_niceguideline.pdf). Date last updated: February 2004. Date last accessed: May 30, 2007.

British Thoracic Society. *BTS guidelines for the management of chronic obstructive pulmonary disease.* *Thorax* 1997; 52: Suppl. V, 1–28.

**References**

1. Pearson MG, Littler J, Davies PDO. An analysis of medical workload – evidence of patient to specialist mismatch. *J R Coll Physicians Lond* 1994; 28: 230–234.
2. Anstey K, Lowe D, Roberts CM, Hosker H. *Report of the 2003 National COPD Audit.* London, Royal College of Physicians/British Thoracic Society, 2004.
3. Ram FSF, Wedzicha JA, Wright J, Greenstone M. Hospital at home for acute exacerbations of chronic obstructive pulmonary disease. *Cochrane Database Syst Rev* 2007; 2: CD003573.
4. Vijayasaritha K, Stockley RA. Causes and management of exacerbations of COPD. *Breathe* 2007; 3: 251–263.
5. Nair S, Thomas E, Pearson SB, Henry MT. A randomized controlled trial to assess the optimal dose and effect of nebulized albuterol in acute exacerbations of COPD. *Chest* 2005; 128: 48–54.
6. Jubran A, Tobin MJ. Reliability of pulse oximetry in titrating supplemental oxygen therapy in ventilator-dependent patients. *Chest* 1990; 97: 1420–1425.
7. BTS Standards of care committee. Non-invasive ventilation in acute respiratory failure. *Thorax* 2002; 57: 192–211.