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Respiratory Medicine Volume 2 Issue 5 June 2008

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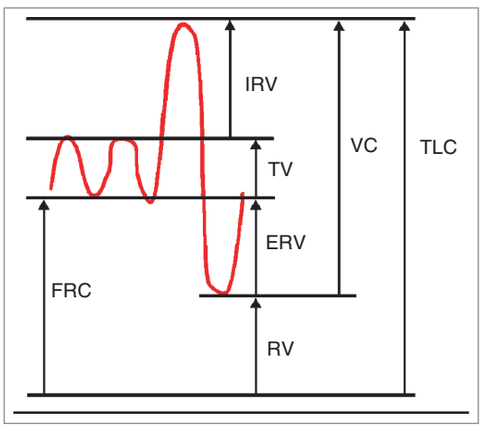
## Notice

# Spirometry for foundation doctors

Philip Ryan, Janette Humphreys and Michael Howard

The interpretation of spirometry is generally covered in the medical undergraduate curriculum, but the principles can be easily forgotten if not encountered on a regular basis. Check you understand at least the first three terms in the glossary (Box 1) and read on. Spirometry may seem complicated, but it is useful to remember that once artefact is excluded, there are only two main patterns of abnormality: obstruction and restriction.

**$FEV_1$** (Forced expiratory volume in one second) – The maximal volume of gas, which can be expired from the lungs in the first second of a forced expiration from a position of full inspiration.
**FVC** (Forced vital capacity) – The maximal volume of gas that can be expired from the lungs during a forced and complete expiration from a position of full expiration.
**PEF** (Peak expiratory flow) – The maximal flow achievable from a forced expiration with an open glottis starting from a position of full inspiration. Used to indicate the presence of airflow obstruction (variable or fixed).




**RVC** (Relaxed vital capacity) – The maximal volume of gas that can be expired from the lungs during a relaxed but complete expiration from a position of full inspiration.
**TLC** (Total lung capacity) – The volume of gas in the lungs and the airways at the position of full inspiration.
**RV** (Residual volume) – The volume of gas remaining in the lungs and airways at the position of full expiration.
**TV** (Tidal volume) – The volume of gas expired or inspired during one breathing cycle.
**FRC** (Functional residual volume) – The volume of gas in the lungs and airways at the end of a tidal expiration.
**ERV** (Expiratory reserve volume) – The maximal volume of gas, which can be expired from the position of FRC.

Box 1: Glossary.

## Spirometry during the foundation years

Foundation doctors will often come across situations where spirometry may be indicated or valuable during their training. Many patients with asthma or COPD are admitted to medical wards and have their spirometry checked prior to going home or for diagnostic purposes. In addition, spirometry is often used to monitor progress in conditions such as cystic fibrosis, and the vital capacity is often used as a bedside measurement of respiratory muscle strength in patients admitted with Guillian-Barré syndrome.

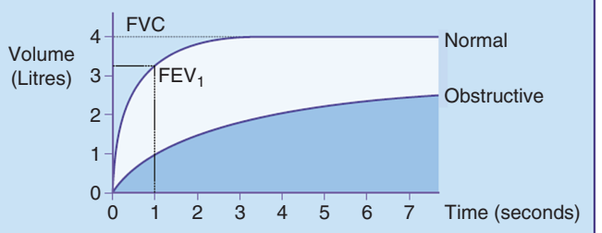
In patients undergoing surgery, spirometric measures are often used to determine whether at-risk patients are safe to undergo general anaesthetic. In general practice it is used extensively to investigate, diagnose and monitor patients with breathlessness, asthma and COPD. As you should do with all clinical data, each time you see spirometry results try and make your own diagnosis before reading the report. Also note that the automatic reports generated by some machines are often incorrect. Check that you understand $FEV_1$ and FVC described in Figure 1.

## Quality is all

It is important that every healthcare professional who undertakes spirometry is adequately trained. Poor-quality spirometry may lead to an incorrect diagnosis and treatment.

Spirometry can be measured on several types of equipment. Some of the simpler types of equipment will just provide an output in numerical terms, like $FEV_1$ and FVC. However, it is very important

**Forced expiratory volume in 1second ($FEV_1$) and forced vital capacity (FVC)**




- **Spirometry gives three important measures:**
  - **$FEV_1$:** the volume of air that the patient is able to exhale in the first second of forced expiration
  - **FVC:** the total volume of air that the patient can forcibly exhale in one breath
  - **$FEV_1$/FVC:** the ratio of $FEV_1$ to FVC expressed as a percentage
- **Spirometry can also be used to measure:**
  - **VC:** slow vital capacity
  - **$FEV_1$/VC:** the ratio of $FEV_1$ to the slow vital capacity
- Values of $FEV_1$ and FVC are expressed as a percentage of the predicted normal for a person of the same sex, age and height
- **COPD can be diagnosed only if $FEV_1$< 80% predicted and $FEV_1$/FVC <0.7 (70%)**
  The severity of the airflow obstruction in COPD is indicated by the extent of $FEV_1$ reduction
- Asthma may show the same abnormalities on spirometry as COPD - if there is diagnostic doubt spirometry following reversibility testing may be used to identify asthma

N.B. Predicted values may be lower in non-caucasians.

Figure 1: Forced expiratory volume in 1 second ($FEV_1$) and forced vital capacity (FVC).

to see the spirogram (volume against time curve) or the flow volume loop (flow rate against volume curve) to determine whether an adequate exhalation has occurred. Without seeing either or both of these graphs it is not usually possible to ascertain whether the manoeuvre has been performed properly. Thus, when trying to interpret results, the first question one needs to ask oneself is 'Has the manoeuvre has been performed correctly and is it free from artefact?'. Artefacts can occur for several reasons. Often the patient fails to take an adequate inspiration, resulting in reduced lung capacity or the patient may not start forcefully exhaling at the very beginning, leading to a slow start and an incorrect $FEV_1$. Some patients stop exhaling before they reach residual volume, again this will lead to underestimation of the vital capacity. Coughing or taking extra breaths will also lead to spurious results. Examples of these common problems are illustrated in Figure 2.

## Interpretation

Once you are satisfied that a representative good-quality exhalation has been achieved, the next thing to look at is the $FEV_1$. If this is reduced it will it indicate either obstruction or restriction. In this case, the FVC and/or the $FEV_1$/FVC ratio will determine whether the overall picture is obstruction, restriction or a mixed picture.

### Obstruction: the numbers

In pure obstruction the $FEV_1$ will be reduced without any reduction in the FVC. $FEV_1$ is essentially a measure of airway diameter, thus if there is airway obstruction, the same amount of air will be exhaled if the vital capacity is unchanged, but it will happen more slowly, i.e. less will be exhaled in the first second. In these cases the $FEV_1$/FVC ratio would be reduced. Obstruction can be localised or generalised. Obstruction is

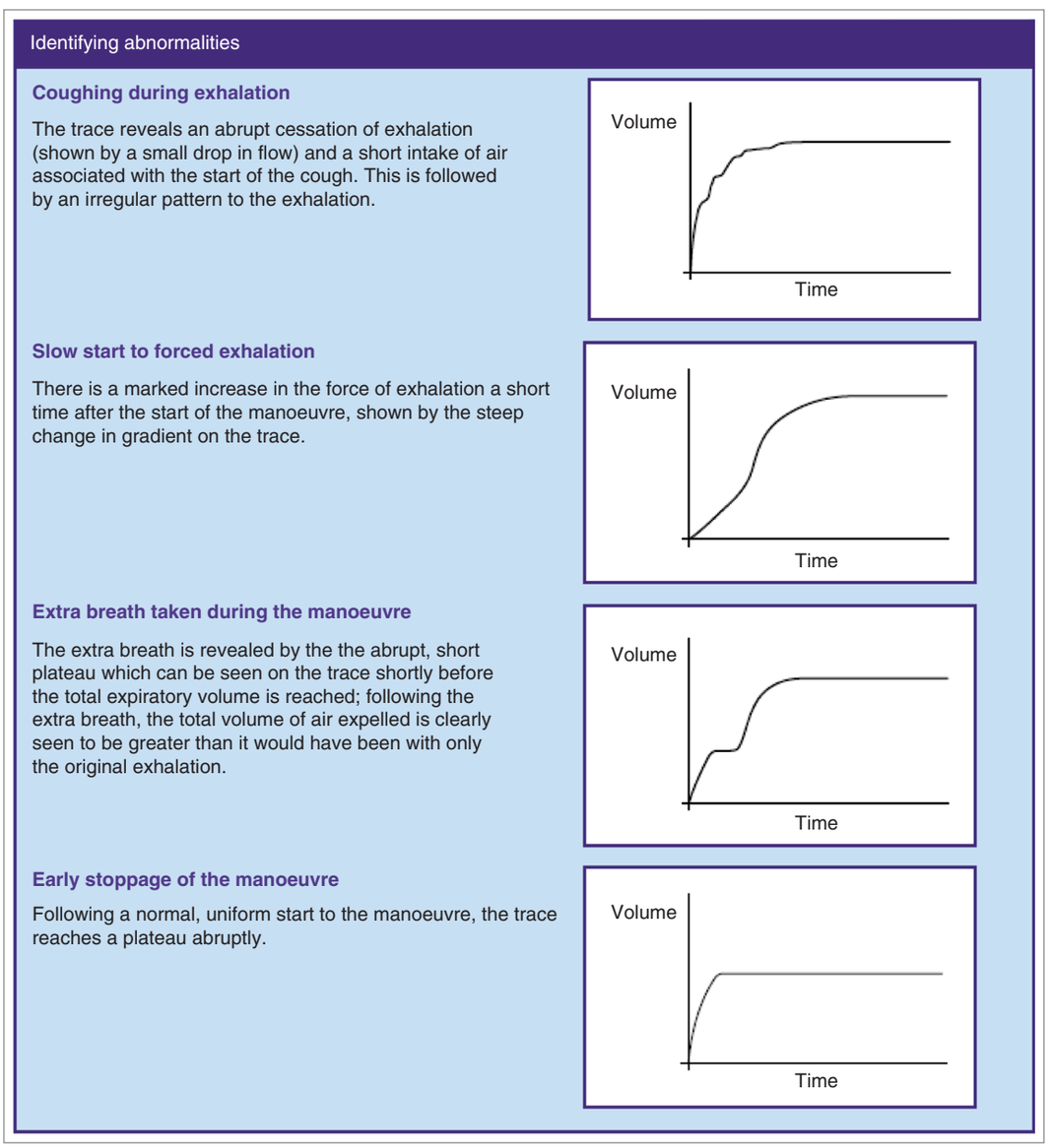



Figure 2: Identifying abnormalities.

commonly generalised and caused by asthma or COPD. Other causes of obstruction include bronchiectasis, whilst localised obstruction may be caused by goitre, upper airway tumour or foreign bodies.

### Obstruction: the volume/time graph

There will be a slow rise of the curve that will eventually reach the predicted FVC. However, in conditions such as severe COPD, the vital capacity may be reduced by lung hyperinflation and the slow rising curve may never reach the predicted FVC, as illustrated in the upper panel of Figure 3.

### Obstruction: the flow rate/volume graph

The peak expiratory flow rate is the peak on the expiratory graph and may be reduced (NB. it is often measured by spirometry in litres/sec, not litres/minute as on a peak flow meter. Therefore multiply by 60 to give a clinically recognisable result). On the expiratory limb as obstruction increases there will be increased 'scalloping' as the flow rate at lower lung volumes slows down (see upper panel Figure 4). Eventually, a 'church and steeple' pattern is seen in severe obstruction typified by severe emphysema (see middle panel Figure 4).

## Restriction: the numbers

If both the $FEV_1$ and FVC are reduced by a similar amount, it is a restrictive picture and the $FEV_1$/ FVC ratio will remain normal or be increased. In pure restriction, the airways have a normal diameter, but the vital capacity is reduced. This results in nearly all the vital capacity being exhaled within the first second and a normal or increased $FEV_1$/FVC ratio.

It is often useful to think of causes of lung restriction like the layers of an onion. Starting from the outside, restriction can be caused by a tight skin (e.g. scleroderma), obesity (the most common cause of restriction), muscle/diaphragmatic weakness, and chest wall/spinal problems (kyphoscoliosis). The next layer in is the pleura where extensive fibrosis or effusion can cause restriction. Finally, the lung parenchyma itself will cause restriction if there is significant pulmonary fibrosis.

## Restriction: the volume/time graph

There will be a normal steep rise to the curve as the airways are not obstructed. It will plateau quickly as the reduced vital capacity will be reached quickly (see lower panel Figure 3).

## Restriction- the flow rate/volume graph

As there is no airway obstruction, the flow rate will be normal with a near-normal peak flow. However, as the volume is reduced by the reduced vital capacity, the flow-volume loop will appear narrower or 'squashed' (see lower panel Figure 4).

## Mixed picture

As in many clinical situations, there may be a mixed picture. As mentioned above, severe airflow obstruction can lead to gas trapping and lung hyperinflation which itself will cause reduced vital capacity. In these cases the obstruction caused by airway narrowing is usually worse than the restriction to vital capacity caused by hyperinflation, so the $FEV_1$/FVC ratio is still reduced. Another common combination is asthma or COPD (obstruction) and obesity (restriction); the latter may be induced or aggravated by steroid treatment.

## COPD

In a patient with an appropriate history COPD is usually diagnosed when the $FEV_1$ is less than 80% of predicted and the $FEV_1$/FVC ratio is less than 70%. NICE COPD guidelines define severity by the $FEV_1$[1].

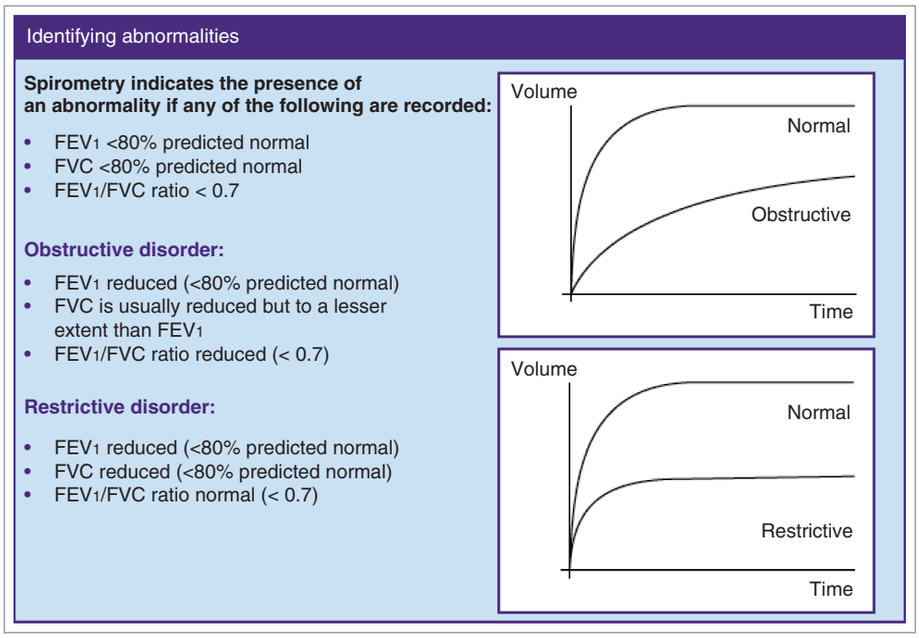



Figure 3: Identifying abnormalities.

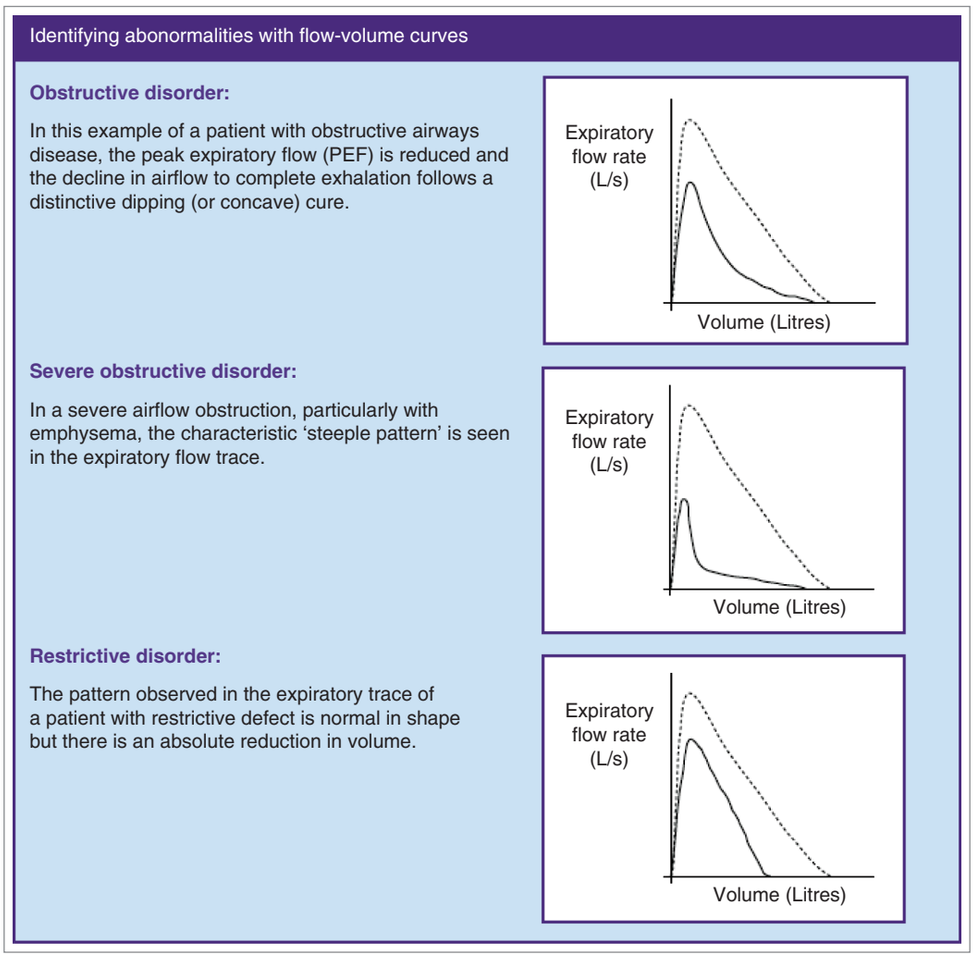



Figure 4: Identifying abnormalities with flow-volume curves.

| Severity | % predicted $FEV_1$ |
|---|---|
| Normal | >80% predicted |
| Mild | 50–80% predicted |
| Moderate | 30–49% predicted |
| Severe | <30% predicted |

## Asthma

As airway narrowing in asthma can vary throughout the day and night, a normal $FEV_1$ does not exclude asthma. However, when present, the demonstration of reversibility is an important adjunct in the diagnosis of asthma. Reversibility is usually demonstrated by undertaking spirometry before and after a nebulised bronchodilator such as salbutamol and looking for a significant increase. Different international guidelines propose different criteria for reversibility:

| Criteria for bronchodilator response |
|---|
| **BTS Asthma Guidelines[2]** |
| >200 ml + 15% increase in $FEV_1$ from baseline |
| **NICE COPD Guidelines[1]** |
| >400 ml increase in $FEV_1$ from baseline |

## Other diagnoses

Rarely patients with upper airway obstruction will present with wheeze, and are mistakenly diagnosed as having asthma. Upper airway obstruction can be acute or chronic. The acute causes of airway obstruction are usually life threatening and may need immediate medical or surgical management.

Fixed upper airway obstruction results in similar flattening of both the inspiratory and expiratory portions of the flow-volume loop (Figure 5a). Its causes include post-intubation stricture, goitre and tracheal tumours.

Variable lesions are characterised by changes in airway lesion calibre during breathing. Depending on their location (intra-thoracic or extra-thoracic), they tend to behave differently during inspiration and expiration.

This effect is exaggerated in the presence of an extra-thoracic obstructing lesion, resulting in the limitation of inspiratory flow seen as a flattening in the inspiratory limb of the flow-volume loop (Figure 5b). This is because the obstruction limits the flow rate and no further increase can occur. It therefore remains constant (i.e. the flow rate line is flat until inhalation ends). During expiration, the air is forced out of the lungs through a narrowed (but potentially expandable) extra-thoracic airway. Therefore, the maximal expiratory flow-volume curve is usually near normal. Causes of variable extra-thoracic lesions include glottic strictures, tumours and vocal-cord paralysis.

Variable intra-thoracic constrictions expand during inspiration, causing an increase in airway lumen and resulting in a normal-appearing inspiratory limb of the flow-volume loop. During expiration, compression by increasing lung pressures leads to a decrease in the size of the airway lumen at the site of intra-thoracic obstruction, producing a flattening of the expiratory limb of the flow-volume loop (Figure 5c). Causes of variable intra-thoracic lesions include malignant tumours and tracheomalacia.

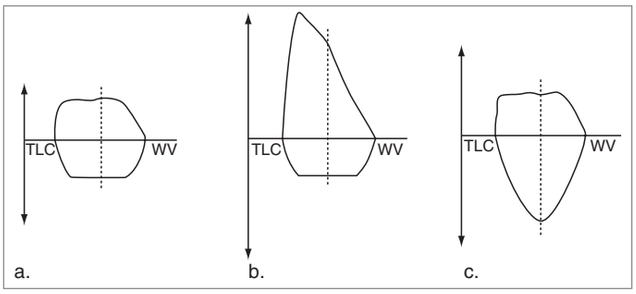



Figure 5: Flow-volume loops showing: a. fixed; b. variable extra-thoracic; and c. variable intra-thoracic upper airway obstruction (adapted from www.nationalasthma.org.au)

## Summary

Spirometry must be undertaken correctly to provide accurate results. It is used in many areas of medicine and a working knowledge of how it is undertaken and interpreted will be extremely beneficial to foundation doctors as well as other clinicians and, more importantly, the patient. Your lung function laboratory, respiratory team or respiratory nurse specialists will usually be able to help you learn more. Online resources, such as the virtual hospital (www.vh.org), enable you to practise interpretation illustrated by several examples.

## Acknowledgements

We are grateful to the COPD Consortium of the British Thoracic Society for allowing reproduction of some of their illustrations (Figures 1–4).

## References

[1] NICE. Chronic obstructive pulmonary disease. *Management of chronic obstructive pulmonary disease in adults in primary and secondary care*, 2004. www.nice.org.uk/CG012NICEguideline

[2] British Thoracic Society/Scottish Intercollegiate Guidelines Network. *British Guideline on the Management of Asthma. A national clinical guideline*, 2008.

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# Arterial blood gas interpretation and application

Jennie Gane

## Some revision

Arterial blood gas (ABG) results can contribute valuable information when making an assessment about the efficacy of respiration. They also tell us about the acid-base state of the blood.

| Respiratory failure | |
|---|---|
| Type 1 | $pO_2$ < 8 kpa on air/$O_2$ or |
| Type 2 | $pO_2$ < 8 kpa on air/$O_2$ with $pCO_2$ >6 kpa |

## Acid-base regulation

In simple terms, for enzymatic reactions to function optimally the acidity (pH) of the tissues must be kept within a constant range. Acidosis and alkalosis refer to the acid-base state of the tissues, whereas acidaemia and alkalaemia refer to the acid-base state of the blood. It is the latter we measure when taking arterial blood gases and it is important to remember it is only an approximation to what is happening in the tissues. The body regulates pH in several ways:

1. the kidneys will regulate the amount of basic $HCO_3$ being filtered into and acidic H+ secreted into the urine (over days)
2. the lungs in the short term can increase or decrease minute ventilation to blow off more or less of the acidic gas $CO_2$
3. serum proteins and haemoglobin will bind free H+.

A metabolic acidaemia is caused by loss of $HCO_3$, or an increase in H+. The pH will fall and the $HCO_3$ will be low on the ABG. The respiratory centre in the brain should respond rapidly by increasing ventilation to expire more of the acidic gas $CO_2$. A low $CO_2$ in the face of a metabolic acidaemia therefore indicates an attempt at compensation (equation below moves to the right). A metabolic alkalaemia is present if the pH is increased with a high $HCO_3$. Some respiratory compensation can occur with an increase in $CO_2$ but clearly this is to a lesser degree or we would stop breathing!

$$HCO_3^- + H^+ \leftrightarrow H_2C_3 \leftrightarrow CO_2 + H_2O$$

A respiratory acidaemia is seen if the pH decreases due to $CO_2$ retention. If the $HCO_3$ is also increased this indicates renal compensation is occurring. If a patient chronically hyperventilates blowing off $CO_2$ and becomes alkalotic the opposite compensatory process will occur. Compensatory mechanisms are successful if the pH moves back into the normal range. It is important to remember that these are essentially holding measures and if the pathological process that caused the acid-base disturbance is allowed to continue there will eventually be decompensation and the pH will drop/increase.

Another important point to note is that overcompensation of the pH does not occur. (Remember – pH is just a negative logarithmic way of expressing the concentration of H+ ions in the serum.)

## Interpreting ABG results: Advanced Life Support, Resuscitation Council UK approach

1. Is there respiratory failure? What is the inspired $O_2$ concentration ($FiO_2$)? Does this suggest an increased alveolar-arterial gradient (see later)?
2. What is the pH? (pH< 7.35 = acidaemia; pH> 7.45 = alkalaemia)
3. Look at the respiratory component i.e $CO_2$ (4.7–6 kPa is normal range)
4. Then look at the metabolic component i.e $HCO_3$ (22–26 mmol/l is normal range) or the base excess (−2 to +2 is normal. Positive values above 2 tell you that there is an excess of base and values below −2 that there is an excess of acid).
5. Combine 2, 3 and 4 to determine if the problem is respiratory, metabolic or mixed.
6. A normal pH may indicate a chronic process that is being compensated for. An abnormal pH may be an acute or acute on chronic problem. Looking at the $CO_2$ and $HCO_3$ in all cases is therefore important.

| Acid-base disturbance | pH | $pCO_2$ | $HCO_3$ |
|---|---|---|---|
| **Respiratory acidaemia** | ↓ | ↑ | ↔ |
| **Respiratory alkalaemia** | ↑ | ↓ | ↔ |
| **Respiratory acidaemia with renal compensation*** | ↔ | ↑ | ↑ |
| Respiratory alkalaemia with renal compensation* | ↔ | ↓ | ↓ |
| **Metabolic acidaemia** | ↓ | ↔ | ↓ |
| Metabolic alkalaemia | ↑ | ↔ | ↑ |
| **Metabolic acidaemia with respiratory compensation*** | ↔ | ↓ | ↓ |
| Metabolic alkalaemia with respiratory compensation* | ↔ | ↑ | ↑ |
| **Mixed acidaemia** | ↓ | ↑ | ↓ |
| Mixed alkalaemia | ↑ | ↓ | ↑ |

- The disturbances in bold are seen most commonly.
- ***Remember** – if the compensatory mechanism fails with time then the pH will move out of the normal range.

## Clinical case 1

A 47-year-old woman with depression, hypertension and irritable bowel syndrome presents with a one-day history of dyspneoa at rest, palpitations and dizziness. She has a recent history of 1 stone weight loss and vague abdominal pains. She has a respiratory rate of 26 and sats of 97% on $O_2$. Her BP is 120/85, heart sounds and chest exam are normal. She is overweight. CXR is unremarkable and her ECG shows a sinus tachycardia with T wave inversion in V1-V3. Her ABG is below:

| | |
|---|---|
| $FiO_2$ | 50% |
| pH | 7.51 |
| $pO_2$ | 10.5 |
| $pCO_2$ | 3.5 |
| $HCO_3$ | 22 |
| BE | −2.5 |
| $sO_2$ | 97% |

**What does the ABG show? What do you think is the most likely diagnosis? What investigations would you request?**

The ABG reveals a respiratory alkalaemia. It might be easy to dismiss this as being secondary to anxiety-induced hyperventilation. The key point to note here is her low $pO_2$ in light of the inspired $O_2$ ($FiO_2$) she is receiving. This is known as an increased alveolar-arterial gradient and indicates a problem with oxygenation. In view of her sudden-onset dyspneoa, normal CXR and ECG suggesting right ventricular strain, one has to consider sub-massive PE. She should have an urgent CTPA and echocardiogram.

This case highlights the importance of considering and documenting the $FiO_2$. In normal atmospheric air the partial pressure of oxygen is 21%/21 kPa. The partial pressure of $O_2$ in the blood of a non-smoking 20 year old is 12.5–13.5 kPa i.e. not all the $O_2$ in the inhaled air diffuses into the pulmonary circulation. The partial pressure decreases with age to approximately 10.8 in a 70 year old.

As inhaled $O_2$ concentration increases the $pO_2$ in the blood should also. As a rough guide you would expect the $pO_2$ to be approx 10 kPa less than the percentage of inspired $O_2$ (assuming that the patient is ventilating adequately and the $O_2$ therapy is delivered with a controlled system). In our patient we would therefore expect her $pO_2$ to be approximately 35–37 kPa. In simple terms this indicates a pulmonary disorder at some level.

Her respiratory alkalaemia may be a combination of a physiological attempt to improve oxygenation as well as secondary to anxiety and pain.

## Clinical case 2

A 60-year-old man is admitted to MAU with confusion. He has a history of heavy alcohol use and a 15 pack year smoking history. His past medical history includes chronic back pain and type 2 diabetes. Medications include prn salbutamol, codeine and metformin. On examination he is rousable to voice. Observations are as follows: BP 145/68, P 72, RR 9/min, BM 10, sats 93% on air and 97% on 35% $FiO_2$. He has some signs of chronic liver disease including ascites and ankle oedema. He is icteric. There are coarse crepitations at his right lung base. His CXR and ABG are shown below:

| | |
|---|---|
| $FiO_2$ | 35% |
| pH | 7.28 |
| $pO_2$ | 10 |
| $pCO_2$ | 6.9 |
| $HCO_3$ | 18 |
| BE | −4 |
| $sO_2$ | 96% |

**How would you describe this blood gas result? What would you do immediately?**

There is a mixed respiratory and metabolic acidaemia. There is evidence of an increased alveolar arterial gradient. It is useful to break the components down:

## The respiratory component

His $pO_2$ is lower than we would expect in spite of 35% $FiO_2$ and his $CO_2$ is high. It is important not to assume this is COPD with obliterated hypoxic drive.

## Why is the $pCO_2$ elevated?

$CO_2$ clearance is dependent on minute ventilation:

Minute ventilation = resp rate times tidal volume (l/min)

As with all unwell patients, remember to think ABC(DE). The patient's GCS is impaired (9 on calculation) and as a result there may be airway compromise. A partially obstructed airway will clearly affect your tidal volume!

It is likely he also has pneumonia (his alcohol intake and low GCS should alert you to the possibility of aspiration). Pneumonia will affect $CO_2$ clearance if the respiratory rate is increased to improve oxygenation to the point that the respiratory muscles become exhausted. This is clearly a life-threatening situation.

Most important here is D disability. He may be encephalopathic secondary to liver failure, which can repress respiratory drive. On examination you see his pupils are pinpoint. The regular codeine he has been taking for back pain has not been metabolised by his liver. You try repeated IM naloxone and find that his respiratory rate, GCS and $pCO_2$ all improve.

Normal ventilation requires:

- An unobstructed airway
- A working respiratory centre in the brain
- Phrenic nerves and diaphragm
- Intercostal nerves, neuromuscular junction and muscles
- Compliant chest wall including skin, ribs and pleura
- Compliant lungs.

## Why is the $O_2$ reduced?

Normal oxygenation depends on:

- An unobstructed airway
- Adequate ventilation
- Enough functioning alveoli
- A functional alveolar arterial basement membrane
- Pulmonary blood flow.

Clearly there are many disease processes that can affect one or more of these. The patient in case 2 has inadequate ventilation, some alveoli filled with pus and possibly some coexistent emphysema.

## The metabolic component

In this case his metabolic acidaemia may be multifactorial. Liver failure can produce an acidosis, he may have coexistent renal failure or a lactic acidosis secondary to sepsis or metformin.

## Metabolic acidosis

This is either due to loss of $HCO_3$/ingestion of H+ or ingestion/production of exogenous acid. Calculating the anion gap is useful sometimes in helping to determine the cause:

$$\text{Anion gap} = [K+] + [Na+] - [Cl-] - [HCO_3-]$$

Normal is 8–16 mmol/l. A value greater than this indicates there must be an exogenous acid present which has not been measured in the above equation. Common exogenous acids include lactate (sepsis, hypoxia, hypotension), ketones (DKA, alcohol), urate (renal failure) or drugs (salicylates, methanol).

## Common pitfalls for junior doctors

- Not taking the $FiO_2$ into account
- Attributing a raised $pCO_2$ to COPD and hypoxic drive
- Assuming the acid-base state is normal if the pH lies within the normal range.

| Glossary | |
|---|---|
| $CO_2$: carbon dioxide | K+: potassium |
| $HCO_3$: bicarbonate | Na+: sodium |
| $FiO_2$: inspired $O_2$ percentage | Cl−: chloride |
| H+: hydrogen ions | GCS: Glasgow coma scale. |

## Reference

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# Prescription of long-term oxygen therapy

J. Coates and D. Laws

***Mr Smith is a 78-year-old man admitted to hospital with an exacerbation of chronic obstructive pulmonary disease (COPD). He is known to have moderately severe COPD with a 50 pack year smoking history; and his usual treatment was Symbicort and tiotropium inhalers. The exacerbation settles with appropriate standard medical management and he is ready to be discharged. Throughout his admission he has required oxygen therapy and is still using it. He raises the question that his GP has mentioned the possibility of having oxygen at home and asks if this can be arranged.***

Long-term oxygen therapy (LTOT) is the provision of oxygen at home for patients who are chronically hypoxaemic at a rate sufficient to raise the level to >8 kPa. To be effective it should be used continuously for at least 15 hours a day, including when the patient is sleeping. It has proven survival benefit in COPD[1] and can improve quality of life in other conditions[2,3]. Once it has been started it is unlikely that it will be stopped. Patients need to be assessed to determine whether oxygen is required, and is safe and adequate for their needs.

## Is LTOT indicated in this patient?

LTOT is indicated in the following conditions where the patient suffers from hypoxaemia chronically; $PaO_2$ ≤7.3 kPa when breathing room air[4]:

- Chronic obstructive pulmonary disease
- Pulmonary vascular disease
- Severe chronic asthma
- Primary pulmonary hypertension
- Interstitial lung disease
- Pulmonary malignancy
- Cystic fibrosis
- Chronic heart failure
- Bronchiectasis

It is not indicated in patients whose arterial oxygen tension is greater than 8 kPa. If the patient has a $PaO_2$ between 7.3 and 8 kPa and has either secondary polycythaemia or clinical/echocardiographic evidence of pulmonary hypertension, then LTOT is indicated.

LTOT may be used in patients who have nocturnal hypoventilation, e.g. secondary to neuromuscular, chest wall or spinal disease, or obstructive sleep apnoea, often in conjunction with a form of non-invasive ventilation during sleep[5].

The final situation in which it is used is in the palliation of dyspnoea in patients with terminal illnesses.

## How is the patient assessed for LTOT?

The assessment process is usually performed by a multidisciplinary team led by a consultant physician with an interest in respiratory medicine.

### Clinical stability

This is defined as an absence of an exacerbation of their chronic lung disease within the last five weeks. Two arterial blood gas samples three weeks apart confirming hypoxia are usually required. In the case we describe, Mr Smith will have to wait before being assessed as he has had a recent exacerbation of his COPD. This is because his hypoxia may improve. If a patient has marked symptomatic hypoxaemia a temporary supply of supplemental home oxygen with a reassessment six weeks after discharge can be arranged.

### Optimum medical management

A clinician must confirm the patient's diagnosis, and ensure medical management has been optimised, as it may result in the patient not requiring LTOT. Smoking cessation must be confirmed. In Mr Smith's case, he has a confirmed diagnosis of COPD and is therefore potentially eligible.

### Assessment of arterial blood gases

The final step is assessment of the level of hypoxaemia using arterial blood gases. These may be in the form of a radial or femoral arterial sample or an ear lobe arteriolised sample. At least two samples are needed, the first after breathing room air and then after receiving supplemental oxygen for 30 minutes. The oxygen should be provided via nasal cannulae at an initial flow rate of 2 litres/minute or using a 24% venturi mask and should be delivered the same as the patient will receive oxygen at home[6]. This will confirm that the patient is actually hypoxaemic and that the supplemental oxygen is enough to raise the $paO_2$ above 8 kPa without causing significant hypercapnia.

## How is the oxygen prescribed?

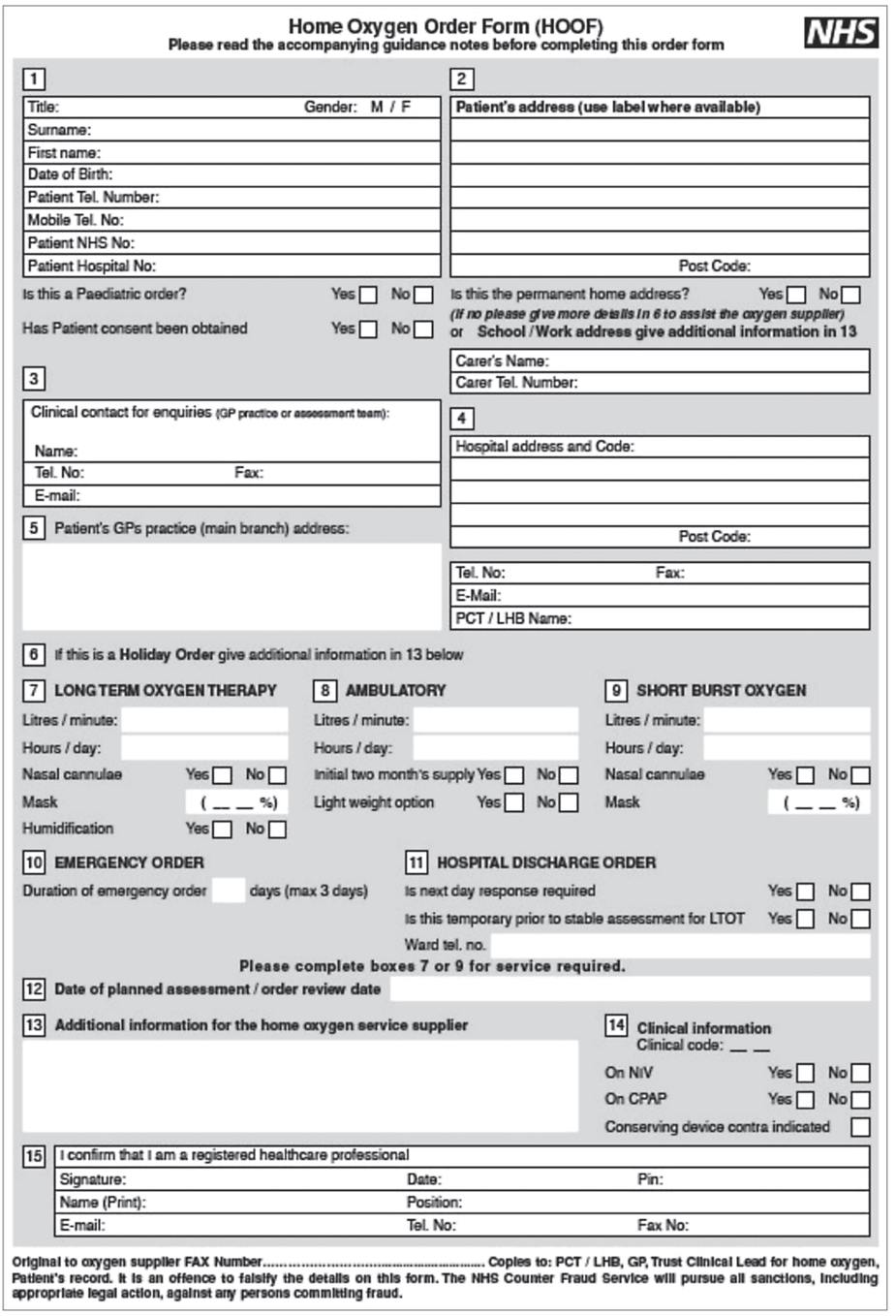
Assuming that Mr Smith is on the optimum treatment for his condition and that he is sufficiently hypoxaemic to require LTOT, the next step in the process is to fill out a home oxygen order form (HOOF) (Figure 1). This can be filled out by any appropriately trained healthcare professional. It requires the patient's and hospital's details, the clinical contact and the type of oxygen that is required. This includes the oxygen flow rate, how many hours it should be worn for and the delivery method (mask or nasal cannulae). Additional oxygen cylinders to allow the patient to use oxygen out of the house may also need to be ordered and supplied. In order to enable the patient's details to be passed onto the oxygen contractors they need to sign a home oxygen consent form (HOCF) (Figure 2).

## What happens now?

Once the oxygen concentrator company receive the HOOF and HOCF then they will install the appropriate equipment in the patient's home, which generally takes around three days. At the same time they will show the patient how to use the equipment.

The patient will also receive education from a member of the multidisciplinary team about oxygen therapy and what it means for them. Topics that are covered include:

- explanation of requirements for taking LTOT for at least 15 hours a day
- the principles of the oxygen concentrator
- safety including fire risk



**Home Oxygen Order Form (HOOF)**
**Please read the accompanying guidance notes before completing this order form**

NHS

1

Title: Gender: M / F
Surname:
First name:
Date of Birth:
Patient Tel. Number:
Mobile Tel. No:
Patient NHS No:
Patient Hospital No:

Is this a Paediatric order? Yes ☐ No ☐
Has Patient consent been obtained Yes ☐ No ☐

2

Patient's address (use label where available)
Post Code:

Is this the permanent home address? Yes ☐ No ☐
*(If no please give more details in 6 to assist the oxygen supplier)*
or School / Work address give additional information in 13

Carer's Name:
Carer Tel. Number:

3

Clinical contact for enquiries (GP practice or assessment team):
Name:
Tel. No: Fax:
E-mail:

4

Hospital address and Code:
Post Code:

Tel. No: Fax:
E-Mail:
PCT / LHB Name:

5 Patient's GPs practice (main branch) address:

6 If this is a **Holiday Order** give additional information in 13 below

7 **LONG TERM OXYGEN THERAPY**
Litres / minute:
Hours / day:
Nasal cannulae Yes ☐ No ☐
Mask ( __ __ %)
Humidification Yes ☐ No ☐

8 **AMBULATORY**
Litres / minute:
Hours / day:
Initial two month's supply Yes ☐ No ☐
Light weight option Yes ☐ No ☐

9 **SHORT BURST OXYGEN**
Litres / minute:
Hours / day:
Nasal cannulae Yes ☐ No ☐
Mask ( __ __ %)

10 **EMERGENCY ORDER**
Duration of emergency order ____ days (max 3 days)

11 **HOSPITAL DISCHARGE ORDER**
Is next day response required Yes ☐ No ☐
Is this temporary prior to stable assessment for LTOT Yes ☐ No ☐
Ward tel. no.

**Please complete boxes 7 or 9 for service required.**

12 **Date of planned assessment / order review date**

13 **Additional information for the home oxygen service supplier**

14 **Clinical information**
Clinical code: __ __
On NIV Yes ☐ No ☐
On CPAP Yes ☐ No ☐
Conserving device contra indicated ☐

15 I confirm that I am a registered healthcare professional
Signature: Date: Pin:
Name (Print): Position:
E-mail: Tel. No: Fax No:

**Original to oxygen supplier FAX Number..... Copies to: PCT / LHB, GP, Trust Clinical Lead for home oxygen, Patient's record. It is an offence to falsify the details on this form. The NHS Counter Fraud Service will pursue all sanctions, including appropriate legal action, against any persons committing fraud.**

Figure 1: Home Oxygen Order Form (HOOF).

- home servicing arrangements and electricity reimbursement
- contact numbers for nurse specialist and oxygen concentrator company.

The patient should be followed up by the respiratory specialist at three months with arterial blood gas measurements on air and on oxygen ensuring continuing LTOT requirement and adequate correction of hypoxaemia. The patient will also be seen in their home, usually by a nurse specialist, providing further education and any support that is required. Beyond this, the patient should be seen in the home every six months with measurement of oxygen saturations and then annually in the hospital for arterial samples whilst on air and supplemental oxygen.

## Other forms of domiciliary oxygen

The HOOF is also used to prescribe other forms of home oxygen e. g. ambulatory oxygen and short-burst oxygen therapy. Ambulatory oxygen is used in two cases. The first is patients that have LTOT and

**Home Oxygen Consent Form (HOCF)**
**Patient agreement to sharing information (to enable the supply of home oxygen)**

NHS

Form issued by: Unit / Surgery (Name, address and contact telephone number)

Person obtaining consent:

Print ..... Signature ..... Title .....

Patient name & HOME address:

D.O.B.: __ __ / __ __ / __ __ __ __

NHS number: _____ / _____ / _____

Patients Telephone Number: _____

I am the patient* named above / I have parental responsibility for the child* named above. My doctor or member of my care team has explained the arrangements for supplying oxygen at home. I understand these arrangements.

I understand that my doctor or member of my care team will give the Oxygen Supplier information about my diagnosis and physical condition* / the diagnosis and physical condition for my child*. This is to enable the Supplier to deliver a system, which will match the need for oxygen. I also understand that information will be exchanged between my hospital care team, my GP or home care team.

**Information:** I agree to the exchange of information between my doctor or member of my care team and the Oxygen Supplier about my* / my child's* diagnosis and physical condition. I understand that the Oxygen Supplier will keep information confidential. The Supplier will not give information to anyone else without my consent, except relevant information provided to check payments to the supplier (see below). I also agree to the exchange of information between my hospital care team, my GP or home care team.

**Access:** I also agree to give the supplier reasonable access to my home, so that the supplier can install, service and remove the oxygen system as required.

**NHS payments to the supplier:** To enable the NHS to prevent and detect any fraud or incorrectness, I consent to the disclosure of relevant information to and by the Oxygen Supplier, my doctor or member of my care team, my Primary Care Trust/Local Heath Board, Health Trust, the Prescription Pricing Authority and the NHS Counter Fraud and Security Management Service.

I understand that I may, if I wish, withdraw my consent at any time.

Patients Signature: _____ Date: _____ or,

**I confirm** that I have 'parental responsibility' for the above named child*.

Parent's Signature: _____ Date: _____

Name (PRINT): _____ Relationship to child: _____

Patient's copy (white)
Unit / Surgery copy (colour copy) for file in patients records.

Figure 2: Home Oxygen Consent Form (HOCF).

are able to leave their house and require supplemental oxygen when out of the home. The second is patients who normally aren't hypoxaemic but who desaturate when they exercise[7,8,9]. Assessment for this is by measuring oxygen saturations when the patient is performing an exercise test whilst breathing air from a cylinder and oxygen and demonstrating an improvement in exercise distance.

Short-burst oxygen therapy is the use of oxygen intermittently for periods of 10 to 20 minutes to relieve dyspnoea. This has little evidence base and is not recommended by many centres[10,11]. It may be used in certain cases with episodic breathlessness not relieved by other treatments.

## Conclusion

Mr Smith may be eligible for LTOT but will need to be formally reassessed again in five weeks. He needs to be given advice about

not smoking, seen by a consultant with an interest in respiratory medicine and will need to be followed up by a multidisciplinary team.

LTOT has a proven role in conditions with chronic hypoxaemia, producing a survival benefit and improving quality of life. Assessment is based around arterial sampling whilst the patient is in a period of clinical stability, proving that they are hypoxaemic and that this can be corrected with the prescribed oxygen. Further information is available in the BTS guidance[12].

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# A new presentation of HIV and PCP

Helen Dillon and Martin Wiselka

***A 51-year-old married man was admitted to the medical admissions unit with a four-week history of progressive shortness of breath and a non-productive cough. He also reported generalised lethargy and 2 stone weight loss over the previous six months. He had no past medical history and was a smoker of 20 pack-years.***

***On examination he was pyrexial at 38.0° C and hypoxic with $O_2$ saturation of 80% on room air. Auscultation of his chest revealed widespread fine inspiratory crepitations and mild expiratory wheeze. Other significant findings were that he was markedly cachexic (BMI = 18) and had extensive oral Candida. His admission chest x-ray showed diffuse infiltrates throughout both lung fields (Figure 1). The patient did not declare any obvious risk factors for HIV infection, but he consented to an HIV antibody test which was performed urgently and found to be positive for HIV1. Subsequent CD4 count was found to be very low at 30 cells/mm$^3$ (normal range 500–1800 cells/mm$^3$). A bronchoalveolar lavage was positive for Pneumocystis jirovecii by immunofluorescence.***

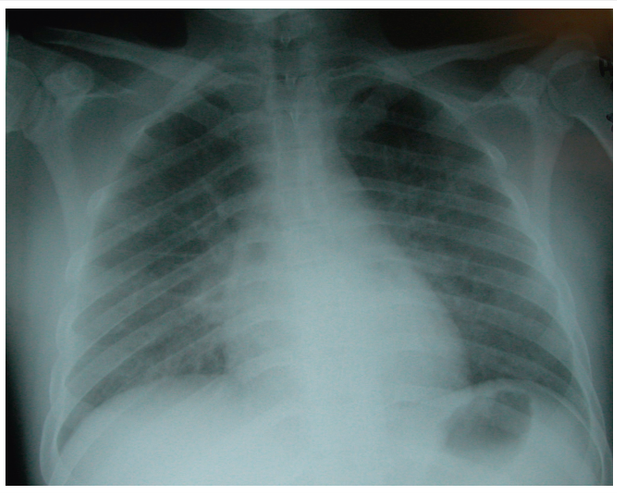

Figure 1: CXR showing perihilar interstitial shadowing associated with *Pneumocystis jirovecii* pneumonia.

## Human immunodeficiency virus (HIV)

HIV is a retrovirus that is transmitted via sexual contact, blood or mother to child. At the end of 2006 an estimated 73,000 persons of all ages were living with HIV in the UK, of whom 21,600 were unaware of their infection[1].

Two species of HIV infect humans: HIV1 and HIV2. HIV1 accounts for the majority of HIV infection worldwide and is the more virulent strain. The virus affects human immune cells, primarily helper T cells (CD4+ cells), macrophages and dendritic cells. HIV leads to a depletion of CD4+ve lymphocytes through direct viral killing and killing of infected CD4+ cells by cytotoxic lymphocytes (CD8 cells). As CD4+ cell numbers decrease, cellular immunity is lost and the body becomes susceptible to a growing number of opportunistic infections and associated malignancies.

A normal CD4+ve lymphocyte count in a healthy HIV-negative adult is >500 cells mm$^3$. As the CD4+ve cell count falls the patient may develop medical problems including reactivation of latent herpes viruses causing recurrences of herpes simplex eruptions, shingles, B-cell lymphomas (associated with EBV), or Kaposi's sarcoma (associated with HHV-8). The risk of opportunistic infection increases significantly once the CD4+ve count falls below 200 cells/mm$^3$ when patients become susceptible to a wide range of opportunistic pathogens (Box 1). In the UK acquired immunodeficiency syndrome (AIDS) is defined by the presence of a serious opportunistic infection, associated malignancy or other indicator condition (Box 1).

*Opportunistic infections*
- Bacterial pneumonia (recurrent)
- Cerebral toxoplasmosis
- Cryptococcal meningitis
- Cryptosporidiosis, chronic intestinal for longer than 1 month
- Cytomegalovirus retinitis/colitis
- Herpes simplex: chronic ulcer(s) (for more than 1 month)
- Histoplasmosis, disseminated or extrapulmonary
- Mycobacterium tuberculosis
- Mycobacterium avium complex (MAC)
- Oesophageal candidiasis
- *Pneumocystis jirovecii* pneumonia
- Progressive multifocal leukoencephalopathy
- Salmonella septicemia (recurrent/persistent)

*Malignancies*
- Cervical cancer (invasive)
- Kaposi's sarcoma
- Lymphoma: Burkitt's, immunoblastic or primary brain

*Others*
- Encephalopathy (HIV-related)
- Wasting syndrome

Box 1: Examples of AIDS-defining conditions[2].

## Pneumocystis pneumonia (PCP)

Pneumocystis pneumonia (PCP) is an opportunistic infection that affects immunosuppressed individuals and in HIV-positive patients is considered an AIDS-defining illness. The incidence has greatly decreased in developed countries with the use of highly active antiretroviral therapy (HAART) and primary PCP prophylaxis in patients with CD4+ counts below 200.

The causative organism is *Pneumocystis jirovecii* (previously known as *Pneumocystis carinii*), a unicellular fungus commonly found in the lungs of healthy individuals. Disease occurs when defects in an individual's immune system allow for uncontrolled replication of the organism in alveolar cells. As the disease progresses an important pathological feature is an increased alveolar-arterial oxygen gradient, leading to significant hypoxaemia.

The diagnosis should be considered in any patient presenting with respiratory symptoms, unexplained hypoxia or weight loss in the presence of known or suspected immune suppression.

## Assessment and history

PCP often presents insidiously and there may be few symptoms and signs other than a progressively increasing dyspnoea and dry cough. The most common symptoms at presentation are shown in Box 2.

*Symptoms*
- Progressive dyspnoea (95%)[3]
- Fever (79–100%)[3]
- Cough (95%)[3]
- Chest pain
- Weight loss
- Rigors
- Haemoptysis

*Findings on examination*
- Fever (84%)[3]
- Tachypnoea (62%)[3]
- Tachycardia
- Hypoxia – particularly exercise-induced desaturation (to <90% on room air). This may be the only clinical finding
- Crepitations and rhonchi – the most common findings on examination of the chest, but auscultation may be normal in up to 50% of patients[3]
- Other evidence of HIV infection/immune suppression, e.g. cachexia, oral candidiasis, generalised lymphadenopathy, oral/genital ulceration.

Box 2: Clinical features associated with Pneumocystis jirovecii pneumonia.

## Investigations

### Blood tests

The most common laboratory abnormality associated with PCP is an elevated LDH seen in more than 90% of HIV-infected patients with PCP. LDH levels also offer prognostic significance[6]. For all known/new cases of HIV infection a current CD4+ve lymphocyte count should be obtained.

### Imaging

The chest x-ray is often the most revealing initial investigation in PCP, although it can be normal in up to 25%[4]. The most common abnormalities are diffuse, bilateral interstitial infiltrates (Figure 1), although pneumothoraces, lobar infiltrates, cysts, nodules and effusions can also be seen[4]. HRCT has a high sensitivity for PCP and the common findings are patchy or nodular ground glass shadowing. These changes are suggestive, but not diagnostic of PCP[5].

### Microbiology

Definitive diagnosis of PCP requires identification of the organism in respiratory specimens. The most rapid and least invasive method of obtaining specimens is to induce sputum production using nebulised hypertonic saline. Immunofluorescence techniques can then be used to identify *P. jirovecii*. If an adequate induced sputum cannot be obtained, bronchoscopy and bronchoalveolar lavage (BAL) should be undertaken. This is, however, a more invasive technique and may not be feasible if the patient is significantly unwell. Nevertheless, BAL specimens will remain positive for pneumocystis in infected cases, even after several days on treatment.

### ABG

Arterial blood gases should be obtained in all patients with suspected PCP and provide a guide to management. Mild PCP is defined in those patients with $PaO_2$ >8.0 kPa on room air. Moderate/severe PCP is defined in those patients with $PaO_2$ <8.0 kPa on room air.

### Exercise testing

Testing for exercise-induced hypoxia is a simple and often useful tool. It can be done by asking the patient to walk briskly up and down the ward or clinic corridor, or perhaps up and down a flight of stairs. A subsequent fall on $O_2$ sats to less than 90% in the correct clinical context is suggestive of PCP.

## Management

Specialist advice should be sought in all cases of proven/suspected PCP. Treatment duration for HIV-positive patients should be 14–21 days with anti-pneumocystis agents. Adjunctive corticosteroids are beneficial in patients who are hypoxic with severe disease. A typical regimen is prednisolone given orally, 40 mg bd for 5/7, 40 mg od for 5/7, 20 mg od for 11/7.

Patients with mild-moderate disease may be managed as outpatients, however, this should only be done under specialist supervision.

### Anti-pneumocystis agents

The agents commonly used to treat PCP[6] are shown in Box 3. The drug of choice is high-dose co-trimoxazole (120 mg/kg daily given in 2–4 divided doses), but some patients are intolerant and develop rashes or hypersensitivity.

- Sulfamethoxazole and trimethoprim (co-trimoxazole)
- Pentamidine (intravenous)
- Atovaquone
- Clindamycin and primaquine

Box 3: Agents commonly used to treat PCP.

## Prognosis

Respiratory failure and an ARDS-like picture are important complications of PCP. Secondary pneumothorax may occur. In HIV-positive patients the mortality of acute PCP is approximately 10% and outcome is related to the presence of advanced immunosuppression at the time of diagnosis. The long-term prognosis of HIV-infected patients has improved immensely over recent years with the introduction of highly active combination anti-retroviral therapy (HAART). Most infected patients can now look forward to a relatively normal life expectancy whilst on regular medication. Initiation of HAART should only be undertaken by a specialist service and is normally deferred until patients are recovered from their acute illness.

## Post discharge: New diagnosis of HIV

Once patients are stable it is important to take a full past medical and social history including sexual history, travel history and any

past/current IV drug use, blood transfusions, or tattoos/piercing. It is vital to try to establish where a patient might have acquired their infection, as contact tracing is an important public health tool.

All patients should be referred to a specialist HIV service where their initial assessments will focus on the factors listed in Box 4.

- HIV testing: Repeat serology, CD4+ count, viral load, resistance profile
- Baseline bloods: FBC, U&E, LFT, CRP, lipid profile
- Serological testing: Hepatitis A/B/C, toxoplasma, treponemal, cryptococcal, CMV
- GUM review and full STI screen
- Other investigations which may be considered: CXR, Quantiferon/T-Spot (for latent M. tuberculosis infection), G6PD screen

Box 4: Investigations in patients newly diagnosed with HIV.

### PCP prophylaxis

Primary PCP prophylaxis should be initiated for all HIV-positive patients with CD4+ <200 cells/mm$^3$. Secondary prophylaxis should be started after an episode of PCP and continued until the CD4+ve cell count is consistently above 200 cells/mm$^3$. The most commonly used agent is oral co-trimoxazole (480 mg daily or 960 mg three times weekly). Other options are oral dapsone (if G6PD screen negative), oral atovaquone or nebulised pentamidine.

### Counselling/personal support

This should include advice regarding support groups, safe sexual practices and post-exposure prophylaxis for future partners.

## Conclusion

PCP is a potentially serious condition that should be considered in any patient presenting with respiratory symptoms, hypoxia, or weight loss in the presence of known or suspected immune suppression. If HIV is suspected, patients should be informed of the need for urgent testing. Specialist advice should be sought early in all cases of suspected/proven PCP and all new diagnoses of HIV/PCP should be appropriately referred on discharge.

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## Further reading

Health Protection Agency: www.hpa.org.uk

British Association for Sexual Health and HIV: www.bashh.org

British HIV Association: www.bhiva.org

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# Community-acquired pneumonia

K. Ostridge and T. Shaw

## Introduction

Community-acquired pneumonia is one of the most common diseases encountered both in the community and hospital settings. Timely diagnosis and appropriate management are vital in preventing the significant morbidity and mortality associated with this condition.

## Epidemiology

The annual incidence of community-acquired pneumonia is 5–11 per 1000[1],and this increases with age. Of patients with community-acquired pneumonia 22–42%[1] require hospital admission and 5–10% of these require intensive medical therapy (ITU)[2].Average mortality rates are 5.7% in the under-74 age group admitted to hospital[3], increasing to 50% if admitted to ITU.

## Aetiology

A single pathogen is identified in up to 85% of cases[1]. Many different pathogens cause community-acquired pneumonia.

| | |
|---|---|
| *Streptococcus pneumoniae* | The most common cause of pneumonia. Occurs frequently during winter epidemics. |
| *Mycoplasma pneumoniae* | Occurs in epidemics every 4–5 years. |
| *Chlamydia pneumoniae* | Epidemics occur in the community. Its direct pathogenic role is not clear as opposed to being associated with it. |
| *Haemophilus influenzae* | Commoner in patients with COPD and nursing home residents. |
| *Legionella* species | Most common during the autumn months and 52% of cases are related to travel. Epidemics occur related to water-containing systems (air conditioning units, water tanks, etc.). Risk factors include recent travel, occupations involved with water systems, recent repair to domestic plumbing systems and immunosuppression. |
| *Chlamydia psittaci* | Associated with bird contact but may have human-human spread. |
| *Staphylcoccus aureus* | Most common in the winter months and is associated with influenza. |

Box 1: Pathogens causing community-acquired pneumonia.

## Clinical features

Clinical features include:

- Fever
- Cough
- Sputum
- Dyspnoea
- Pleuritic chest pain
- Raised respiratory rate
- Tachycardia
- Localising signs on chest examination, including crackles, bronchial breathing, decreased air entry and vocal resonance.

No clinical findings can reliably diagnose community-acquired pneumonia and diagnosing it clinically without a chest radiograph is inaccurate. The presence of a normal chest examination makes an underlying diagnosis of pneumonia unlikely.

Although it is not possible to distinguish aetiological agents by clinical symptoms/signs there are certain features associated with particular agents.

| | |
|---|---|
| *Streptococcus pneumonia* | Acute onset and high fever associated with increasing age and co-morbidities. |
| *Mycoplasma pneumoniae* | Younger patients and may get haemolysis or skin/joint involvement. |
| Legionella | Younger patients, severe infection, multisystem disease (raised LFTs and CK). |

Box 2: Features associated with underlying pathogen.

## Investigations

Oxygen saturation

If these are lower than 92% on air then an arterial blood gas should be performed[4].

Chest radiograph

Consolidation is the most common sign. Other features include pleural effusions, cavitation or lymphadenopathy.

Blood tests

- WCC >20 × $10^9$/L or <4 × $10^9$/L indicate severe infection. WCC >15 × $10^9$/L indicates bacterial infection, particularly pneumococcus[5].
- U+Es, LFTs used to assess severity.
- CRP is a more sensitive marker of pneumonia than WCC and serial markers can be used to monitor response to treatment[6].

### Microbiological investigations[4]

- Blood cultures are recommended for all patients admitted to hospital.
- Sputum culture is recommended for all patients with severe community-acquired pneumonia who are not improving. Also for patients who are expectorating purulent sputum and who have not received prior antibiotic.
- Pneumococcal urinary antigen should be performed in all severe patients.
- Legionella urinary antigen should be performed for severe community-acquired pneumonia or risk factors for legionella (see aetiology section).
- Mycoplasma serological assays can be used.
- Serological tests also available for Chlamydia, influenza, adenovirus and respiratory syncytial virus.

## Severity assessment

Community-acquired pneumonia presents as a wide spectrum of disease and therefore has a variable clinical course and mortality rates. It is important to assess the severity of community-acquired pneumonia in order to establish the risk to the patient and guide management.

Factors associated with poor prognosis[3,7,8]:

- Age over 65
- Raised respiratory rate is one of the most reliable indicators of poor prognosis in community-acquired pneumonia
- Altered mental state
- Low blood pressure
- Hypoxaemia
- Leucopenia or leucocytosis
- Bilateral involvement or multilobar pneumonia on chest radiograph
- Positive blood culture.

Predictive models have been developed using these factors to risk-stratify patients. The system most commonly used in the UK is the CURB-65 score[9].

| Confusion | Abbreviated Mental Test Score <8 |
|---|---|
| **U**rea | >7 mmol/l |
| **R**espiratory rate | >30/min |
| **B**lood pressure | systolic<90 mmHg or diastolic<60 mmHg |
| **65** | age >65 years |
| CURB-65 = 0–1 | Low risk of death, treat as having non-severe pneumonia, may be suitable for home treatment |
| CURB-65 = 2 | Increased risk of death, should be considered for hospital admission |
| CURB-65 > 3 | High risk of death, should be managed as having severe pneumonia |

Box 3: CURB-65 score.

## Management

All patients should be appropriately investigated and assessed for severity.

### General measures

Oxygen should be given to keep saturations above 92%. Volume status should be assessed and intravenous fluids given if necessary.

### Antibiotic management[4]

Antibiotic therapy is the mainstay of treatment of community-acquired pneumonia, reducing the length of illness, reducing the risk of complications and lowering mortality. Most often the specific causative agent will not be identified so the choice of antibiotic will be empirical. Mild/moderate pneumonia is usually treated with oral antibiotics while severe community-acquired pneumonia is treated with intravenous therapy. Whilst choosing appropriate antibiotics local guidelines must be taken into account. Some trusts have issued guidance based not only on the best agent to use, but also local difficulties with *Clostridium difficile*.

| | Preferred treatment | Alternative |
|---|---|---|
| Mild/moderate Oral medication | Amoxicillin +/– erythromycin or clarithromycin | Erythromycin/ clarithromycin or levofloxacin/moxifloxacin |
| Severe IV medication | Co-amoxiclav or cefuroxime or cefotaxime or ceftriaxone + erythromycin clarithromycin | Levofloxacin/moxi-floxacin + benzylpenicillin |

Box 4: Antibiotic treatments for community-acquired pneumonia.

Patients treated initially with parenteral antibiotics should be transferred to an oral treatment as soon as clinical improvement occurs and the temperature has been normal for 24 hours.

Patients with non-severe and uncomplicated pneumonia require treatment with appropriate antibiotics for seven days. For patients with severe community-acquired pneumonia 10 days of treatment is usually recommended.

Therapy can be aimed at a specific pathogen if identified by microbiological investigation.

| Pathogen | Preferred antibiotic | Alternative |
|---|---|---|
| *Streptococcus pneumoniae* | Amoxicillin or benzylpenicillin | Erythromycin/ clarithromycin or cefuroxime/cefotaxime |
| *Mycoplasma pneumoniae* | Erythromycin or clarithromycin | Tetracycline or fluoroquinolone |
| Legionella | Clarithromycin +/– rifampicin | Fluoroquinolones |
| *Haemophilus influenzae* | Amoxicillin or co-amoxyclav | Cefuroxime/cefotaxime or fluroquinolone |
| *Chlamydia psittaci* | Tetracycline | Erythromycin or clarithromycin |

Box 5: Pathogen-specific antibiotic treatment.

## Complications

### Parapneumonic effusions and empyemas

Parapneumonic effusions develop in 36–57% of patients with community-acquired pneumonia admitted to hospital[10].This can be a common cause for persistent fevers and treatment failure and is associated with a raised mortality. All effusions seen on x-rays should be aspirated and sent for microscopy and culture. Unless the fluid is frankly purulent (in which case it should require immediate drainage) a sample should be placed in a heparinised arterial blood gas syringe. If the effusion has a pH <7.2, measured using a blood gas analyser, then it should also be drained. Pleural effusions may be drained using a small seldinger or traditional cut down intercostal drain. Small drains require regular flushing (e.g. normal saline 20 ml tds) to ensure patency. Thrombolytic therapy was shown not to improve outcomes in a recent study[11], though it is still favoured by some.

## Lung abscess

Lung abscess is a rare complication more often associated with aspiration pneumonia. Treatment is with prolonged IV antibiotics and possible surgical intervention.

## Follow-up

Patients should be reviewed at around six weeks following discharge from hospital. Often a chest radiograph is performed at this stage, although the evidence for this is inconclusive. Chest radiograph is recommended at six weeks for all those patients who have persistent symptoms or physical signs or who are at higher risk of underlying malignancy (especially smokers and those over 50 years)[4]. Further investigations should be considered in patients with persisting signs, symptoms, and radiological abnormalities. This may include referral through local (cancer) MDTs.

## Prevention

Vaccination can be used as a preventive tool in high-risk patients. Currently available vaccines include influenza A and *Streptococcus pneumoniae*[4].

Influenza A vaccine is currently recommended for patients with chronic lung disease or diabetes, those who are immunosupressed, over the age of 65, healthcare workers and those with cardiac, renal or liver disease.

Pneumococcal vaccine is recommended for asplenic individuals and those with renal disease, heart disease, lung disease, liver disease, diabetes mellitus, and immunodeficiency.

## Conclusion

A working knowledge of community-acquired pneumonia is essential for every junior doctor. Developing skills in the diagnosis and assessment of patients with the disease, as well as keeping an up-to-date view of management strategies will be vital in minimising morbidity and mortality in many patients.

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# Guidelines for managing community-acquired pneumonia

Raj Parthasarathy, Saibal Ganguly and Charlotte Cannon

***Can improved outcomes be achieved when patients with community-acquired pneumonia are managed in accordance with the national guidelines?***

Effective management of community-acquired pneumonia (CAP) is associated with significant reduction in morbidity, mortality and better use of health service resources. The aim of this audit cycle was to assess whether the British Thoracic Society Updated guidelines were adhered to when managing patients with CAP. It looked into whether the duration of hospital stay would be affected when patients were assessed for the severity and an evidence-based approach was undertaken while investigating and treating them. The audit involved a retrospective note review of all patients admitted to the acute assessment unit with a diagnosis of pneumonia over a period of six months between January and July 2005 and a re-audit was undertaken between December 2005 and June 2006. We were able to show a significant improvement in most aspects of management of CAP and specifically an early discharge among patients with mild pneumonia when guidelines were followed.

## Aim

To audit the management of CAP in the Acute Assessment Unit against the hospital guidelines based on the British Thoracic Society update 2004. The principal factors taken into consideration were the documentation of severity assessment, investigations undertaken, antibiotics prescribed, time taken for administration of first dose antibiotic, referral to intensive care unit for patients with severe pneumonia, duration of hospital stay and follow-up in outpatient clinic.

## Method

Files of all patients admitted to the acute assessment unit and fulfilling ICD-10 criteria for pneumonia over a period of 6 months between January and July 2005 were studied retrospectively. A re-audit was undertaken for a period of 6 months from November 2005 to April 2006.

Inclusion criterion was diagnostic criteria for CAP being fulfilled. Immunocompromised patients and those with malignancy and chronic lung diseases were excluded.

## Results

### Audit (January–July 2005)

Among the 97 patients coded as having pneumonia, 59 satisfied the inclusion criteria for the audit. Only two patients had their CURB-65 score documented. The CURB-65 score was then calculated retrospectively. There were 29, 18 and 12 patients in the mild (CURB-65 = 0 to 1), moderate (CURB-65 = 2) and severe (CURB-65 = 3 to 5) categories, respectively (Table 1).

In the mild category, 93% of patients had some form of microbiological investigation carried out, even though this is not recommended routinely[12,13,14]. None of the patients in the moderate and severe categories had the appropriate investigations undertaken.

Of those patients in the mild and moderate categories, 70% and 94%, respectively received intravenous antibiotics without an appropriate reason being documented (see Table 2). The intravenous therapy most commonly used was the second-generation cephalosporin cefuroxime.

Only 42% of patients in the severe category received appropriate initial antibiotic therapy. All patients who were treated with intravenous cefuroxime received a suboptimal dose of 750 mg three times daily as opposed to 1500 mg three times daily as specified in the guideline. Three out of four patients with effusion had a diagnostic pleural tap.

| CURB-65 score | Sputum C/S | Blood C/S | Urinary Legionella Ag. | Appropriate investigations |
|---|---|---|---|---|
| 0–1 (n=29) | 5/16 [31.3%] | 21/29 [72.1%] | 1/29 [3.44%] | 27/29 [93.1 %][(1)] |
| 2 (n=18) | 1/10 [10%] | 7/18 [39%] | 2/18 [11%] | None |
| 3–5 (n=12) | 1/12 [8.3%] | 6/12 [50%] | 0/12 | None [8.3% [(2)]] |

Table 1: Investigations performed.

[(1)] Microbiological investigations were performed though not recommended routinely.

[(2)] 8.3 % when atypical screen was excluded.

| CURB-65 score | IV treatment | PO treatment | Appropriate antibiotic therapy |
|---|---|---|---|
| 0–1[(2)] | 20/29 [70%][(1)] | 9/29 [30%] | 14 % |
| 2[(3)] | 17/18 [94%][(1)] | 1/18 [6%] | None |
| 3–5 | 9/12 [75%] | 3/12 [25%] | 42% |

Table 2: Antibiotics administered.

[(1)] Intravenous therapy was administered without any appropriate reason being documented.

[(2)] 42% received cefuroxime and macrolide as the initial antibiotic.

[(3)] 72% received cefuroxime and macrolide as the initial antibiotic.

# Audit

| | Audit [n=59] | Re-audit [n=54/71] | Standards |
|---|---|---|---|
| Severity assessment | 3.4% | 76% | 100% |
| **Investigations** | | | |
| Mild (0–1) | 93%(1) | 88.9%(1) | None required routinely |
| Moderate (2) | 0 | 50% | 100% {Blood and sputum C/S} |
| Severe (3–5) | 0 [8.3%](2) | 9% [58.8%](2) | 100% {Blood, sputum and urinary antigen/ atypical screen} |
| **Appropriate antibiotics** | | | |
| Mild (0–1) | 14% | 44.4% | PO amoxicillin |
| Moderate (2) | 0 | 42.8% | PO amoxicillin + macrolide |
| Severe (3–5) | 42% | 77.3% | IV cefuroxime/co-amoxiclav + macrolide |
| First dose antibiotic < 2 hrs | 12% | 33% | < 2 hrs |

Table 3: Comparison of initial audit and re-audit.
(1) Some form of microbiological investigation was performed though not recommended.
(2) Percentage in brackets signifies appropriateness in investigations performed when atypical screen was excluded.

Only 12% of all patients received the first dose of antibiotic within two hours of admission (Table 3). Of the patients with severe pneumonia, a third had their first dose within two hours of admission. None of the patients in the severe category were considered for intensive care management. All six patients with do not attempt resuscitation orders succumbed to the infection.

In the mild category, 10% and 40% of patients were discharged on the same day and within two days, respectively.

Fifty nine per cent (35 out of 59) of patients had an outpatient follow-up. Fifteen were with the GP and the rest in a hospital clinic. Seventeen had repeat chest x-ray (CXR) performed in 6–8 weeks.

## Interventions undertaken

The audit results were presented in the grand round. The protocol for management of CAP was added to the Acute Assessment Unit Treatment protocol list. The junior doctors were enlightened about the treatment protocol. All junior doctors who newly joined the department had relevant induction into the protocols and guidelines for the management of commonly encountered illnesses. The results were also published in the Quality Support Services newsletter and a re-audit was planned.

## Re-audit: November 2005 to April 2006

In this period 140 patients were coded as having pneumonia. Of these, 71 patients satisfied the inclusion criteria for the audit. Severity assessment based on the CURB-65 score was carried out in 54 patients (76.05%). Among those assessed there were 18, 14 and 22 patients in the mild (CURB-65: 0–1), moderate (CURB-65 = 2) and severe (CURB-65 = 3 to 5) categories, respectively.

A significant proportion of patients within the moderate category (50%) had appropriate investigations as compared to none in the initial audit. Among the severe category, when atypical screen was excluded, 58.8% of patients had appropriate investigations as opposed to 8.3% in the initial audit. Very few patients in the severe category had atypical screen done. In the mild category, patients underwent microbiological investigations as routine in both the audits.

Significant improvement was observed in the number of patients who received appropriate antibiotics in all three categories. The percentage of patients who received appropriate antibiotics when the CURB-65 score was calculated were 44.4%, 42.8% and 77.3% in the mild, moderate and severe categories, respectively (see Table 3). This is a considerable increase when compared to 14%, 0 and 42% in the mild, moderate and severe categories in the initial audit where severity was not assessed. Of the seven patients who had pleural effusion, four had a diagnostic tap.

More than half the number of patients with severe pneumonia received the first dose of antibiotic within two hours of admission. Overall a third of the patients received the first dose within two hours of admission (Table 3). Two patients in the severe category were considered for and admitted to the intensive care unit. One patient died in intensive care. Nine of the patients for whom resuscitation was deemed inappropriate succumbed to their illness. Patients with severe pneumonia received 1.5 g cefuroxime unless contraindicated.

The percentage of patients with mild pneumonia discharged on the same day and within two days of admission were 50% and 61.1%, respectively. Over 50% (28 out of 54) of patients had a follow-up organised in 6–8 weeks' time. A follow-up CXR was done in 22 patients.

## Discussion

It has been shown in previous studies that CURB-65 scoring (Box 1) is superior and more reliable in identifying low-risk patients who can be safely discharged[2,3]. It is considered to be a powerful tool in predicting mortality among seriously ill patients (CURB-65 score of 3 or more). A reduction in 30-day mortality was shown among patients treated for CAP when guidelines were followed[1]. Lim *et al*[4] have shown a considerable increase in mortality as the score increases, with the worse being when the scores are between 3 and 5.

The initial audit clearly showed that we failed to comply with the national guidelines. We were unsuccessful in recognising the

C: New mental confusion Abbreviated Mental Test score of 8 or less
U: urea >7 mmol/l
R: respiratory rate 30/min or more
B: blood pressure – systolic BP <90 mmHg or diastolic BP <60 mmHg
65: age 65 or more

Box 1: CURB-65 score.

low-risk group who could have been managed in the community. This led to unnecessary hospital admissions and use of hospital resources. Patients were subjected to an array of investigations and many received intravenous antibiotics (predominantly intravenous cefuroxime) without appropriate clinical reasoning. Cephalosporin antibiotics, cefuroxime in particular, have been shown to be associated with a higher incidence of *Clostridium difficile*-associated diarrhoea[5,6]. *Clostridium difficile* infection is on the rise and has a significant adverse impact on inpatient healthcare delivery in the UK[7,8,9].

On the other hand, patients with severe pneumonia were treated with a lower dose of cefuroxime. Garton *et al* have demonstrated that cefuroxime at a dose of 1500 mg three times a day results in serum levels greater than the minimal inhibitory concentration (MIC) for respiratory pathogens over a prolonged period of time as compared to half the dose (750 mg three times a day)[15]. A review by intensive care physicians was not sought for patients with severe pneumonia.

In contrast to the general idea that doctors do not follow guidelines[10], our audit cycle showed that when junior and senior doctors were informed about the guidelines they did adhere to them in practice. All junior doctors who joined the department received appropriate induction. They were made aware of the protocols and guidelines they had to follow for the management of commonly encountered clinical situations. They were required to follow these guidelines as a minimum requisite.

David S *et al*[11] have shown that rapid antibiotic delivery and appropriate antibiotic selection reduced the length of hospital stay of patients with CAP. Our audit cycle showed a significant rise in the percentage of patients who received antibiotics early.

When treating a patient with CAP a physician should use the CURB-65 score as a tool to assess the severity but should be guided by his clinical judgement on the best management plan[13].

## Conclusions

Accurate severity assessment plays a vital role in the use of appropriate investigations and antibiotics in CAP and influences the duration of hospital stay. This enhances the clinical and cost-effectiveness of the treatment provided. This audit cycle has clearly shown that a more sensible approach to the use of investigations and antibiotics based on the guidelines has improved outcomes. However, the audit did not look into antibiotic-associated complications. We did not review the time taken for discharge in patients in the moderate and severe categories, as there were social factors and other co-morbidities that affected the outcome. A re-audit is planned for in two years.

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# Respiratory arrest: a case for urgent treatment

David Mbamalu and Ashis Banerjee

A 22-year-old previously fit male was brought to the emergency department in respiratory arrest. Earlier on the same day, he had developed progressively increasing shortness of breath. He was administered nebulised salbutamol by paramedics en route to hospital, but rapidly became apnoeic and had a seizure on the way to hospital.

On arrival in the resuscitation room, he was unconscious, with a Glasgow coma score of 4/15 (E1, M2, V1). His vital signs were recorded as: pulse rate 108 beats per minute, blood pressure 90/54 mmHg, and temperature of 34.2°C. Pulse oximetry revealed an oxygen saturation of 65%. He was noted to be apnoeic.

His initial assessment and management were as follows:

Airway: Guedel airway insertion; administration of oxygen by non-rebreathing mask at 15 litres per minute; proceeding to rapid sequence induction and tracheal intubation.

Breathing: assisted ventilation.

Circulation: peripheral venous access in both upper limbs.

Disability: GCS 4/15; signs of decerebration were noted; pupils unequal but reactive to light; capillary blood glucose: 15 mmol/l.

**Question 1.**

A portable chest x-ray (Figure 1) was obtained in the resuscitation room shortly after arrival. What does the x-ray show?

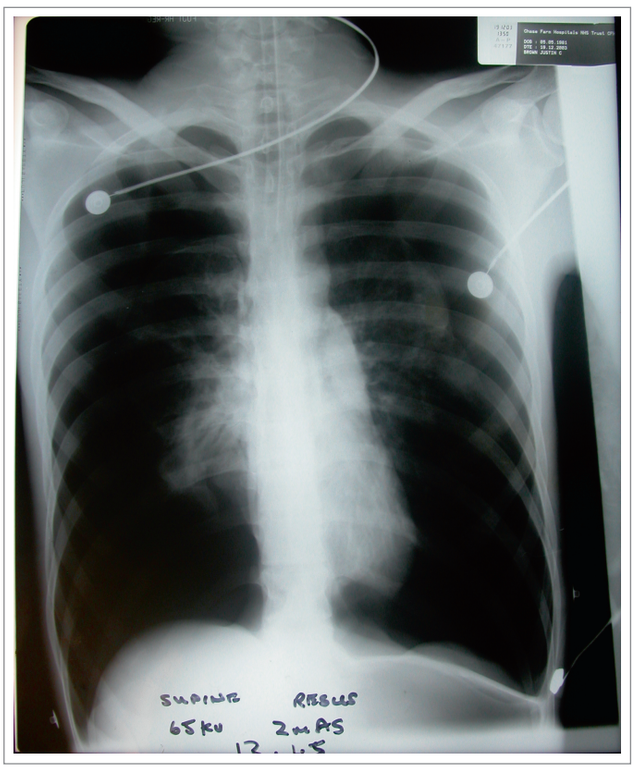


Figure 1.

**Answer**

Bilateral large pneumothoraces, with central trachea and tracheal tube in situ.

**Question 2.**

How would you manage the patient in the light of these radiological findings?

**Answer**

Immediate decompression of the pleural spaces is indicated.

Bilateral intercostal intubation was performed after needle decompression for our patient. Initial aspiration is recommended in the British Thoracic Society (BTS) guidelines for large pneumothoraces with complete lung collapse. In this life-threatening situation a trial of aspiration was felt to be contraindicated. Chest drains were placed simultaneously bilaterally, while the patient was in the resuscitation room (Figure 2).

All patients with bilateral spontaneous pneumothorax should be subsequently referred for consideration of surgical management as guided by the BTS guidelines. Surgery involves resection of blebs or bullae and obliteration of the pleural space. The approach may involve either video-assisted thoracoscopy or open thoracotomy. The procedures for pleural space obliteration include parietal pleurectomy, abrasion pleurodesis or laser pleurodesis.

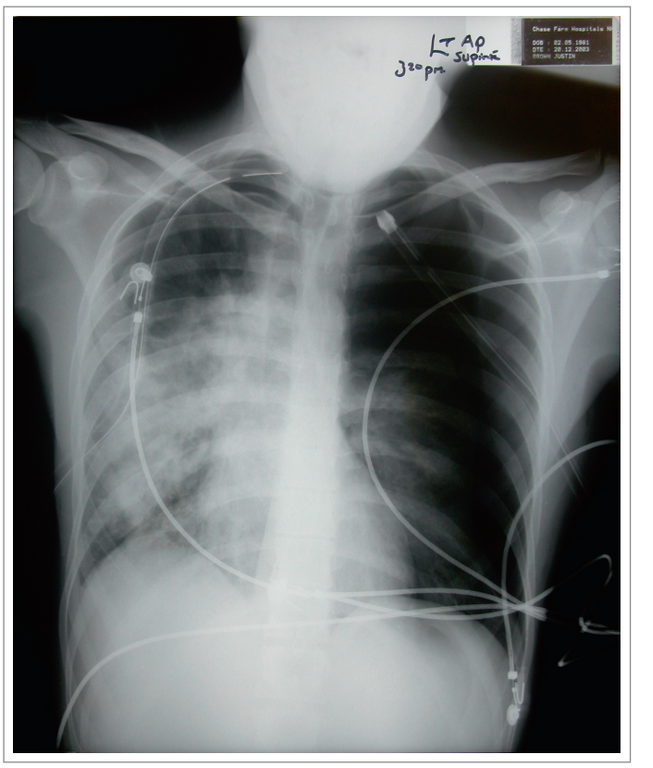


Figure 2.

**Question 3.**

What are the potential causes of this condition?

**Answer**

Bilateral spontaneous pneumothorax may be due to a variety of causes, many of which are also cause unilateral spontaneous pneumothorax. These include:

1. Primary
   - Sub-pleural apical blebs and bullae.
2. Secondary
   - COPD; asthma
   - Interstitial lung disease: lymphangioleiomyomatosis (LAM); sarcoidosis
   - Cystic fibrosis
   - Infections: tuberculosis; pneumocystis carinii pneumonia in AIDS; Staphylococcal pneumonia; necrotising Gram negative pneumonia
   - Connective tissue diseases: Marfan's syndrome; Ehlers-Danlos syndrome
   - Neoplasm: metastases; primary neoplasm
   - Pneumoconioses
   - Hyperbaric oxygen therapy.
3. Unilateral spontaneous pneumothorax becoming secondarily bilateral via pleuro-pleural communications, which have been reported with major invasive thoracic procedures, specifically mediastinal surgery. The buffalo or bison has a single pleural cavity, one of the few mammals to do so.

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# A patient with diffuse lung disease

Vijay Joshi and Ashis Banerjee

***A 68-year-old man presented with a one-week history of progressive breathlessness with effort intolerance and orthopnoea. For the preceding five months he had also noted dysphagia to solids. He had a 20 pack-year smoking history, and a history of ischaemic heart disease.***

***On admission he was in moderate respiratory distress, hypoxic and apyrexial. Chest examination revealed bilateral crackles with a mild expiratory wheeze and dullness to percussion at the right lung base. The remainder of the examination was normal.***

## What first-line investigations would you perform?

- Full blood count
- Electrolytes
- Inflammatory markers: C-reactive protein (CRP)
- 12-lead ECG
- Chest x-ray
- Arterial blood gases

### Results of these investigations

Admission blood tests showed elevated white cell count with neutrophilia, hyponatremia, and normal C- reactive protein (CRP).

A 12-lead ECG excluded acute ischaemia.

The chest x-ray is shown in Figure 1.

Arterial blood gases revealed type 1 respiratory failure.

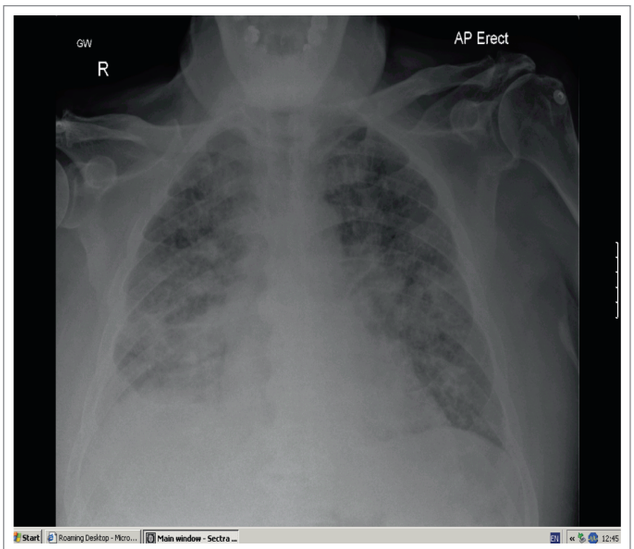


Figure 1: Chest x-ray.

## What does the chest x-ray show, and what is the differential diagnosis?

The chest x-ray shows bilateral diffuse interstitial infiltrates and a right pleural effusion.

When a patient presents with progressive breathlessness, lung crackles and diffuse parenchymal infiltrates on chest radiograph, the differential diagnosis includes infective pneumonia, pulmonary oedema, interstitial lung disease including acute exacerbation of idiopathic pulmonary fibrosis, and neoplastic disease, e.g. bronchoalveolar cell carcinoma, lymphangitis carcinomatosis (Box 1).

## In the light of the chest x-ray, what further assessment is required?

Once interstitial lung disease (ILD) is suspected a stepwise approach including review of the history, physical examination, laboratory investigations and imaging such as high resolution computed tomography (HRCT) is required to secure the clinical diagnosis.

Clinical history should focus on occupational exposures to asbestos and coal dust, environmental exposures (e.g. contact with birds), medications (e.g. amiodarone, methotryxate), patient travel, systemic diseases (collagen vascular diseases, vasculitis, sarcoidosis), risk factors for immunosuppression, and cigarette smoking.

The diagnostic clues from the history and blood tests (eosinophilia, autoantibodies, avian precipitins and raised angiotensin-converting enzyme levels), and characteristic radiological features on HRCT (nodules, thickened septa, reticulation, ground glass opacities, honeycombing and involvement of pleura) reveal the primary cause of interstitial lung disease in the majority of instances.

Lung function tests typically show restrictive defect as evidenced by reduced lung volumes, impaired gas transfer and hypoxaemia. A reduced transfer factor for carbon monoxide and transfer coefficient are typical of interstitial lung involvement.

Bronchoscopy with bronchoalveolar lavage sampling (BAL) is very useful in detecting opportunistic infections in the setting of immunosuppression in addition to its diagnostic role in sarcoidosis, hypersensitivity pneumonitis, vasculitis, etc.

If the diagnosis remains unclear, then lung biopsy, either bronchoscopic or thoracoscopic, is indicated. Surgical lung biopsies play a key role in the histopathological classification of interstitial lung disease, which has implications for the prognosis and response to treatment, especially in idiopathic interstitial pneumonia[1].

- Pleural involvement:
  Pleural plaques and benign pleural effusion due to asbestos exposure; lymphangitis carcinomatosa; radiation pneumonitis; lymphangioleiomyomatosis (LAM) (chylous pleural effusion)
- Hilar/mediastinal lymphadenopathy:
  Sarcoidosis; lymphoma, berylliosis
- Hilar nodal eggshell calcification
  Silicosis; sarcoidosis
- Pneumothorax:
  Eosinophilic granuloma; LAM
- Increased lung volumes: (commonly reduced lung volumes are noted in ILD)
  Interstitial lung disease superimposed on COPD; eosinophilic granuloma, chronic hypersensitivity pneumonitis

Box 1: Chest x-ray findings in interstitial lung disease.

## How would you treat the patient initially?

He was treated with a combination of antibiotics (for possible infective pneumonia), diuretics (element of left heart failure) and oral steroids (for probable underlying interstitial fibrosis). Whilst on this treatment he remained unwell with intermittent fever, which raised the possibility of diffuse interstitial lung involvement secondary to miliary tuberculosis, sarcoidosis or vasculitis.

## In treatment non-responders what further investigations are required?

As initial treatment failed to improve the patient's clinical condition the following tests were undertaken:

1. Repeat blood tests including FBC, CRP, which revealed persistently raised white cell count, normal CRP and negative vasculitic screen. Serum angiotensin convertase enzyme levels (ACE) were normal. Tumour markers (CEA: 4666 ng/ml, Ca125: 644 U/ml) were raised.
2. Pleural fluid analysis showed lymphocytosis but no malignant cells.
3. Echocardiogram showed antero-apical hypokinesia in keeping with history of ischaemic heart disease.
4. CT scan of chest, abdomen and pelvis (Figures 2 and 3) revealed extensive mediastinal, hilar and para-aortic lymphadenopathy with diffuse interstitial parenchymal involvement. No focal primary site of cancer was seen apart from oesophageal thickening/ oedema. Benign renal and hepatic cysts were also noted.
5. Barium swallow excluded oesophageal stricture but revealed slight mucosal thickening.
6. Bronchoscopy for tissue diagnosis bronchoalveolar lavage sampling was planned but severe hypoxia precluded bronchoscopy. Unfortunately his condition deteriorated gradually and he died.

A post-mortem examination was performed. Macroscopically both lungs looked fibrotic, with no pleural involvement. An irregular firm nodule (1×1 cm) was seen in the left upper lobe. Widespread hilar and para-aortic lymphadenopathy was apparent. The lower end of oesophagus and cardia of the stomach showed mucosal irregularity and thickening of the wall suggesting extraluminal compression. The remaining abdominal viscera were normal.

Histology revealed diffuse, poorly differentiated metastatic adenocarcinoma with focal signet ring cell features involving lungs, peri-pancreatic tissue, lymph nodes, oesophagus, stomach. Immunohistochemistry was positive for CEA, CK7, CK20 and negative for TTF1, consistent with metastatic adenocarcinoma with primary from distal oesophagus/stomach.

## Learning point

Despite initial work-up, if the diagnosis remains uncertain the possibility of malignancy should be considered in the differentials of bilateral interstitial infiltrates. Prompt lung biopsy is necessary to make the correct diagnosis. The primary cancer may remain occult and will only be discovered at autopsy, as illustrated in our patient. This highlights the fact that progressive breathlessness secondary to pulmonary involvement may be the first or the only manifestation of occult cancer[2].

Literature review suggests that various malignancies of non-pulmonary origin manifest with progressive breathlessness and diffuse interstitial lung involvement. The published case reports reveal that the most common underlying primary cancer was a diffuse gastric adenocarcinoma[2,3,4]. In these patients, despite several examinations including gastroscopy, the primary cancer remained occult and the definitive diagnosis was apparent only at autopsy[3].

Dennstedt et al reported six cases of pulmonary lymphangitis carcinomatosis (PLC) from occult stomach carcinoma in young patients who had bilateral interstitial infiltrates on chest radiograph and the possibility of cancer was only confirmed at autopsy[5]. In these patients the underlying lymphangitis carcinomatosis or microscopic tumour embolisation to the lungs was postulated as the cause of recent onset dyspnoea[2].

Park et al have reported a case of endobronchial metastasis from stomach cancer (poorly differentiated adenocarcinoma) in a young woman with diffuse interstitial infiltration on chest radiograph[4].

Another case report features diffuse pulmonary involvement by mycosis fungoides in a 55-year-old man who presented with progressive dyspnoea and history of skin lesions. The interstitial pattern on HRCT followed by transbronchial biopsy confirmed the previously undiagnosed malignancy[6].

In a patient with hypogammaglobulinaemia, an interstitial lung disease may suggest lymphangitis from adenocarcinoma of stomach[7].

Rarely, adenocarcinoma of the lung itself has presented as diffuse interstitial process with lymphangitis of pulmonary origin in young adults[8].

In patients with cancer the progressive breathlessness could be due to an occult recurrent thromboembolism. The association of pulmonary thromboembolism and diffuse interstitial inflammation has been recognised in such patients at autopsy[9].

Rarely, diffuse interstitial lung infiltrates can be myelomatous in origin or secondary to systemic amyloidosis associated to a multiple myeloma[10].

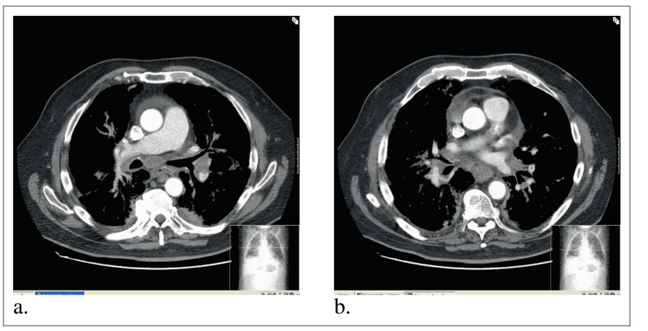


Figure 2: a. Hilar and b. Mediastinal lymphadenopathy.

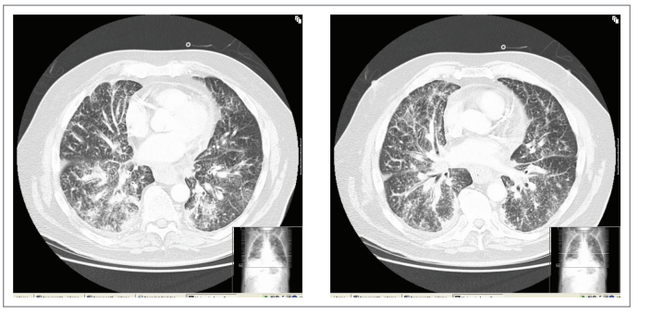

Figure 3: Diffuse interstitial infiltration of the lungs.

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# Assessment and management of haemoptysis

Vijay Joshi and Ashis Banerjee

***A 56-year-old man attends the emergency department following a history of intermittent haemoptysis over the preceding two weeks. This commenced with streaking of blood, and he coughed up a cupful of fresh blood on the morning of attendance. There was no history of chest pain. He has no systemic symptoms, and is otherwise in good general health. He has been a smoker from the age of 19, currently smoking 10 cigarettes a day. On examination, there are no localising signs in the chest. How would you manage him further?***

Haemoptysis is a common problem encountered in clinical practice. It is a non-specific symptom but can indicate significant underlying lung disease and requires prompt, early assessment and through investigation. Haemoptysis persisting longer than two weeks should be taken as being potentially due to lung cancer unless proven otherwise.

## Definition

Haemoptysis is defined as the coughing up of blood derived from the lower respiratory tract as a result of pulmonary/bronchial haemorrhage.

The severity of haemoptysis is arbitrarily divided into mild, moderate and severe depending on an estimate of the amount of revealed bleeding. Mild haemoptysis signifies the expectoration of less than 30ml blood per 24 hours or streaking or the presence of flecks of blood in the sputum. Moderate haemoptysis has been arbitrarily defined by a blood loss of 30-200ml/24 hours, and severe by a blood loss in excess of 200ml/24 hours. Massive haemoptysis has been arbitrarily defined as blood loss of more than 200ml per hour or more than 500ml over 24 hours,or bleeding leading to haemodynamic disturbance. There is, however, no consensus definition and, in any case, quantification of blood loss can be imprecise.

Haemoptysis should be differentiated from bleeding from alternative sources other than the lower respiratory tract, i.e. upper airways, nasal cavity, oral cavity or gastrointestinal tract. A history of expectoration of blood (bright red or pink) or blood streaked/frothy sputum, associated cough, and a past history of lung disease favour haemoptysis. The presence of dyspeptic symptoms, associated epistaxis and a previous history of gastric/liver disease suggest haematemesis.

**Tracheobronchial source**
- Lower respiratory tract infection (LRTI)
- Neoplasm (bronchogenic carcinoma, endobronchial metastatic tumour, Kaposi's sarcoma, bronchial carcinoid)
- Bronchiectasis
- Broncholithiasis
- Airway trauma
- Foreign body

**Pulmonary parenchymal source**
- Infective: lung abscess, pneumonia, tuberculosis (cavitating and non-cavitating disease, active/inactive disease)
- Vasculitides/alveolar haemorrhage syndromes: Goodpasture's syndrome, idiopathic pulmonary haemosiderosis, Wegener's granulomatosis, lupus pneumonitis

**Primary vascular source**
- Pulmonary arterio-venous malformation
- Pulmonary embolism with infarction
- Pulmonary venous hypertension (e.g. mitral stenosis)

**Miscellaneous/rare causes**
- Pulmonary endometriosis
- Systemic coagulopathy
- Anticoagulants or thrombolytic agents
- Lung contusion

Box 1: Causes of haemoptysis[1]

## History

Historical clues may help to identify the anatomical site of bleeding, differentiating between haemoptysis and pseudohaemoptysis/haematemesis. History taking should cover the following areas:

- Time and mode of onset of haemoptysis
- Estimated volume of blood expectorated, and whether mixed in with sputum or not
- Time course: intermittent or constant; if intermittent, the frequency of haemoptysis
- Precipitating and relieving factors
- Systemic symptoms suggestive of common underlying causes e.g. lung cancer (weight loss, anorexia), bronchiectasis/lung abscess (cough with copious purulent sputum), pneumonia/tuberculosis (fever, night sweats, productive cough), pulmonary embolism (pleuritic chest pain, acute dyspnoea), chronic obstructive pulmonary disease (wheeze, reduced effort tolerance), or left heart failure (exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea)
- Risk factors including recent travel or contact with tuberculosis, immunosuppression (e.g. HIV positive status); risk factors for thromboembolism
- An occupational history, with attention to environmental exposure to asbestos
- Past medical history, including previous episodes of haemoptysis, a history of chronic lung disease (e.g. chronic obstructive pulmonary disease, tuberculosis, bronchiectasis), malignancy (e.g. bronchogenic carcinoma; breast, renal or colon cancers), and co-existing diseases like renal, cardiac, collagen vascular diseases, and bleeding disorders
- Current medication: anticoagulation
- Immunisation status, including BCG vaccination
- A personal history, focusing on smoking (active/ passive; pack years) and alcohol consumption
- Menstrual history, as association with menses suggests catamenial haemoptysis
- Concerns of the patient, particularly regarding possibility of lung cancer.

## Examination

Baseline observations should include pulse rate, respiratory rate, temperature, blood pressure and oxygen saturation on pulse oximetry.

On general examination, signs of pallor, cyanosis, digital clubbing, nicotine staining of fingers, lymphadenopathy (neck, supraclavicular, axillary), peripheral oedema and evidence of cutaneous ecchymosis/telangiectasia should be sought. Evidence of bleeding from the oral or nasal mucosa should be noted. The presence of unilateral leg swelling and calf tenderness suggests underlying deep vein thrombosis.

Examination of the lungs should seek signs of consolidation, wheeze or trauma. Cardiovascular examination includes an evaluation of jugular venous pressure and cardiac murmurs. Abdominal examination should look for evidence of hepatomegaly or masses. If lung cancer is strongly suspected, look for evidence of Horner's syndrome, superior vena caval obstruction and hoarseness of voice.

## Investigations

Investigations can be done on an outpatient basis, but patients with significant bleeding require hospitalisation for resuscitation.

## Initial investigations

Initial investigations may include:

- Venous blood: full blood count; urea and electrolytes; coagulation screen
- 12-lead ECG
- Chest x-ray: In 20–30% of patients the chest x-ray is non-localising or normal. An abnormal chest x-ray may require to be followed on with high resolution CT scanning
- Sputum microscopy and culture

- Nodule(s)/mass(es): carcinoma, metastatic disease, vasculitides
- Atelactasis (lobar/segmental): pulmonary embolism, lung cancer
- Hilar/mediastinal lymphadenopathy: lung carcinoma, sarcoidosis, lymphoma
- Cavitary lesion: lung abscess, tuberculosis, necrotising pneumonia; a fungus ball appearance indicates aspergilloma
- Air space consolidation: pneumonia, lung carcinoma
- Reticulo-nodular densities: Wegener's granulomatosis, vasculitides
- Dilated peripheral airways/tramline shadows: bronchiectasis
- Hilar/mediastinal calcification
- Alveolar infiltrates (diffuse/ patchy): pulmonary oedema, haemosiderosis
- Cardiomegaly/pulmonary congestion: chronic heart failure
- The chest x-ray may be normal in bronchitis, upper respiratory tract infection, and pulmonary embolism
- In about 20–30% of patients with haemoptysis in a tertiary care setting chest x-ray is normal or non-localising[2]. In primary care settings the majority of patients with haemoptysis have lower respiratory tract infection and will have a normal chest x-ray. There are number of series that have examined this problem and have found the overall incidence of lung cancer of up to 3% in these patients which justifies further active investigations including bronchoscopy and CT scanning. Factors favouring malignancy include age over 40 years, current smokers, haemoptysis lasting for more than a week[3].

Box 2: Chest x-ray findings that might be associated with haemoptysis.

- Flexible fibreoptic bronchoscopy (FOB): in high-risk patients, often with a normal or non-diagnostic chest x-ray, FOB should be considered to rule out malignancy. It is diagnostic for central endobronchial disease (CT may miss upper airway pathology) and permits tissue/bronchial biopsy, bronchial lavage or brushings for histological diagnosis. Direct visualisation of the bleeding site allows for injection with a vasoconstricting agent or balloon catheter insertion for tamponade to control massive haemoptysis
- Selective pulmonary angiography
- High-resolution CT scan.

In general, a combination of high-resolution CT scan and bronchoscopy should allow for the detection of most significant causes of haemoptysis.

## Second line investigations

These include CT pulmonary angiography (CTPA) to exclude pulmonary embolism, echocardiography to assess for pulmonary hypertension and left ventricular dysfunction, bronchial angiogram (and bronchial artery embolisation of a bleeding artery where visualised), and lung function tests with transfer factor in vasculitides with lung haemorrhages. In around 30% of patients no cause may be found despite appropriate investigations (cryptogenic haemoptysis), but this carries good prognosis.

## Management

The overall goals of management of the patient with massive haemoptysis are threefold: control of the source of bleeding, prevention of aspiration and treatment of the underlying cause, e.g. coagulation deficits. As with any potentially serious condition, evaluation of the 'ABCs' (airway, breathing, and circulation) is the initial step.

The most common presentation is acute, mild and transient haemoptysis caused by lower respiratory tract infection, either bronchitis or pneumonia, which settles with treatment of the underlying cause. Low-risk patients with normal chest radiographs can be treated with close monitoring and appropriate oral antibiotics, if clinically indicated. If haemoptysis persists or remains unexplained, specialist input in the form of chest clinic referral is indicated.

The mortality rate from massive haemoptysis depends on the bleeding rate and aetiology. Haemoptysis greater than 1,000 ml per 24 hours in the presence of malignancy carries a mortality rate of 80%;[4] therefore, massive haemoptysis warrants an aggressive approach to treatment.

Airway protection (via keeping the bleeding lung and the head dependent, and by early tracheal intubation) and ventilation of the non-bleeding lung is vital for adequate gas exchange. Selective intubation or the use of a bronchial blocker may be preferable to a double lumen tracheal tube, requiring skilled anaesthetic support. Airway maintenance is crucial to prevent asphyxiation from flooding of the alveoli and airways with blood. Supplemental oxygenation and full cardiovascular support including fluid resuscitation are essential. Rigid bronchoscopy is preferable as it allows localisation of the bleeding site and bronchial tamponade with a balloon catheter can be performed. Bronchial artery embolisation, and surgical resection of the bleeding lobe are rarely required if all measures fail to prevent bleeding.

### Management of massive haemoptysis[4]

Massive haemoptysis usually arises from systemic bronchial arteries, rather than the low-pressure pulmonary arterial system. The amount of bleeding has no relationship to the seriousness of the underlying pulmonary lesion. The key management principles are:

- High-flow inspired oxygen
- Placement of the bleeding lung in the dependent position
- Bronchoscopy to clear the airway, localise the site of bleeding and institute local control measures. This is best achieved by a combination of rigid and fibre-optic flexible bronchoscopy under general anaesthesia
- Initial clearing of the major airways is followed by irrigation with 10 ml aliquots of normal saline or 1;200,000 adrenaline solution and suction
- The fibre-optic bronchoscope can be used for balloon catheter tamponade if bleeding is from a segmental bronchus
- If bleeding continues, the normal or good lung should be isolated by:
  - selective endo-bronchial intubation of the non-bleeding lung using a double-lumen endo-bronchial tube to allow isolation and ventilation of either lung as required
  - bronchial blocking (e.g. with a balloon catheter, such as a Foley catheter) in the affected lung via a single-lumen tube
  - using a combination endotracheal tube and bronchial blocker placed under bronchoscopic control (e.g. the Univent tube)
- Once the normal lung has been protected from aspiration, and if the patient is deemed to be operable, definitive surgical treatment can proceed
- In patients not suitable for lung (lobe) resection but who continue to bleed (especially from cavities in the lung, which are almost always fed by bronchial arteries), embolisation of the bronchial artery under radiographic control is an alternative
- Radiation treatment may be helpful if the bleeding is due to cavitating cancer

Poor prognostic factors include: increasing age, preexisting lung/cardiac disease, respiratory compromise, hypoxia, ongoing haemoptysis and shock.

## Specific management in this scenario

Management of this patient should involve investigations to exclude lung cancer, particularly in view of his smoking history. He should undergo first line investigations including venous blood testing for full blood count and urea and electrolytes, and a 12-lead ECG. A chest x-ray is essential to look for any focal lesion which will guide subsequent tests. A normal chest x-ray does not exclude the presence of a small bronchial neoplasm. After initial screening, he should be referred to the chest clinic with a view to be seen within two weeks for further evaluation including bronchoscopy and CT scan of the chest.

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# Chronic cough

D. Lonsdale, M.V. Holmes, E.M. Giddings and B. Lams

***A 67 year old, who had never smoked, presented with a productive cough for 6 months, and then developed features of pneumonia which persisted for a further 2 months despite antibiotics. A CT scan revealed an endobronchial dense opacity in the right main bronchus (Figure 1). Bronchoscopy identified a chicken bone wedged across the intermediate bronchus (Figure 2), which was successfully removed with forceps. In retrospect, the bone is visible on his initial chest x-ray (CXR) adjacent to the right heart border (Figure 3, arrow).***

Chronic cough is defined as one that lasts more than 8 weeks[1,2]. Common causes in adults include rhinosinusitis, postnasal drip, asthma, chronic obstructive pulmonary disease (COPD), smoking, gastro-oeophageal reflux disorder (GORD), angiotensin-converting enzyme inhibitor therapy and, more rarely, eosinophilic bronchitis[1,2,3]. These conditions often present with normal findings on chest radiography, unlike in our case, and require appropriate further investigation.

The British Thoracic Society recommends a systematic approach to diagnosis and treatment[2]. First, a detailed history including occupational and smoking history should be taken together with relevant physical examination. Second, spirometry and a CXR are mandatory. Further investigation may include ear, nose and throat examination with fibreoptic laryngoscopy (rhinosinusitis, postnasal drip), lung function with bronchodilator response (asthma, COPD), bronchial provocation testing (asthma) and sputum eosinophilia. A trial of proton pump inhibitors and alginates is recommended prior to ambulatory pH monitoring for suspected GORD. Bronchoscopy is essential in all cases of suspected inhaled foreign body. Smoking cessation is encouraged.

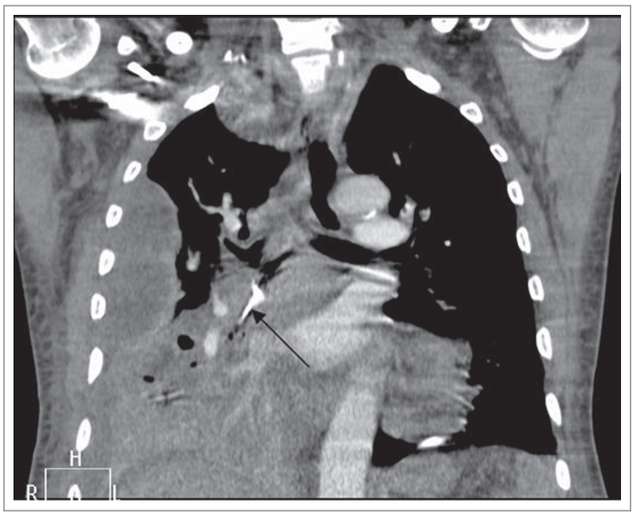


Figure 1: Coronal CT showing opacity in intermediate bronchus (arrow) with consolidation of right middle and lower lobes and a right pleural effusion.

Figure 2: Chicken bone recovered at bronchoscopy shown with pencil to demonstrate its scale.

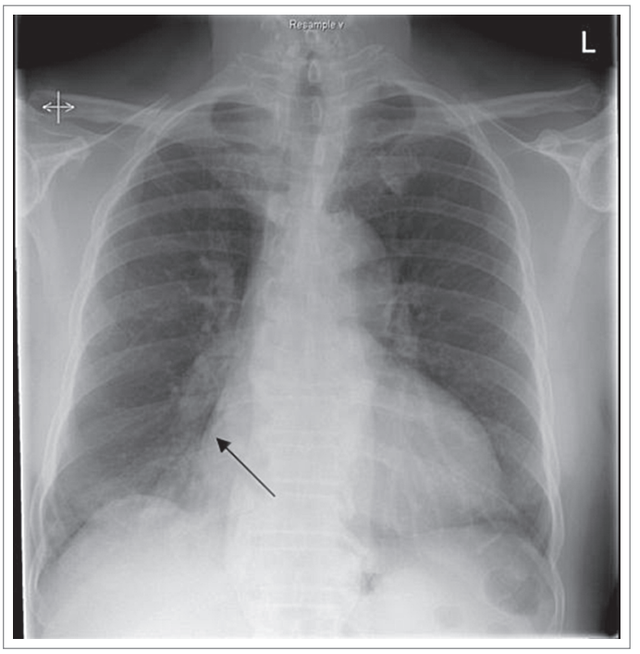


Figure 3: Admission PA chest X-ray. The chicken bone is visible as an opacity at the right heart border (arrow).

## Summary

In situations where persistent infection is the causative factor of non-resolving pneumonia, consideration must be made of atypical organisms, especially if co-existent immunosuppression, and obstructing lesions (tumour, foreign body). This case illustrates that foreign bodies may be a cause of chronic cough and non-resolving pneumonia even if there is no recollection of aspiration.

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# Breathlessness, lung fibrosis and the limitations of Occam's razor

S. Deshmukh, M. Warwick-Sanders and S.C. Allen

## Abstract

***We present an elderly woman who was admitted to hospital with an apparent exacerbation of a chronic condition. Investigation revealed that her symptoms and signs were caused by three pulmonary pathologies. One was previously diagnosed, one was acute and one became apparent for the first time after admission. We take this opportunity to discuss not only the current guidance available for the management of one of this patient's pathologies, idiopathic pulmonary fibrosis, but also to take a lateral-thinking look at the changing principles of diagnosis and management in an acute medical world populated by many patients of advanced age with pathology in multiple systems.***

## Case report

Mrs FM, an 88-year-old woman presented to hospital with an increase of the longstanding weakness in her right arm, and a cough productive of green sputum. Apart from the weakness, there were no other acute focal neurological symptoms. In addition, she was incontinent of urine, which was a new symptom, though she did not have dysuria, increased urinary frequency or rigors.

Her past medical history included type 2 diabetes, chronic renal failure, ulcerative colitis, hypertension, idiopathic pulmonary fibrosis and a left hemisphere stroke resulting in slight residual weakness of the right arm and leg.

Neurological examination revealed no new signs compared with her previous admissions, and a CT scan of her brain suggested no new cerebral pathology. Respiratory examination showed a respiratory rate of 26/minute, a prolonged expiratory phase, widespread fine 'velcro' inspiratory crepitations, and coarse crackles at the right lower zone. She had a tympanic membrane temperature of 38°C, a total white blood cell count of 23.0 × $10^9$/L (92% neutrophils), and her urinary dipstick was positive only for nitrites. Her chest radiograph showed extensive fibrotic change and some softer shadowing at the right lower zone. Her oxygen saturation on air was 88%.

The working diagnosis at this stage was lower respiratory tract infection with underlying pulmonary fibrosis and she was treated initially with antibiotics and supplementary oxygen. Further discussion with the patient brought to light a long history of cigarette smoking (total exposure about 20 pack-years), and a tendency for wheezy breathlessness and daily sputum, particularly in the winter. There was no history of industrial dust exposure. Although a diagnosis of asthma or chronic obstructive airways disease (COPD) had never been made, it was likely that airflow obstruction was also contributing to her symptoms and signs. Bronchodilator drugs by nebuliser and oral corticosteroids were added to her treatment regimen. She was unable to perform full spirometry, but her $FEV_1$ improved from 1.12L to 1.38L after treatment. Therefore, this patient's main symptom, dyspnoea, was caused by three separate lung pathologies: pulmonary fibrosis, diffuse airflow obstruction and infection. She improved sufficiently to be discharged home.

One of these pathologies, pulmonary fibrosis, will be discussed in more detail.

## Pulmonary fibrosis

This is a broad term for a collection of pulmonary disorders, all of which eventually result in the laying down of fibrotic tissue in the lung parenchyma. The usual presentation is with breathlessness and dry cough. Many patients have an abnormal chest radiograph at the time of presentation, though that is not invariable. There is a wide differential diagnosis. For example, it has to be differentiated from pulmonary oedema, infection and neoplastic disorders. In some patients it is a manifestation of a systemic disorder, such as rheumatoid disease, or drug-induced, for example by amiodarone. When no cause is identified it is referred to as idiopathic or cryptogenic.

*Synonyms:* Idiopathic pulmonary fibrosis (IPF), cryptogenic pulmonary fibrosis, idiopathic interstitial pneumonia (IIP) (this term is increasingly being favoured)[1]. However, not all IIPs are IPF and responses to treatment vary.

It forms about 15% of the workload of respiratory physicians but also commonly presents to other specialties[2].

The ERS/ATS classification is widely accepted and divides diffuse parenchymal lung disease (DPLD) into four main groups[1]:

1. Idiopathic interstitial pneumonias
2. DPLD of known cause (Tables 1, 2 and 3)
3. Granulomatous DPLD (e.g. sarcoidosis)
4. Other forms of DPLD.

For the sake of this article we will concentrate on chronic DPLDs.

| Agent inhaled | Disease caused |
|---|---|
| Inorganic dusts | Asbestosis<br>Silicosis<br>Coal workers pneumoconiosis<br>Aluminium lung<br>Siderosis (iron)<br>Stanossis (tin) |
| Organic dusts | Farmer's lung (thermo-actinomycetes in mouldy hay)<br>Bagasossis (thermo-actinomycetes in mouldy sugar cane)<br>Cheese worker's lung (mouldy cheese)<br>Bird fancier's lung (avian protein on feathers) |

**Table 1:** Chronic DPLD due to occupational or environmental agents.

| | |
|---|---|
| Antibiotics | Nitrofurantoin, sulphasalazine |
| Anti-inflammatories | Gold, penicillamine, aspirin |
| Cardiovascular agents | Amiodarone |
| Chemotherapeutic agents | Bleomycin, methotrexate |
| Illicit drugs | Heroin, methadone, talc |
| Miscellaneous | Oxygen, radiation |

**Table 2:** DPLD due to drugs.

| | |
|---|---|
| Connective tissue disorders | Systemic sclerosis, systemic lupus erythematosus<br>Sjogren's syndrome, ankylosing spondylitis<br>Rheumatoid arthritis, polymyositis<br>Mixed connective tissue disorders, Churg Strauss syndrome |
| Neoplastic | Lymphoma, lymphangitis carcinomatosa |
| Vasculitis | Wegener's granulomatosis, microscopic polyangiitis |
| Inherited disorders | Tuberous sclerosis, neurofibromatosis |

**Table 3:** DPLD due to systemic diseases.

## History taking in pulmonary fibrosis

*Length of history*: The time of onset, overall duration and rate of progression of the disease are useful in providing clues to the diagnosis. The onset can be classified into acute (usually <3 weeks), episodic and chronic. Every effort should be made to compare the current radiographs to previous ones. The radiological onset can be earlier than clinical one, or vice versa.

*Occupational history:* A detailed account of extent of exposure to fibrogenic dusts, including the mode and duration of exposure and the use of protection gear should be gathered in both working and retired patients. It is sometimes possible to get more detailed information, including previous radiographs from employers. Hobbies and pastimes should be enquired especially regarding birds.

*Past medical history*: History of malignancy and radiotherapy will sometimes point to the aetiology, as will a history of a systemic inflammatory disorder (RA, SLE etc). Previous use of and failure to respond to diuretics is a common finding in patients in whom the diagnosis was mistakenly thought to be pulmonary oedema. Asthma and rhinitis are invariable findings in Churg Strauss syndrome.

*Family, smoking and drugs history*: IPF and sarcoidosis can be familial[3]. Patients with Goodpasture's syndrome are invariably smokers whereas those with sarcoid and extrinsic allergic alveolitis very rarely are[4]. There are many drugs that can cause DPLD (see Table 2).

## Laboratory and diagnostic tests

1. *Blood tests:* All forms of the disease may be associated with an elevated ESR, CRP and serum immunoglobulins. After acute exposure to an antigen, neutrophilia and lymphopenia are common, though eosinophilia is rare. If a suggestive exposure history is elicited serum precipitins against suspected antigens should be part of the workup. If found, serum precipitins suggest sufficient exposure to the causative agent to mount an immune response.

   Serologic tests to screen for collagen vascular diseases are necessary to help exclude these diagnoses. Such tests include rheumatoid factor, anti-DNA antibody (single and double stranded), anti-smooth muscle and anti-mitochondrial antibodies, and anti-neutrophil cytoplasmic antibody.

2. *Current and previous plain chest radiographs:* In the acute phase specific or diagnostic changes are often absent. Chronic states show a diffuse reticulonodular infiltrate, or more established fibrosis. Comparison of images is helpful to time the progression of the disease.

3. *CT scanning:* HRCT is considered if the diagnosis is either unclear or is likely to be IPF. HRCT is capable of visualising the lung with excellent spatial resolution and provides images akin to gross pathological examination. HRCT is able to detect DPLD not visible on plain radiographs. A confident diagnosis is more likely to be made and more likely to be correct with HRCT. Since it provides a cross-sectional view a more accurate estimate of the disease process and other co-existing pulmonary diseases is possible. This minimises – but does not negate – the need for histological diagnosis. HRCT is able to differentiate between active and inactive disease. Predominant ground glass pattern is more likely to represent active disease and helps to predict response to appropriate therapy especially in IPF. It is felt that reticulonodular shadowing follows ground glass pattern and is more likely to represent inactive and chronic disease. Traction bronchiectasis with ground glass pattern is found with fibrosis and absence of traction bronchiectasis with ground glass pattern suggests active inflammation.

4. *Lung function tests:* In patients with advanced disease, reduction in total lung capacity, vital capacity and residual volume are found. A restrictive ventilatory pattern is usually noted, with a normal or increased $FEV_1/FVC$ ratio. Arterial blood gases usually show a normal pH, resting hypoxia and normal $pCO_2$. Oxygen desaturation during exercise is almost always found. Diffusing capacity of the lung for carbon monoxide ($DL_{CO}$) is reduced. Lung function tests are used to monitor the progression of the disease. Caution should be exercised in smokers as it is possible to misinterpret the extent of the restrictive defect due to co-existent obstructive lung disease.

5. *Echocardiography:* Pulmonary hypertension may at times mimic, and is a frequent complication of, pulmonary fibrosis.

6. *Bronchoscopy/bronchoalveolar lavage:* Bronchoscopy and bronchoalveolar lavage is considered if any of the following are likely: sarcoidosis, infection, malignancy or organising pneumonia. Trans-bronchial biopsy can help to confirm the diagnosis of sarcoidosis, hypersensitivity pneumonitis and other broncho-centric diseases.

## Treatment

Patients with suspected DPLD should be referred to chest physicians for management and consideration of specific treatment. They are managed by a multidisciplinary team of physicians, radiologists, pathologists and physiotherapists. The choice of treatment and likelihood of a response varies between types of DPLD.

The management of IPF is discussed here; the management of individual types of DPLD can be found in relevant textbooks. It must be noted that treatment is usually concerned with stabilising the disease and preventing decline rather than improving it.

*Corticosteroids and IPF: conclusions of the British Thoracic Society guidelines on DPLD*[5]:

- There has been no placebo-controlled trial of steroids in IPF, so there is no direct evidence that steroids improve survival.
- A proportion of patients do respond in terms of symptoms (about 50%) and lung function (about 25%).
- Although a steroid response is associated with better survival, it is not clear if this is due to the treatment itself.
- There are no comparative data on dose regimens or on the length of time for which steroids or other treatment should be used.

Treatment recommended for IPF is as follows:

- Recommended initial treatment is therapy with: oral prednisolone 0.5 mg/kg alone for patients with predominant ground glass pattern. If the ground glass pattern is NOT predominant, a combination of oral prednisolone and azathioprine is recommended. Most treatment regimens also contain N-acetylcysteine.
- More detailed aspects of management are outside the scope of this paper but can be found in published guidelines[5].

## Discussion

Our patient presented with worsening of her respiratory symptoms due to three pulmonary pathologies and also had at least two other organ pathologies that contributed to her overall constellation of symptoms and signs at the time of admission (worsening of her longstanding hemiparetic weakness and urinary incontinence, both probably as a consequence of the hypoxia). As such, she was representative of a large proportion of patients presenting acutely to hospital; being elderly, having a number of established co-morbidities and an acute illness due to mixed acute and chronic pathology. Patients presenting in this way lay a challenge to one of the classic teachings of clinical medicine, namely, the principle of parsimony of causation, sometimes known as Occam's razor. To paraphrase, this states that a single diagnosis that explains all the symptoms and signs is likely to be correct. However, though this might be sustainable in theory when all the information is clear, it is a hazardous rule of thumb in modern medical practice. This is because in frail elderly people with multiple co-morbidities it is unusual to have all the information, and it is not uncommon to have symptoms and signs with more than one cause. The casual application of Occam's razor can stop the clinician from continuing to look for undiagnosed, and often treatable, pathology.

Occam's razor is the concept that when two competing ideas seem to explain the facts, the simpler is more likely to be true. It should be added that this simpler idea is not certainly true. It is just preferred until more data can clarify the situation. Therein lies the hazard in clinical practice, particularly when determining treatment. Some aspects of the dictum remain useful to the jobbing doctor. For example, the more bizarre and complicated an idea is, the less likely it is to be the true explanation, and, the assumptions introduced to explain an observation must not be multiplied beyond necessity.

Hickam's dictum is a counterargument to the use of Occam's razor in the medical profession, and is arguably more appropriate to modern medical practice. It is commonly summarised as: "Patients can have as many diseases as they damn well please". One can easily reason the contemporary need for this rubric. It is much more likely for a person to have several common diseases rather than a single rare disease that explains the range of symptoms. It is not uncommon, especially in elderly patients, to have multiple pathologies, multiple causes of dominant symptoms and therefore multiple diagnoses. Indeed, in such cases, it is usual for multiple diagnoses to have separate causes rather than a single unifying cause.

Some commentators have raised doubts regarding the assessment methods adopted under the auspices of Modernising Medical Careers but this case proves that modern methods can be used critically to appraise older concepts. This case helps to liberate our thought process from the clutches of that elusive "unifying diagnosis".

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