123 Doc EDUCATION

# FOUNDATION YEARS JOURNAL

JULY 2009

Volume 3, Issue 6: Respiratory

Contents



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FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 6

## Foundation Years Journal

**Foundation Years Journal** is an international peer-viewed journal which seeks to be the pre-eminent journal in the field of patient safety and clinical practice for Foundation Years' doctors and educators. The Journal welcomes papers on any aspect of health care and medical education which will be of benefit to doctors in the Foundation training grade in the UK or international equivalents.

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## Volume 3, Issue 6: Respiratory

**Foundation Years Journal** is the ONLY journal for Foundation Years doctors and educators, specifically written according to the MMC curriculum. It focuses on one or two medical specialties per month, each issue delivers practical and informative articles tailored to the needs of junior doctors. The Journal closely follows the Foundation Years syllabus to provide the best educational value for junior doctors. In addition to good clinical and acute care articles, assessment questions give junior doctors the chance to gauge their learning.The answers will be published in the next issue, but 123Doc will advance answers to clinical tutor subscribers so they can engage their students in the learning process. Each issue provides comprehensive clinical cases for trainees as well as practical teaching assessments for educators. Readers will benefit from:

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## Aim and scope

**The Foundation Years Journal** is published by 123doc and is aimed at doctors in Foundation Training programmes, their educational and clinical supervisors, as well as medical students and other doctors (particularly international medical graduates) who intend to start Foundation training in the United Kingdom.

## Journal sections

The Journal has been redesigned and various sections have been introduced to map the Journal more closely to the Foundation programme curriculum. You can view the curriculum from **http://www.foundationprogramme.nhs.uk/pages/home/training-and-assessment**.

**The sections are the following:**

**1. Good Clinical Care (syllabus section 1)**
This section deals with various aspects of patient management including history, examination, diagnosis, record keeping, safe prescribing and reflective practice. Articles could also refer to other aspects of care including time management, decision-making, patient safety, infection control, clinical governance, nutrition, health promotion, patient education, public health and ethical and legal issues.

**2. Good Medical Practice (syllabus section 2)**
Articles could be on learning, research, evidence-based guidelines and audit.

**3. Training and Teaching (syllabus section 3)**

**4. Professionalism in Practice (syllabus sections 4, 5 and 6)**
This section includes relationship with patients, communication skills, working with colleagues, probity, professional behavior and personal health.

**5. Patient Management (syllabus section 7)**
Articles should be focused on the recognition and management of the acutely ill patients, core skills in relation to acute illness, resuscitation, management of the "take", discharge planning, selection and interpretation of investigations.

**6. Practical Procedures (syllabus section 8)**

**7. Test Yourself**
The intention is to provide a vehicle whereby trainees and educational supervisors can present original and review articles mapped against the Foundation curriculum.

## Submission of manuscript

All articles submitted to the Journal must comply with these instructions. Failure to do so will result in return of the manuscript and possible delay in publication.

Manuscripts must be submitted exclusively by email (see detailed instructions below). Manuscripts should be written in English of a sufficiently high standard that is intelligible to the professional reader who is not a specialist in the particular field. Where contributions are judged as acceptable for publication, the Editor or the Publisher reserve the right to modify the manuscripts to improve communication between author and reader. Authors whose native language is not English are strongly recommended to have their submissions checked by a person knowledgeable of the language. If extensive alterations are required, the manuscript will be returned to the author for revision.

## Covering letter

The manuscript must be accompanied by a covering letter bearing the corresponding author's signature. Papers are accepted for publication in the Journal on the understanding that the content has not been published or is being considered for publication elsewhere. This must be stated in the covering letter. If authors submit manuscripts relating to original research in the field of education, the corresponding author must state that the protocol for the research project has been approved by a suitably constituted Ethics Committee and that it conforms to the provisions of the Declaration of Helsinki (as revised in Edinburgh 2000), available at **http://www.wma.net/e/policy/b3.htm**. All investigations involving human subjects must include a statement that the subject gave informed consent and patient anonymity should be preserved.

The covering letter must contain an acknowledgement that all authors have contributed significantly and that all authors are in agreement with the content of the manuscript.

Authors should declare any financial support or relationships that may give rise to a conflict of interest.

## Submitting a manuscript

Manuscripts should be submitted by email to **(agnes@123doc.com)**. We do not accept manuscripts submitted by post. Corresponding authors must supply an email address as all correspondence will be by email. Authors should use double spacing when submitting their manuscript. Two files or documents should be supplied: the covering letter and manuscript. The covering letter should mention the title, authors, their contribution, provenance, journal section where their work is to be considered (see above) and any conflict of interests. Please supply the files in Word 2003 format.

Figures should be supplied as a separate file, with the figure number incorporated in the file name. High-resolution figures (at least 300 d.p.i.) saved as jpeg files should be submitted.

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## Manuscript style

Unless otherwise stated manuscripts should follow the style of the Vancouver agreement detailed in the International Committee of Medical Journal Editors' revised "Uniform Requirements for Manuscripts Submitted to Biomedical Journals: Writing and Editing for Biomedical Publication", as presented at **http://www.ICMJE.org/**.

**Abbreviations**

Abbreviations should be used sparingly to facilitate reading the article by reducing repetition of long, technical terms. Initially you must use the word in full, followed by the abbreviation in parentheses. Thereafter use the abbreviation only.

**Units**

All measurements must be given in SI or SI-derived units.

**Trade names**

Drugs should be referred to by their generic names, rather than brand names.

## References

All articles must be referenced appropriately. To reference the Journal please use the following abbreviation FYJ-123Doc. (The Vancouver system of referencing should be used and some examples are given below).

References should be cited using superscript Arabic numerals in the order in which they appear. If cited in tables or figure legends, number according to the first identification of the table or figure in the text.

In the reference list, the references should be numbered and listed in order of appearance in the text. Cite the names of all authors, when seven or more list the first three followed by et al. Names of journals should be abbreviated in the style used in Index Medicus, and be in italic font. Reference to unpublished data and personal communications should appear in the text only.

**References should be listed in the following forms:**

**Journal article**

Vassallo M, Vignaraja R, Sharma JC, et al. The Impact of Changing Practice on fall Prevention in a Rehabilitative Hospital. The Hospital Injury Prevention (HIP) Study. *J Am Geriatr Soc 2004,* 52:335-9. Book Azeem T, Vassallo M, SamaniNJ. *Rapid review of ECG interpretation.* London UK: Manson Publishing 2005.

**Chapter in a book**

Martin GM. Biological mechanisms of ageing. In: J Grimley Evans, T Franklin Williams (eds), *Oxford Textbook of Geriatric Medicine,* 1st edn. New York: Oxford University Press 1992, 41-48.

**Journal article on the internet**

British Geriatrics Society position paper. Dementia ethical issues **http://www.bgs.org.uk/Publications/Position%20Papers/psn_dementia_ethics.html**.

**Tables**

Tables should be self-contained and complement, but not duplicate, information contained in the text. Number tables consecutively in the text in Arabic numerals. Table should be double-spaced and vertical lines should not be used to separate columns. Column headings should be brief, with units of measurement in parentheses; all abbreviations must be defined in footnotes. Footnote symbols: †, ‡, §, should be used (in that order) and *, **, *** should be reserved for P-values. The table and its legend/footnotes should be understandable without reference to the text.

**Line figures**

Line figures should be sharp, black and white graphs or diagrams, drawn professionally or with a computer graphics package. Lettering must be included and should be sized to be no larger than the Journal text.

**Colour figures**

We encourage authors to submit colour figures and graphics that facilitate the comprehension of the article.

**Figure legends**

Type figure legends on a separate page. Legends should be concise but comprehensive - the figure and its legend must be understandable without reference to the text. Include definitions of any symbols used and define/explain all abbreviations and units of measurement. The Journal accepts the following types of articles (as title please):

## Case Based Discussion

These are mainly intended for inclusion in sections 1 and 5 as highlighted above and should be about 1000-1500 words long. The CBD can focus on various aspect of patient care such as presentation, treatment or prescribing. The articles should include areas that are evaluated in the case based discussion assessment tool of the foundation programme .

**The manuscript should be set out in the following sections:**

- Abstract: this should refer to salient points from the case being presented together with a mention of what aspects are being discussed.
- Case History: this relates to the initial presentation and should include the clinical setting, clinical problem, investigations and treatment. The history section should also include an ongoing update (e.g. 2 days later, a week later, etc.) of patient progress and management.
- Discussion: this section should include a critical analysis of patient management in relation to clinical assessment, investigations, differential diagnosis, treatment, follow-up, professionalism and clinical judgement. The discussion should also include a discussion about the ongoing management issues and decisions. It is important to note that the case based discussion is not a review of a particular condition.
- Two best of 5 MCQs to be included in the Test Yourself section, with answers and detailed teaching notes explaining the answers. The answers only are NOT sufficient and it should be kept in mind when writing the teaching notes that the reader may take the test questions independently from reading the article.

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Authors writing a case based discussion should not write a short history and then write an article about the condition that the patient presented with. Such information can easily be obtained from a text book and is not the scope of Journal. Case based discusions written in this style will be returned to the author without being published.

## Practical Procedures

Manuscripts on practical procedures should be about 1000–1500 words long. They should be set out in the following sections:

- History: this should describe the presentation of the patient and mention why or how the patient ended up needing the procedure.
- The procedure itself.

**This should include:**

- indications and contraindications
- explaining the procedure to the patient (including possible complications) and gaining informed consent for procedures
- preparing the required equipment, including a sterile field
- position the patient and give pre-medication/sedation or local anaesthesia as required and involving the anaesthetist where appropriate
- safely disposing of equipment, including sharps
- documenting the procedure, including labelling samples and giving instructions for monitoring and aftercare
- recording complications and the emergency management of such complications when appropriate.

Adequate pictures and diagrams need to be supplied in order to make the procedure as clear as possible.

Two best of 5 MCQs for inclusion in the test yourself section, including answers and detailed teaching notes. The answers only are NOT sufficient and it should be kept in mind when writing the teaching notes that the reader may take the test questions independently from reading the article.

## Audit

Manuscripts, 1500–2000 words long, on audit are encouraged. The Journal will only publish high quality audit i.e. completed audit cycles or audits that have led to guideline development. Part 1 audits or surveys will not be accepted for publication.

## Review Articles

We are interested in review articles on any aspect of the curriculum that is of relevance to our readership. They should be a maximum 3000 words long, 30 references, 250 word structured abstract, 4 tables OR figures.

**We would consider reviews on any of the following:**

- Good Medical Practice
- Teaching and Training
- Professionalism
- Medical reviews subject to prior discussion with the editorial team as to the appropriateness of the article

## Shorter Reflective Practice Articles

We are always pleased to receive short pieces of a thoughtful nature that describes the personal or professional experiences of colleagues working with patients or their relatives. They should have a maximum of 1000 words. As suggested in the Foundation Programme Portfolio (Reflective Practice) these articles should describe:

- What made the experience memorable?
- How did it affect you?
- How did it affect the patient?
- How did it affect the team?
- What did you learn from the experience and what if anything would you do differently next time?

**Some aspects to be considered in these articles are:**
Communication with the patient, ethical issues, aspect of your works with colleagues, probity and honesty, personal health.

## Research Papers

**The Foundation Years Journal** would welcome research articles on Medical Education. Other research papers would be considered if thought to be of interest to the readership of the Journal. Articles should be written using the following headings (title page, abstract, introduction, methods, results, discussion acknowledgements, references, tables, illustrations legends.). They should be of a maximum of 2500 words of text, plus abstract, 30 references, 3 tables or figures. Manuscripts including a structured abstracts should have a maximum of 250 words using the headings introduction, methods, results, conclusion. The title page should contain (i) the title of the paper; (ii) the full names of the authors; and (iii) the addresses of the institutions at which the work was carried out together with; (iv) the full postal and email address, plus facsimile and telephone numbers, of the author to whom correspondence about the manuscript should be sent.

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# ASTHMA

David Gibeon and Dan Ornadel

**A 34-year-old lady presents to A&E with a 4-hour history of rapidly increasing shortness of breath and wheeze. Her only previous medical history was of asthma, which had been diagnosed in her childhood.**
**Good Clinical Care.**

A 34-year-old lady presents to A&E with a 4-hour history of rapidly increasing shortness of breath and wheeze.

Her only previous medical history was of asthma, which had been diagnosed in her childhood. She had multiple admissions to hospital as a child but her symptoms had been well controlled on inhalers until 6 weeks previously when she developed a coryzal illness. In the past 7 days she had noticed fevers and a productive cough of green sputum.

Her peak expiratory flow rate is 360L/min at best. Her husband states that she has been down to just 100L/min for the past 3 weeks and over the past 3 days she has not been able to perform peak flows at all.

On examination she looks sweaty, tired and is using her accessory muscles to breathe. Her respiratory rate is 36 breaths per minute, pulse 130 beats per minute, oxygen saturations 90% on 15 litres oxygen via a non-rebreather mask and her temperature is 38 degrees Celsius. Auscultation of her chest reveals a loud polyphonic expiratory wheeze bilaterally with crackles at the left base.

## What do you think is wrong here?

The patient has a known diagnosis of asthma and her recent history is suggestive of a chest infection. The likely diagnosis is that she has an infective exacerbation of asthma. The observations indicate that she is very unwell and should receive urgent treatment. This lady is likely to have been taken directly to the resuscitation department.

The patient in this case has a potentially life-threatening asthma attack. The BTS guidelines on the management of asthma in adults provide a summary of the assessment process[2].

# ASTHMA

David Gibeon and Dan Ornadel

## Management Of Acute Asthma In Adults

### MANAGEMENT OF ACUTE ASTHMA IN ADULTS

### ASSESSMENT OF SEVERE ASTHMA

B **Health care professionals must be aware that patients with severe asthma and one or more adverse psychosocial factors are at risk of death.**

☑
- Keep patients who have had near fatal asthma or brittle asthma under specialist supervision indefinitely
- A respiratory specialist should follow up patients admitted with severe asthma for at least one year after the admission

### INITIAL ASSESSMENT

#### MODERATE EXACERBATION

- increasing symptoms
- PEF >50-75% best or predicted
- no features of acute severe asthma

#### ACUTE SEVERE

Any one of:
- PEF 33-50% best or predicted
- respiratory rate ≥25/min
  heart rate ≥110/min
- inability to complete sentences in one breath

#### LIFE THREATENING

In a patient with severe asthma any one of:
- PEF <33% best or predicted
- $SpO_2$ <92%
- $PaO_2$ <8 kPa
- normal $PaCO_2$ (4.6-6.0 kPa)
- silent chest
- cyanosis
- feeble respiratory effort
- bradycardia, arrhythmia, hypotension
- exhaustion, confusion, coma

#### NEAR FATAL

Raised $PaCO_2$ and/or requiring mechanical ventilation with raised inflation pressures

| | |
|---|---|
| **Clinical features** | Severe breathlessness (including too breathless to complete sentences in one breath), tachypnea, tachycardia, silent chest, cyanosis or collapse<br>*None of these singly or together is specific and their absence does not exclude a severe attack* |
| **PEF or $FEV_1$** | PEF or $FEV_1$ are useful and valid measures of airway calibre. PEF expressed as a % of the patient's previous best value is most useful clinically. In the absence of this, PEF as a % of predicted is a rough guide |
| **Pulse oximetry** | Oxygen saturation ($SpO_2$) measured by pulse oximetry determines the adequacy of oxygen therapy and the need for arterial blood gas (ABG). The aim of oxygen therapy is to maintain $SpO_2$ ≥92% |
| **Blood gases (ABG)** | Patients with $SpO_2$<92% or other features of life threatening asthma require ABG measurement |
| **Chest X-ray** | Chest X-ray is not routinely recommended in the absence of:<br>- suspected pneumomediastinum or pneumothorax<br>- suspected consolidation<br>- life threatening asthma<br>- failure to respond to treatment satisfactorily<br>- requirement for ventilation |

**Assessment Of Severe Asthma (BTS GUIDELINES)**

# ASTHMA

David Gibeon and Dan Ornadel

## Management Of Acute Asthma In Adults

### MANAGEMENT OF ACUTE ASTHMA IN ADULTS

#### CRITERIA FOR ADMISSION

- B — Admit patients with any feature of a life threatening or near fatal attack.
- B — Admit patients with any feature of a severe attack persisting after initial treatment.
- C — Patients whose peak flow is greater than 75% best or predicted one hour after initial treatment may be discharged from ED, unless there are other reasons why admission may be appropriate.

#### TREATMENT OF ACUTE ASTHMA

##### OXYGEN

- C — Give high flow oxygen to all patients with acute severe asthma.
- A — In hospital, ambulance and primary care, nebulised $\beta_2$ agonist bronchodilators should be driven by oxygen.
- A — Outside hospital, high dose $\beta_2$ agonist bronchodilators may be delivered via large volume spacers or nebulisers.
- C — The absence of supplemental oxygen should not prevent nebulised therapy being given if indicated.

##### $\beta_2$ AGONIST BRONCHODILATORS

- A — Use high dose inhaled $\beta_2$ agonists as first line agents in acute asthma and administer as early as possible. Reserve intravenous $\beta_2$ agonists for those patients in whom inhaled therapy cannot be used reliably.
- ☑ In acute asthma with life threatening features the nebulised route (oxygen-driven) is recommended.
- A — In severe asthma (PEF or $FEV_1 < 50\%$ best or predicted) and asthma that is poorly responsive to an initial bolus dose of $\beta_2$ agonist, consider continuous nebulisation.

##### STEROID THERAPY

- A — Give steroids in adequate doses in all cases of acute asthma.
- ☑ Continue prednisolone 40-50 mg daily for at least five days or until recovery.

##### IPRATROPIUM BROMIDE

- B — Add nebulised ipratropium bromide (0.5 mg 4-6 hourly) to $\beta_2$ agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to $\beta_2$ agonist therapy.

##### OTHER THERAPIES

- B — Consider giving a single dose of IV magnesium sulphate for patients with:
  - acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
  - life threatening or near fatal asthma.
- ☑ IV magnesium sulphate (1.2-2 g IV infusion over 20 minutes) should only be used following consultation with senior medical staff.
- B — Routine prescription of antibiotics is not indicated for acute asthma.

##### REFERRAL TO INTENSIVE CARE

Refer any patient:
- requiring ventilatory support
- with acute severe or life threatening asthma, failing to respond to therapy, evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnea
  - ABG analysis showing ↓ pH or ↑ $H^+$
  - exhaustion, feeble respiration
  - drowsiness, confusion
  - coma or respiratory arrest

**Criteria For Admission (BTS GUIDELINES)**
Note: An updated guideline for the treatment of acute asthma is due in May 2009.

# ASTHMA

David Gibeon and Dan Ornadel

## What treatment would you administer?

The emergency treatment of asthma includes: high-flow oxygen; nebulised bronchodilators; steroids (this can be oral prednisolone if the patient is well enough to swallow tablets or intravenous hydrocortisone if not); intravenous magnesium; and a prompt decision regarding whether the patient requires invasive mechanical ventilation. In this case, the patient will also require antibiotics to cover for a community-acquired pneumonia. The BTS provide a summary of the management guidelines[2].

## What investigations would you order?

Many investigations can take place while treatment is being administered. Intravenous access will be accompanied by blood tests for a full blood count, urea and electrolytes, CRP and blood cultures. An arterial blood gas will provide important information and guide further treatment. A chest radiograph may show focal consolidation or a pneumothorax.

## Background

Asthma is one of the most common chronic diseases worldwide. In the UK there are over 5 million people receiving medication for asthma and there are approximately 1,400 deaths each year in the UK directly attributable to asthma. Asthma may occur at any time in life, although it tends to develop during infancy and childhood. **http://www.asthma.org.uk**

## Pathophysiology

Asthma is a chronic inflammatory condition of the airways characterised by recurrent episodes of airway obstruction. Airways become blocked by plugs of mucus and inflammatory exudates with accompanying vasodilatation and oedema. Chronic inflammation leads to structural changes. These include bronchial smooth muscle hypertrophy and hyperplasia, new vessel formation, interstitial collagen deposition resulting in basement membrane thickening and airway wall remodelling.

Hyper-responsiveness of the airways is due to a variety of exogenous and endogenous stimuli and a specific pattern of mucosal inflammation involving activated mast cells, eosinophils and T lymphocytes.

In most cases asthma is an allergic disorder that is mediated by immunoglobulin E (IgE) dependent mechanisms. IgE is synthesised and released by B lymphocytes and circulates in the blood before binding to Fc receptors on the surface of mast cells; basophils; eosinophils; monocytes; macrophages; and platelets[1]. The two main receptors for IgE are FCεR1$_{(C1)}$, known as the high affinity receptor; and FCεR2$_{(C2)}$, known as the low affinity receptor. Following binding to these receptors a number of mediators are released, which include histamine; prostaglandins; leukotrienes; chemokines; and cytokines. These mediators are responsible for many of the features that are associated with allergy and include bronchoconstriction in asthma.

## Diagnosing asthma

There is no gold standard test and the diagnosis of asthma is often a clinical one. If patients are incorrectly labelled as suffering from asthma from the outset then they may be subjected to years of inappropriate therapy. The presence of wheeze, breathlessness and chest tightness are secondary to variable airflow obstruction. Alternative diagnoses should always be considered and the history should explore whether symptoms are related to occupational exposures.

| Features that increase the probability of asthma: |
|---|
| • More than one of the following symptoms: wheeze, breathlessness, chest tightness and cough, particularly if:<br>- symptoms worse at night and in the early morning<br>- symptoms in response to exercise, allergen exposure and cold air<br>- symptoms after taking aspirin or beta blockers. |
| • History of atopic disorder. |
| • Family history of asthma and/or atopic disorder. |
| • Widespread wheeze heard on auscultation of the chest. |
| • Otherwise unexplained low FEV1 or PEF (historical or serial readings). |
| • Otherwise unexplained peripheral blood eosinophilia. |

| Features that lower the probability of asthma: |
|---|
| • Prominent dizziness, light headedness, peripheral tingling. |
| • Chronic productive cough in the absence of wheeze or breathlessness. |
| • Repeatedly normal physical examination of chest when symptomatic. |
| • Voice disturbance. |
| • Symptoms with colds only. |
| • Significant smoking history (i.e. >20 pack years). |
| • Cardiac disease. |
| • Normal PEF or spirometry when symptomatic. |

| |
|---|
| • Note: normal spirometry when not symptomatic does not exclude the diagnosis of asthma. Repeated measurements of lung function are often more informative than a single assessment. |

**Table 1: Clinical features which aid the diagnosis of asthma[2].**

# ASTHMA

David Gibeon and Dan Ornadel

**Asthmatic patients often have specific triggers that may lead to a hospital admission. Common causal factors for asthma are inhaled allergens. These may include house dust mite, cats, dogs or fungi. Outdoor pollens from grasses and trees are common inhaled allergens. Certain types of food may be linked with asthma exacerbations.**
**Good Clinical Care.**

| Without airflow obstruction |
|---|
| • Chronic cough syndromes. |
| • Hyperventilation syndrome. |
| • Vocal cord dysfunction. |
| • Rhinitis. |
| • Gastro-oesophageal reflux. |
| • Heart failure. |
| • Pulmonary fibrosis. |

| With airflow obstruction |
|---|
| • COPD. |
| • Bronchiectasis*. |
| • Inhaled foreign body*. |
| • Obliterative bronchiolitis. |
| • Large airway stenosis |
| • Lung cancer*. |
| • Sarcoidosis*. |
| • *May also be associated with non-obstructive spirometry. |

**Table 2: Differential diagnosis of asthma[2].**

## Causal factors

Asthmatic patients often have specific triggers that may lead to a hospital admission. Common causal factors for asthma are inhaled allergens. These may include house dust mite, cats, dogs or fungi. Outdoor pollens from grasses and trees are common inhaled allergens. Certain types of food may be linked with asthma exacerbations.

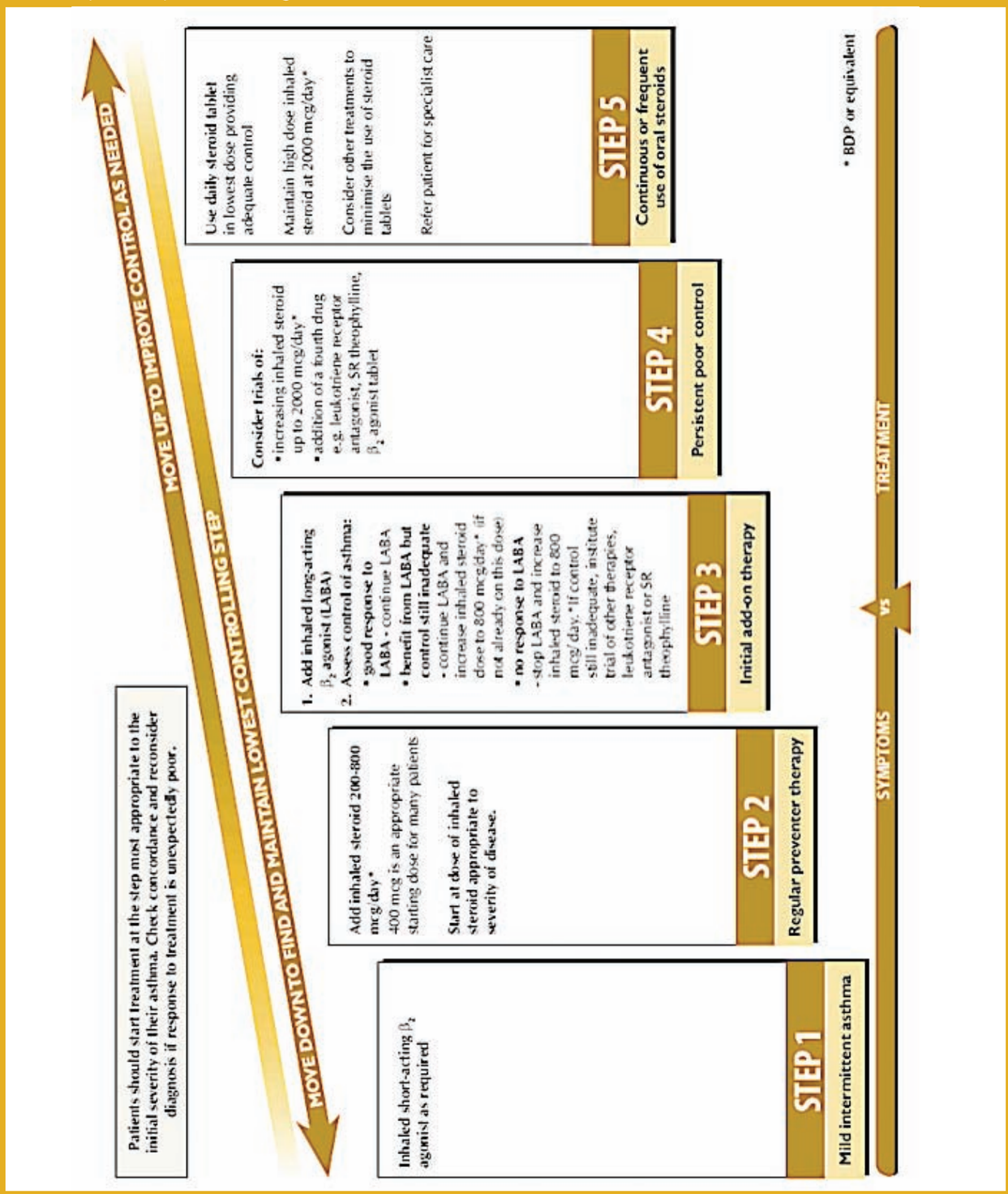
Certain drugs can be linked with bronchoconstriction. These include beta blockers and non-steroidal anti-inflammatory drugs (NSAIDs). Cigarette smoke can also be linked to triggering asthma attacks while smoking itself causes inhaled therapy to be less effective.

Common triggers for asthma include infection (which may be viral or bacterial), exercise and emotional stress. In addition changes in weather conditions and temperature have been associated with asthma exacerbations.

# ASTHMA

David Gibeon and Dan Ornadel

## Summary Of Stepwise Management In Adults




(BTS GUIDELINES)

# ASTHMA

David Gibeon and Dan Ornadel

## Monitoring asthma

Follow-up of asthmatics is an important part of ensuring that their symptoms are well controlled and that their medication is appropriate. Most asthmatics can be followed-up in primary care. Their GP or practice nurse will be able to ensure that inhaler technique is optimal and monitor symptom variability, serial peak flow readings and spirometry measurements.

Patients seen in respiratory outpatient clinics at their local hospital will have a very similar approach. In some cases the respiratory physician may decide to investigate further. This is usually when patients are not responding well to therapy or there are elements of their history or symptoms that suggest an alternative diagnosis.

For example: patients who report regular sputum production may have bronchiectasis; and patients who have fine end inspiratory crackles may be suffering with fibrotic lung disease.

Blood tests may demonstrate a peripheral blood eosinophilia. While this is often seen in asthmatics it is important to appreciate that a prominent eosinophilia requires further tests to ensure the patient does not have ABPA or a vasculitic process, such as Churg-Strauss syndrome. An eosinophilia seen in patients on oral steroids is unusual and may suggest non-compliance with therapy or an alternative diagnosis. Further blood tests such as an IgE level, aspergillus RAST and ANCA may be helpful in such cases.

Pulmonary function tests are helpful in demonstrating the severity of disease and the addition of bronchodilator reversibility is often helpful. This is established by repeating spirometry after the administration of a Ð2-agonist to see if there is an improvement in the FEV1 and FVC.

A chest radiograph may provide clues to an alternative diagnosis and prompt further imaging. Hyper-expanded lungs in a smoker may point towards emphysema rather than asthma. An HRCT may demonstrate fibrotic lung disease, bronchiectasis, lung masses or emphysema.

Specialist asthma clinics at tertiary centres will have access to further tests. These often include: skin prick tests to common aeroallergens; sputum eosinophilia count; exhaled nitric oxide levels (a marker of airway inflammation); and a PC20 (the administration of methacholine to a level that produces a 20% fall in FEV1).

## Difficult to treat asthma in adults

Patients who are treated at steps 4 or 5 of the BTS guidelines but are still experiencing symptoms and frequent exacerbations may be classed as having "difficult asthma". These patients will usually be on high dose inhaled corticosteroid therapy, a long acting Ð2-agonist and additional therapy, such as leukotriene antagonists and oral aminophylline. The prevalence of difficult asthma is approximately 5–10% of adults with asthma[3]. In many cases patients may have frequent courses of oral prednisolone therapy and in some cases they may have had many months or even years of continuous therapy. This will put them at risk of steroid induced side effects, such as gastro-oesophageal reflux; peptic ulcers; osteopenia and osteoporosis; thin skin; easy bruising; adrenal suppression; weight gain; and diabetes.

Patients with difficult asthma are important to identify because they are subject to greater risk of fatal and near fatal exacerbations. They should be seen by a respiratory physician and may subsequently be referred to a tertiary centre to see a respiratory physician who has a special interest in difficult asthma.

## New therapies

Anti-immunoglobulin E: Omalizumab (Xolair) is a humanised anti-IgE monoclonal antibody that is licensed in the European Union as add-on therapy for the treatment of severe allergic (IgE mediated) asthma. Patients must fulfil certain criteria to be eligible and the drug is administered subcutaneously on a 2–4 weekly basis for a trial period of 16 weeks. The dose is based on IgE levels and weight, and if effective without significant side effects then treatment is lifelong.

Various immunosuppressive drugs, such as ciclosporin and methotrexate, may be used in severe asthmatics to try and reduce the amount of oral steroid therapy required (i.e. a steroid sparing agent) and improve symptoms. They are usually implemented and monitored at a tertiary centre.

Bronchial thermoplasty involves delivering controlled thermal energy delivered to the airway wall during several bronchoscopic procedures. This aims to reduce smooth muscle mass that is felt to be an important part in severe asthma. Long-term studies to evaluate this treatment are awaited.

Current areas of interest and research include nucleic acid therapy, phosphodiesterase inhibitors and tyrosine kinase inhibitors. There is also research into the development of monoclonal antibodies to counteract individual cytokines, chemokines and adhesion molecules involved in asthma.

# ASTHMA

David Gibeon and Dan Ornadel

## Questions (true or false)

**1. The treatment of an acute asthma attack in hospital includes:**

**a.** Nebulised salbutamol and atrovent.
**b.** Intravenous atropine.
**c.** Oral prednisolone or intravenous hydrocortisone.
**d.** Oral antibiotics in all patients.
**e.** Intravenous magnesium.

**2. The most appropriate investigations in a 34-year-old woman presenting with acute severe asthma include:**

**a.** Spirometry.
**b.** Peak expiratory flow rate.
**c.** Arterial blood gas.
**d.** Chest radiograph.
**e.** HRCT.
**f.** D-dimer.

**3. Which of the following is the most likely arterial blood gas (ABG) finding on room air in a patient presenting with a severe asthma attack?**

**a.** pH 7.4, p02 13.5, pC02 4.8, HCO3 24, BE 0.1.
**b.** pH 7.35, p02 6.0, pC02 8.4, HCO3 38, BE 8.
**c.** pH 7.2, p02 8.5, pC02 3.5, HCO3 20, BE -7.
**d.** pH 7.48, p02 9, pC02 3.6, HCO3 27, BE 0.5.

**4. Asthmatic patients:**

**a.** Should all be on montelukast.
**b.** Are at step 4 of the BTS guidelines when they are taking a regular LABA?
**c.** May have a peripheral blood eosinophilia.
**d.** May have nasal polyposis and aspirin sensitivity.
**e.** May develop symptoms secondary to stress.

## Answers

**1. a, c and e are all TRUE.**

**2. b, c and d are TRUE.**

Measuring peak expiratory flow rate will help identify patients who may need admission to hospital. In some cases patients may be too unwell to perform this and will have a potentially life threatening asthma attack.

ii. Although a CXR is not routinely recommended according to BTS guidelines, it can provide additional information in many cases – excluding a pneumothorax and identifying any areas of consolidation.

**3. d is the most likely.**

Arterial blood gases in asthmatic patients can provide important information and may change over time. Hypoxemia may be apparent during asthma attacks. Early in their presentation an increased respiratory drive may lead to a Respiratory Alkalosis that is usually secondary to hyperventilation and a correspondingly decreased PaC02. However, a normal or raised PaC02 should alert the physician to potential failure of the respiratory system to cope with the demands of severe airway narrowing. The patient may begin to tire and use accessory muscles to breathe. The PaC02 will rise and the blood gas may demonstrate a Respiratory Acidosis.

**4. c, d and e are all TRUE.**

Montelukast is not necessary in all asthmatic patients and is usually given on a trial basis to see if there is any benefit. A regular LABA forms part of step 3 of the BTS guidelines. Many asthmatics will demonstrate a peripheral blood eosinophilia. The triad of asthma, nasal polyposis and aspirin sensitivity is known as Samters Triad. Stress can be a trigger to many asthmatic patients.

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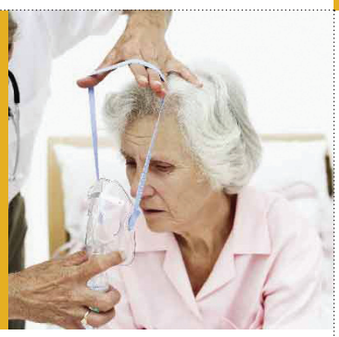
# CASE BASED DISCUSSION: HAEMOPTYSIS

Ronan Astin and Himender K Makker

**You are asked to see a 54-year-old lady who presented to the A&E with a recent history of haemoptysis. She had complained of a worsening cough over the preceding 3 months, though put this down to her smoking, having smoked 5 cigarettes per day since her mid-teens.**
**Good Clinical Care.**



You are asked to see a 54-year-old lady who presented to the A&E with a recent history of haemoptysis. She had complained of a worsening cough over the preceding 3 months, though put this down to her smoking, having smoked 5 cigarettes per day since her mid-teens. Over the last 1 week, she had noticed blood on coughing. This was not associated with any shortness of breath or chest pain. She is extremely anxious. She had not been on any recent long haul journeys and has no history of DVT or PE. She had no significant past medical history, and she was on no regular medications. Her family history includes one bother with a history of angina; a father who died of an MI aged 75; and a mother who had TB as a young adult, but is alive and generally well. Initial blood tests show Hb 14.5; MCV 88.6; WCC 7.8; CRP<3; urea 5.6; creatinine 108; and INR 1.1.

Haemoptysis is a common but non-specific symptom, often encountered in the acute setting, both in presentations to A&E and in current inpatients and is one of the leading reasons for referral to specialist respiratory services as an outpatient. It is important to have confidence in safely assessing these patients in order to institute appropriate management plans. It is also important to realise that many such patients will be anxious and scared, and providing reassurance is valuable.

## How would you assess this patient?

Haemoptysis can range from the very small volume (specks of blood) to massive (defined as 100–600ml in a 24-hour period). The latter has a high mortality, though fortunately is rare, and its management differs from the former considerably. It is therefore important to ascertain the extent of any bleed and in so doing, assess the severity of the situation. Using the ABC(DE) algorithm is an appropriate way of triaging patients and should be the first form of assessment in A&E.

In this case, the patient is maintaining her airway, her breathing is stable with a respiratory rate of 12 breaths per minute and oxygen saturations of 96% on room air, with a heart rate of 78bpm and BP 160/85.

## What would you do next?

Once you have confirmed that the patient is stable, further assessment should be made by way of taking a formal history and examining the patient.

**Points to ascertain in the history:**

- Time frame: hours, days, weeks, months; how often is blood expectorated?
- Volume of blood expectorated: use measurements (e.g. spoonful, half egg cup, egg cup, etc.) being aware that patients often initially overestimate blood loss.
- Associated symptoms: cough, sputum, fevers, night sweats, weight loss, voice change.
- Site of bleed: ensure blood is truly haemoptysis – enquire regarding epistaxis and haematemesis.
- Past history: previous lung disease? Previous malignancy, TB or other infections?
- Drug history: any anticoagulant predisposing to bleed?

**Essential enquiries:**

- smoking history
- cough, fever, other signs LRTI
- nasopharyngeal or GI bleeding
- anticoagulation

**Haemoptysis may be the only sign of an endobronchial carcinoma.**

**On examination:**

- May be normal.
- Assessment of respiratory and cardiovascular compromise.
- Signs of infection, collapse, effusion, clubbing, lymphadenopathy or DVT.

In this case, the patient gives a history of expectorating flecks of blood mixed with phlegm with no systemic features of infections, but a weight loss of 4kg in 6 months.

# CASE BASED DISCUSSION: HAEMOPTYSIS

Ronan Astin and Himender K Makker

## Which investigations would you request?

Bedside assessment of oxygen saturations and arterial blood gas measurement, if indicated by low saturations (usually if less that 92%), should be carried out. A urine dipstick can be very helpful (see later). The next investigation of choice, in this case is chest X-ray. This may suggest a diagnosis (TB, bronchiectasis, malignancy, pulmonary haemorrhage, abscess) and may obviate the need for further imaging or guide further investigations.

## Chest X-ray is normal. What would you do next?

In this case, the patient gives a history of small volume haemoptysis, is cardiovascularly and respiratorily stable and chest X-ray is normal. There is no clear cause for her haemoptysis after initial assessment; there is no evidence to suggest PE, pneumonia, bronchiectasis, TB. In view of her smoking history and weight loss, the possibility of carcinoma needs to be ruled out and this patient requires further investigation. This can safely be done as an outpatient. She can be discharged from A&E and further urgent outpatient investigations requested, such as:

- CT thorax and Upper Abdomen
- Bronchoscopy.

She underwent CT thorax which suggested a possible lesion in the right-middle lobe, with enlarged mediastinal lymph nodes. On bronchoscopy a tumour was visualised in the right-middle lobe bronchus and confirmed to be adenocarinoma on biopsy.

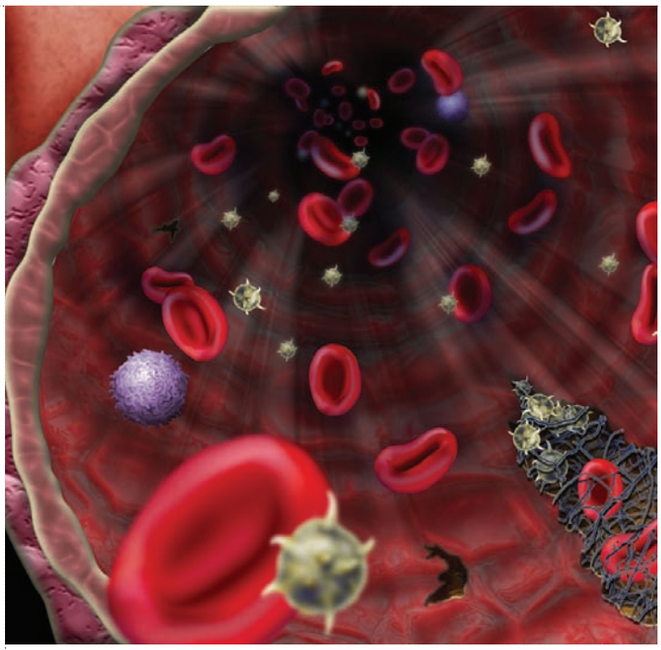
## Haemoptysis

Haemoptysis is a common but non-specific symptom with many possible causes, from an uncomplicated pneumonia to being the first and, occasionally, the only presenting symptom of lung cancer. In up to one-third of cases, no cause is found after investigations. Unless there is a clear cause, all cases of haemoptysis require investigation.

| **Haemoptysis in a smoker should always raise the suspicion of malignancy. The usual clinical differential lies between:** |
|---|
| • Malignancy |
| • TB |
| • PE |
| • Bronchiectasis |

**Table 1: Common differential diagnoses.**

The most common differential in clinical practice is shown in Table 1. A more extensive list of causes is shown in Table 2 and separated into common and rare. We will concentrate on the common causes.

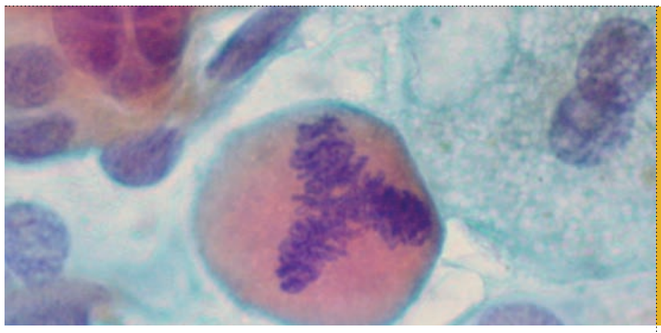


| **Common** |
|---|
| Pneumonia/acute bronchitis |
| Exacerbation of COPD |
| Bronchiectasis |
| Carcinoma of the bronchus |
| Pulmonary tuberculosis |
| Pulmonary embolus |
| Warfarin/anticoagulation |
| Rupture of mucosal vessel (e.g. coughing) |
| **Rare** |
| Vascular abnormalities (AVM) |
| Mycetoma |
| Lung abscess |
| Vaculitis (Wegener's, SLE) |
| Goodpasture's syndrome |
| Mitral valve disease |
| Severe pulmonary hypertension |
| Cystic fibrosis |
| Aspergillus |

**Table 2: Common and rare.**

# CASE BASED DISCUSSION: HAEMOPTYSIS

Ronan Astin and Himender K Makker



**Tumour can be benign (e.g. carcinoid) or malignant. Good Clinical Care.**

## Causes

**Bronchial tumour** – a history of small volume haemoptysis may be the only presenting symptom of a tumour. Tumour can be benign (e.g. carcinoid) or malignant. Associated symptoms of chronic cough, weight loss, smoking history, etc. with signs on examination of cachexia, clubbing and lymphadenopahty, increases the suspicion of malignancy. Haemoptysis due to neoplasia increases with age, accounting for approximately 20% of cases among the elderly.

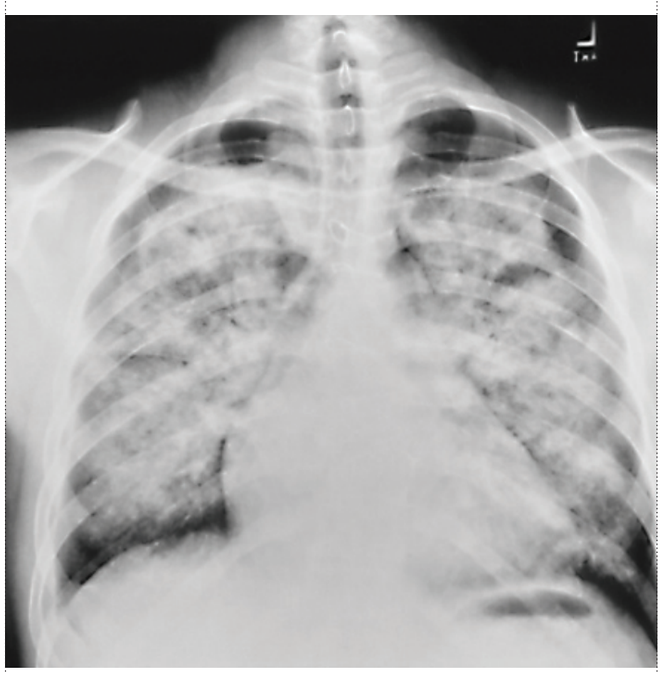
**Pneumonia** – usually a short history of haemoptysis in association with a productive cough and evidence of infection (pyrexia, raised serum inflammatory markers, consolidation on the chest X-ray). Often caused by pneumococcal infections. Blood-tinged sputum in the setting of an upper respiratory tract infection in an otherwise healthy, young (age <40 years) non-smoker does not warrant an extensive diagnostic evaluation if the haemoptysis subsides with resolution of the infection.

**Bronchiectasis** – suspect in a patient with recurrent episodes of "bronchitis" over several years prior to presentation. Usually chronic cough productive of sputum, recurrent chest infections and airflow obstruction (wheeze). Often small volume, intermittent haemoptysis, but can occasionally be massive. Caused by proliferation of blood vessels around bronchiectatic cavities and erosion into an arteriole wall and can produce massive haemoptysis (see later).

**Tuberculosis** – usually patients present with chronic cough, often productive, associated with night sweats, fevers and weight loss. Look for lymphadenopathy on examination and usually there is evidence of infection on the chest X-ray. Haemoptysis in TB increases the likelihood of smear positive ("open") disease and if admitted to hospital, the patient should be nursed in a negative pressure side room. If a negative pressure side room is not available, the patient should be nursed in a side room with barrier precautions according to local policy for suspected tuberculosis. Where there is the possibility of multi-drug resistant tuberculosis then advice should be sought immediately from a microbiologist and/or chest physician, and consideration given to transferring the patient to a unit with appropriate negative pressure facilities.

**Pulmonary embolism** – usually a short history of breathlessness and small volume haemoptysis. May have associated pleuritic chest pain; tachycardia; cardiac arrhythmia; hypoxia; or hypocapnia on arterial blood gas measurement and associated ECG changes of PE. Chest X-ray may be normal or show evidence of atelectasis, pleural effusions or oligaemia (in massive PE). Small volume haemoptysis is not a contraindication for therapeutic anticoagulation.

**Vasculitis/alveolar haemorrhage syndromes** – the most common of these is Goodpasture's syndrome, followed by Wegener's granulomatosis, SLE, Churg-Strauss and Polyarteritis Nodsa. In Goodpasture's, an antibody against the glomerular basement membrane reacts with the pulmonary alveolar basement membrane and causes alveolar haemorrhage. The history is usually no more than a few weeks, with generalised symptoms of lethargy, tiredness and difficulty in breathing. Often there will be renal involvement and a urine dipstick may show protein or blood and in cases of pulmonary haemorrhage, a chest X-ray may show bilateral alveolar shadowing.



**Pulmonary haemorrhage in Goodpasture's syndrome.**

# CASE BASED DISCUSSION: HAEMOPTYSIS

Ronan Astin and Himender K Makker

## Investigations

Small volume haemoptysis can usually be investigated on an (urgent) outpatient basis.

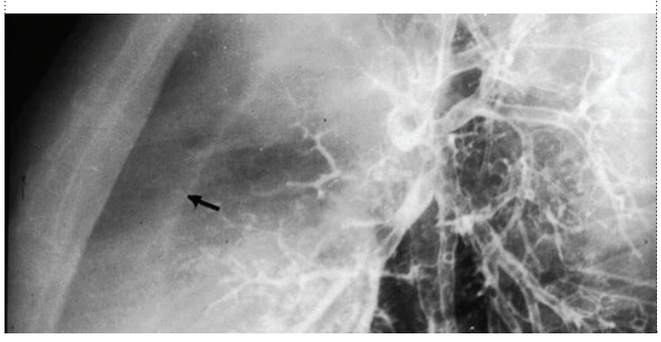
Most cases of haemoptysis that have no visible cause on CT scan or bronchoscopy will resolve within 6 months without treatment, with the notable exception of patients at high risk for lung cancer.

**First line investigations:**

- Blood tests – FBC, clotting, renal function, inflammatory markers with ESR, group and save, vasculitic screen (ANCA, ANA, GBM), if vasculitis suspected.
- Sputum – send for MCS and also for AFB smear and culture. Three sputum samples should be sent for AFBs.
- Urine – dipstick for protein and blood. If positive for protein, further quantification should be carried out. Send for microscopy, looking for red cell casts suggestive of vasculitis.
- Chest X-ray – may show focal area of parenchymal abnormality or consolidation, mass lesion, lymphadenopathy, Arterio-venous malformation or alveolar shadowing suggestive of pulmonary haemorrhage.
- CT chest – should be done prior to bronchoscopy so that the pick up rate endoscopically is increased should an abnormality be identified radiologically. May diagnose the cause, such as an AVM.
- Bronchoscopy – may endoscopically identify bleeding point, may allow biopsy of mass. In some cases may be therapeutic (particularly rigid bronchoscopy in massive haemoptysis – see later).

**Second line investigations are:**

- CTPA – if PE suspected.
- Bronchial angiography – most sensitive during an episode of bleeding, though not readily available. May identify likely bleeding site even if bleeding point is not directly seen.
- Bronchial artery embolisation – only available in specialist centres, aimed at embolise the bleeding vessel.
- ENT review – to rule out upper airway bleed.
- ECHO – to rule out mitral valve disease and pulmonary hypertension – moderate to severe hypertension can lead to bleeds especially since many such patients are anticoagulated.



## Dealing WIth Massive haemoptysis

Haemoptysis is considered massive if 100–600ml are expectorated in 24 hours. It is a medical emergency with a mortality of up to 80%, though fortunately is a rare occurrence. Causes of massive haemoptysis are bullet pointed below. Massive haemoptysis can be usefully defined as any amount that is haemodynamically significant or threatens ventilation, in which case the initial management goal is not diagnostic but therapeutic.

**Causes of massive haemoptysis:**

- lung carcinoma
- bronchiectasis
- mycetoma of lung cavities (e.g. aspergilloma)
- tuberculosis
- lung abscess
- foreign body
- trauma

**Assessment along ABC algorithm should be used:**

- Airway protection and ventilation; if compromised, anaesthetic help should be sought immediately since intubation may be necessary in order to secure the airway and aid lavage. High flow oxygen can be administered and suctioning may aid in clearing the airway. Protected the good lung from blood draining into it from the affected lung may be achieved by lying the patient on the affected side (if known).
- Assessment of cardiovascular stability; blood pressure and heart rate measurement; tachycardia of >100bpm or hypotension (systolic BP <90mmHg) are warning signs of a potentially massive haemoptysis and fluid resuscitation should be instituted with a volume expander as in other cases of circulatory compromise though wide bore cannulae. Blood should be taken for crossmatch and clotting.
- Reverse deranged clotting; contact haematology for advice; will usually require FFP and vitamin K.
- Nebulsied adrenaline.
- Oral tranexamic acid.
- Chest X-ray and CT chest (+/- CTPA).
- Early Bronchoscopy, if the patient is stable enough (rigid bronchoscopy, if available) may allow identification of bleeding point, adrenaline injection or tamponade.
- CT angiography; can identify bleeding point either through visualisation during bleeding episode or implied by tortuous vessels around likely site of bleed.
- Bronchial artery embolisation; specialist centre only.
- Surgery; resection of bleeding lobe (only as a final option).

# CASE BASED DISCUSSION: HAEMOPTYSIS

Ronan Astin and Himender K Makker

## Assessment Questions

**1. A 62-year-old gentleman presents to A&E with a 1-week history of blood tinged sputum. He tells you that he coughed up flecks of blood mixed with phlegm spontaneously and denies any epistaxis or haematemsis. He is known to have COPD and AF, and among his medication he tells you he is on warfarin. His chest X-ray is normal. His BP is 140/85; HR 78; sats 96% on air. Initial blood tests show Hb 14.3; WCC 6.3; CRP<3; and INR is 4.1. What would you do?**

**a.** Admit under the medical team and request an urgent CT thorax.
**b.** Admit for bronchoscopy, once INR normalised.
**c.** Reassure and send home for GP follow-up.
**d.** Discharge home and organise an urgent outpatient CT thorax.
**e.** Discontinue warfarin.

**2. A 32-year-old lady presents to A&E with a 1-week history of shortness of breath after flying to the UK from East Africa. She remembers coughing up flecks of blood for a few days at the beginning of this period and has had a non-productive cough since. She is on no medications other than the oral contraceptive pill. On examination her chest is clear on auscultation. Heart rate 102, BP 136/78. Chest X-ray shows blunting of the right costophrenic angle only. Full blood count shows tests show Hb 12.6 and WCC 9.4. An ABG shows pH 7.53; pCO2 3.2; pO2 12.3; BE 2.1; HCO3 22. What is the most likely diagnosis?**

**a.** Pulmonary Tuberculosis.
**b.** Pulmonary embolism.
**c.** Pneumonia.
**d.** Lung carcinoma.
**e.** Nothing.

**3. You are called to A&E to see a 54-year-old gentleman who gives a history of coughing up a cupful of fresh red blood 1 hour ago. He is known to the chest department with a diagnosis of bronchiectasis, has been recently well and is not on anticoagulants. On initial assessment his heart rate is 120bmp, BP 90/64, SaO2 96% on air and breathing spontaneously. What is your next step?**

**a.** Organise a chest X-ray.
**b.** Take blood for FBC, renal function and clotting.
**c.** Cannulate with two wide bore cannulae, taking blood and commence IV gelofusin.
**d.** Dipstick urine.
**e.** Reassure and discharge home.

## Answers

**1. Answer: d.**
This gentleman presents with small volume haemoptysis and is apparently cardiovasculalry stable. He can therefore be investigated as an outpatient. Although the haemoptysis may be caused by his warfarin therapy, the fact he has COPD suggests a significant smoking history which raises the suspicion of malignancy. A normal chest X-ray should not deter from further investigation. He warrants a CT thorax and upper abdomen and likely will need a bronchoscopy, both of which can be carried out on an urgent outpatient basis.

**2. Answer: b.**
Although TB should always be considered in patients from high-risk areas presenting with respiratory symptoms and chest X-ray findings suggesting a pleural effusion would be consistent with a diagnosis of pulmonary TB, the short history with risk factors for PE (long haul flight, oral contraceptive pill) with tachycardia and a blood gas suggesting hyperventilation (hypocapnia) must make PE the most likely diagnosis.

**3. Answer: c.**
This gentleman presents with a significant bleed and cardiovascular compromise suggesting massive haemoptysis. Although investigations including blood tests, chest X-ray and urine all need to be done, the priority is initial resuscitation. After ensuring the airway and breathing are not compromised, IV access should be gained and fluid resuscitation commenced without delay.

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# SMOKING CESSATION

Burhan Khan and John Rees

**A 52-year-old accountant has recently been diagnosed with chronic obstructive pulmonary disease (COPD). He has smoked 20 cigarettes per day (cpd) since he was 18 years old.**
**Good Clinical Care.**

A 52-year-old accountant has recently been diagnosed with chronic obstructive pulmonary disease (COPD). He has smoked 20 cigarettes per day (cpd) since he was 18 years old.

## What would you like to do next?

The single most important thing a health care professional can do to improve the health of their patient is to help them quit smoking. The advice should be clear, for example, "I think it is important for you to stop smoking now. Cutting down is not enough"; strong, for example, "As your doctor, I need you to realise that to stop smoking is the most important step you can take to protect your current and future health"; and personalised, for example, "It is cigarette smoking that has resulted in the damage to your lungs. If you continue smoking, your breathing and lungs will continue to get worse."

The next step consists of assessing whether the patient is willing and ready to make a quit attempt. If willing, then provide assistance in the form of helping to form a quit plan, encouraging nicotine replacement therapy and offering key advice, and support. If the patient is not willing, then further educating and motivation is required.

## What treatment options would you consider?

Two kinds of interventions are available: psychological; and pharmacological treatments. Non-pharmacological approaches, such as individual or group counselling and cognitive behavioural therapy, are used to address the important role of behavioural conditioning which is not completely addressed using pharmacotherapy. Other approaches include hypnosis and acupuncture, albeit evidence is lacking that they increase the likelihood of sustained success. Self-help programmes, telephone counselling, computer technology and exercise all have been proven to help. There is a range of pharmacological agents to help smokers quit including NRT, antidepressants and nicotine receptor partial agonists.

## What are the benefits of stopping smoking?

The benefits of stopping smoking are considerable. Stopping smoking is undoubtedly the single most important step a person can take to improve their well-being. Quitting smoking can prevent tobacco-related disease within only a few years of cessation: it halves the risk of lung cancer; attenuates lung function decline; improves reproductive health; halts the occurrence of various heart diseases; and ameliorates mental health problems. Ex-smokers are less likely to suffer from post-operative complications. The advantages are also transferred to their surroundings, for instance, environmental and partners are less likely to develop cancer than those who live with continuing smokers. The pecuniary benefits are also significant.

## Introduction

Worldwide, over a billion people smoke. In the last century alone the tobacco epidemic killed 100 million people globally. At present, with 5.4 million deaths every year, the 21st century could see the toll reach 1 billion deaths[1]. In the UK alone, from 1950–2000, approximately 6.3 million people died from smoking. Currently 28% of adults in the UK smoke but the overall prevalence has remained essentially static over last 10 years. Every year there are 120,000 deaths, 365,000 hospital admissions and 1.2 million GP consultations; all related to tobacco. Smoking reduces a smoker's life expectancy by an average of 10 years[2], around 12 minutes for every cigarette smoked.

In more recent years the tobacco companies have been focusing their efforts on developing countries, opening up new markets and economies; targeting hundreds of millions of potential new smokers. So though tobacco-attributable deaths are projected to decline by 9% between 2002 and 2030 in high income countries, they are to double from 3.4 million to 6.8 million in low and middle income countries.

# SMOKING CESSATION

Burhan Khan and John Rees

## Smoking and health

Tobacco kills 1 person every 6 seconds[3]. It remains the most important cause of preventable morbidity and early mortality in the world today. It is the only legal consumer product that can harm everyone exposed to it – and it kills up to half of those who use it as intended.

Tobacco in all its forms is harmful, for instance, cigarettes, smokeless, bidis, water pipes, etc. and adversely affects nearly every part of the body. The different varieties of tobacco all have one thing in common: nicotine, an alkaloid that mediates its effects by the release of dopamine, a reward neurotransmitter. Cigarettes, as opposed to other forms of tobacco, have become popular because they are designed to be particularly efficient nicotine delivery devices: from the alveoli into the bloodstream and via the pulmonary veins to the heart, nicotine reaches the brain in less than 20 seconds. There is unequivocal conclusive evidence of the harmful effects of tobacco whether it be first, second or third hand[4].

## Approaches to smoking cessation

Most smokers would like to stop smoking, but few can do so without help. Only 1% of smokers stop with willpower alone. Over 70% of smokers see a physician each year, but only 20% are encouraged to quit! A medical visit is when health is salient and smokers may be more receptive and motivated. All health professionals should take this opportunity to discuss smoking with patients, be able to offer accurate advice on forms of assistance available, including medications, help lines, self-help materials and specialist services, and encourage smokers to use effective forms of assistance and refer to the smoking cessation service as much as possible. The five steps for helping smokers quit are outlined in Table 1.

However, quitting is not easy and more than 70% of adult smokers have made at least one attempt. Brief opportunistic advice from a medical professional - even as little as 60 seconds - may trigger a quit attempt in 40% of cases. However, more intensive strategies are clearly more effective. In order to use consultation time effectively, it is necessary to understand the natural history of quitting and utilise use of smoking cessation services sensibly, alongside the correct use of pharmacotherapy. Identification of individual characteristics that predict success in smoking cessation is highly desirable, as this could help match smokers with the most effective cessation strategy and identify those who might need more intensive treatment. The predictors of smoking cessation include[5]:

**1. Gender:** many studies suggest that men have better long-term outcomes. It is possible that an interaction between gender and other factors is important in determining the outcome of smoking cessation and could explain this discrepancy. These include concerns of weight gain and higher rates of depression where women are more likely to use smoking as a means of handling negative emotions.

**2. Age at smoking initiation:** it is proposed that early exposure to tobacco could detrimentally affect a developing brain, leading to greater nicotine dependence later in life. It has been convincingly demonstrated that those who start smoking at age <14 are more likely to become heavy smokers than those who start age 20[6].

**3. Depression:** the association between nicotine dependence and affective disorders, especially depression, is well known but not clearly understood. However, a history of depression is not a barrier to smoking cessation. This group of patients is likely to experience intense withdrawal symptoms and thus may benefit from intensive pharmacological treatment and consider using antidepressants or referral to a specialist. To systematically ascertain the presence of depression, validated questionnaires are available, but the simple question "Did you feel down during most days of the past 2 weeks?" has also been found to valid[7].

**4. Nicotine dependence:** the severity of nicotine dependence is inversely proportional to successful cessation and is usually assessed by means of the Fagerström Test for Nicotine dependence[8] (FTND) Table 2; severe dependence score >7. This identifies those smokers who require high dose pharmacotherapy, as they are likely to experience more intense withdrawal symptoms, may relapse early and may require multiple attempts. The two most important questions are a) the time to the first cigarette in the morning, and b) the number of cigarettes smoked daily; albeit the first question alone can be used as a proxy. Those who smoke their first cigarette with 1 hour of waking and smoke more than 10–15 cigarettes per day are significantly addicted to nicotine[9].

**5. Alcoholism:** this is a negative prognostic factor and stopping drinking is likely to increase successful smoking cessation. In these patients, intense programmes including behavioural therapy for smoking cessation have been shown to be effective.

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**6. Motivation:** rating motivation on a scale of 0–10 can be very useful to assess willingness by asking "How important is it for you to give up smoking?" and perceived self-efficacy by asking "If you were to decide to stop smoking, how confident are you that you would succeed?" If readiness to quit is high but self-efficacy low, treatment and support are critical for success. If self-efficacy is high, but willingness is low, effective health education is needed. If both variables are high, a quit date can be set imminently; but if both are low, then motivation and self-efficacy need to be built up.

**7. Previous cessation attempts:** a history of previous cessation attempts has been consistently shown to predict smoking cessation; those with an attempt lasting >5 days are more likely to succeed. Moreover, exploring previous relapses helps identify ways to prevent future relapses.

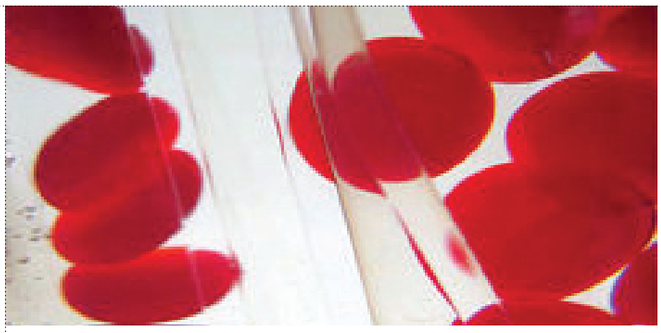
**8. Social/family environment:** occupational social class, number of smokers in the household, marital status and level of support from family members are important predictors. Those married to non- or ex-smokers and with supportive spouses are more likely to be successful.

## Pharmacotherapy

A number of non-pharmacological smoking cessation strategies exist, for instance, willpower, alternative therapies, brief advice and behavioural support in small groups or individually. But pharmacotherapy in conjunction with behavioural intervention is the cornerstone in the treatment of tobacco dependence. It includes nicotine replacement therapy (NRT), bupropion, and second line treatments like varenicline[10]. Except in the presence of contraindications, these drugs should be used in almost all patients attempting to quit smoking. In those smokers not currently motivated or able to quit, a smoking reduction strategy also has a role as a gateway to complete cessation; albeit there is scarce data of health benefits.

When nicotine is inhaled and absorbed it attaches to nicotinic acetylcholine receptors and causes dopamine release in the mid-brain. After repeated ingestion of nicotine, the motivational system is altered to create a "drive", somewhat similar to hunger, except for cigarettes. This drive is experienced as a need to smoke and increases in the minutes to hours since the last cigarette and is influenced by triggers, reminders, stress and distractions. It usually reduces over weeks of not smoking but can re-emerge unexpectedly. After repeated nicotine exposure, abstinence results in unpleasant withdrawal symptoms including depression. Nicotine substitution can relieve some of the need to smoke by raising the tonic depression of nicotinic acetylcholine activity; they can do this without themselves being addictive.

NRT is available in various forms (see Table 3), is effective (overall odds ratio versus placebo of 1.8) and well tolerated. There is little evidence that one NRT is more effective than another and choice should be guided by individual preference. In smokers requiring more intensive treatment, NRT can be used in higher doses or in combination. Combination NRT has been reported to improve outcome, but long-term results are conflicting.



## The future

Several newer agents are being developed. Cytisine (Tabex®), a natural insecticide present and a partial agonist of nicotinic acetylcholine receptors and has been used for several decades in Eastern and Central European countries. Nicotine vaccines that work by producing antibodies that bind to nicotine and prevent it from crossing the blood-brain barrier may be effective in preventing relapse in recent ex-smokers as well as helping current smokers to quit. Early (phase II) trials are underway of two vaccines; NicVAX (Nabi, Florida, USA) and NicQb (Cytos, Zurich, Switzerland). Biomarkers can directly or indirectly measure smoke products or by-products in body tissues that provide an objective indication of the extent of smoke intake over a defined period. They can serve as motivational and monitoring tools.

## Conclusion

There are a number of guidelines available[5,11,14] but despite the clear benefits of helping smokers to quit, nicotine addiction is undertreated. Whether this reflects physicians' indifference or scepticism towards the efficacy of smoking cessation programmes or reflects the lack of understanding of the natural history of cessation, underuse of smoking cessation services, improper use of drugs or lack of awareness is not clear.

If all GPs advise 50% of smokers to stop in 1 year, it would entail 20 hours per GP per year but would result in at least 55,000 additional ex-smokers. Moreover, if an additional 25% of those made a quit attempt using NRT or bupropion, an additional 27,000 additional ex-smokers would ensue; and if an additional 5% of those made a quit attempt by attending a specialist service, an additional 16,000 ex-smokers. If smokers' clinics recruit an additional 0.5% of smokers wanting to stop but not attending a GP, an additional 2,900. The NHS could therefore create 100,900 ex-smokers in a year!

The current strategies are working but progress is too slow, particularly among the less well off members of the community. In the meanwhile smoking will continue to kill. Each percentage point reduction in prevalence will eventually save almost 4,000 lives per year.

## Useful links

*www.nhs.uk/smokefree*
*www.nice.org.uk*
*www.nosmokingday.org.co.uk*
*www.ash.org.uk*

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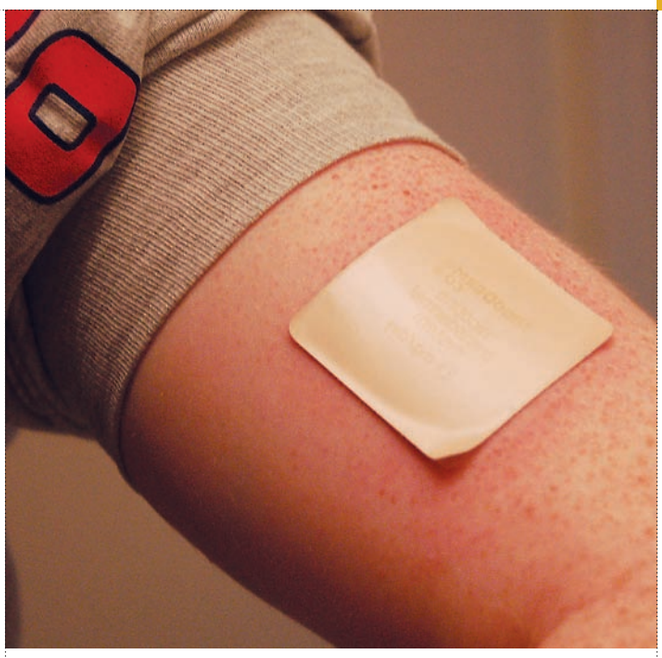
| | Action | Strategy |
|---|---|---|
| **1. Ask** | Systematically identify all tobacco users at every visit. | Vital signs should be expanded to include tobacco use. |
| **2. Advise** | Strongly urge all smokers to quit. | Advice should be:<br>• Clear<br>• Strong<br>• Personalised. |
| **3. Assess** | Identify smokers willing to make a quit attempt. | If the patient is motivated to attempt quitting, provide assistance.<br>If not willing to make a quit attempt, provide motivational intervention. |
| **4. Assist** | Help the patient with a quit plan. | Encourage NRT.<br>Set a quit date.<br>Inform family, friends and co-workers.<br>Prepare the environment.<br>Review previous quit attempts.<br>Anticipate challenges. |
| | Key advice. | Total abstinence from smoking is essential.<br>Drinking alcohol is associated with relapse.<br>The presence of other smokers in the household is associated with lower success rates. |
| **5. Arrange** | Schedule follow-up contact. | Follow-up visit soon after quit date.<br>If smoking has recurred, review the circumstances and identify problems to anticipate in future quit attempts. |

**Table 1: The five A's.**



| Question | Response | Score |
|---|---|---|
| 1. How soon after you wake up do you smoke your first cigarette? | Within 5 min<br>6–30 min<br>31–60 min<br>After 60 min | 3<br>2<br>1<br>0 |
| 2. Do you find it difficult to refrain from smoking in places where it is forbidden? | Yes<br>No | 1<br>0 |
| 3. Which cigarette would you hate most to give up? | The first one in the morning<br>Any other | 1<br>0 |
| 4. How many cigarettes per day do you smoke? | <10<br>11–20<br>21–30<br>> 31 | 0<br>1<br>2<br>3 |
| 5. Do you smoke more frequently during the first hours after waking than during the rest of the day? | Yes<br>No | 1<br>0 |
| 6. Do you smoke if you are so ill that you are in bed most of the day? | Yes<br>No | 1<br>0 |
| **Total score (0–10)** | | |

**Table 2: Fagerström Test for Nicotine dependence (FTND)[8].**

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| | Advice | Dosage |
|---|---|---|
| **First Line** | | |
| Nicotine patch | To be used with caution in pregnancy and in cardiovascular diseases. Patients should refrain from smoking while on the patch. At the start of each day, a new patch should be placed on a new relatively hairless location between the neck and the waist. Local skin reactions are common but usually mild and self-limiting. | Nicorette®<br>First 8 weeks - 15mg/16hrs<br>then 2 weeks - 10mg/16hrs<br>then 2 weeks - 5mg/16hrs<br><br>Nicotinell ®<br>Smoker <20cpd - 14mg/24hrs<br>Smoker >20cpd - 21mg/24hrs<br><br>Reduce dose every 3–4 weeks |
| Nicotine gum | See above for cautions. Adverse effects include mouth soreness, hiccups, dyspepsia, and jaw ache; but are generally mild and transient. | Available in 2mg (for patients who smoke less than 25 cigarettes per day (cpd), max 30 pieces per day) and 4mg (for those who have failed with 2mg or smoke more than 25 cpd; max 20 pieces per day). |
| Nicotine spray | | 500 mcg/metered spray, apply 1 spray into each nostril as required to maximum twice an hour for 16 hours daily (max 64 sprays/day). |
| Nicotine inhaler | Nicotine-impregnated plug. | 10mg/cartridge, inhale when urge to smoke occurs, initially between 6 and 12 cartridges daily for up to 8 weeks, then half the number of cartridges over 2 weeks and then stop in 2 weeks. |
| Bupropion (Zyban®) | Inhibits neuronal reuptake of dopamine. Contraindicated in patients with a history of seizures, eating disorders, CNS tumour, recent MAOI use, or who are experiencing acute symptoms of alcohol or benzodiazepine withdrawal. | Start 1–2 weeks before quit date, initially 150mg daily for 6 days, then 150mg twice daily for 7–8 weeks. |
| **Second Line** | | |
| Nortriptyline | Tricyclic antidepressant with anti-cholinergic side effects, cardiac conduction disturbances, and orthostatic hypotension. | 75–150mg/day. |
| Varenicline (Champix®) | Selective nicotine receptor partial agonist. | Start 1–2 weeks before quit date, initially 500mcg daily for 3 days, then 500mcg twice daily for 4 days, then 1mg twice daily for 11 weeks. |

**Table 3: Pharmacotherapy.**

## Questions

Answer true or false to the following questions.

**1. Cigarette smoking can cause the following conditions:**
**a.** Lung cancer.
**b.** Acute leukaemia.
**c.** Sarcoidosis.
**d.** Tuberculosis.
**e.** Bladder cancer.

**2. Symptoms after recently stopping smoking include the following:**
**a.** Insomnia.
**b.** Increased expectoration.
**c.** Headache.
**d.** Hallucinations.
**e.** Weight loss.

**3. Recommended pharmacotherapy for smoking cessation include the following:**
**a.** Nicotine patch.
**b.** Nicotine inhaler.
**c.** Varenicline.
**d.** Antidepressants.
**e.** Nicotine lozenges.

**4. General advice to patients who are attempting to stop smoking include the following:**
**a.** New year resolutions to quit smoking always fail.
**b.** One single cigarette will not lead to a relapse.
**c.** Set a quit date.
**d.** Inform family, friends and neighbours.
**e.** Stop drinking coffee.

**5. A patient states that he has previously quit smoking on four occasions with nicotine patches, for up to three months duration. Which of the following statements is correct:**
**a.** Indicates that the patient is likely to relapse again.
**b.** There is no point reviewing previous failed attempts.
**c.** Repeated quit attempts is common.
**d.** The most recent failed attempt was 2 weeks ago, an interval before the next quit attempt is advisable.
**e.** Nicotine patches should not ever be used again in this patient.

# SMOKING CESSATION

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## Answers

**1.**

**a. True. b. True. c. False. d. True. e. True.**

Among many diseases, cigarette smoking causes lung cancer and bladder cancer and more recently acute myeloid leukaemia. There is also evidence that smoking increases the risk of TB infection, as well as progression from latent TB to disease and increases the mortality. There is no association of smoking and sarcoidosis; the exact aetiology remains unknown.

**2.**

**a. True. b. True. c. True. d. False. e. False.**

Physical withdrawal from nicotine is temporary, but it can be uncomfortable while it lasts. Common withdrawal symptoms include cravings to smoke, irritability, insomnia, fatigue, inability to concentrate, headache, cough, sore throat, constipation, dry mouth, sore tongue and gums, postnasal drip and tightness in the chest. Increased expectoration is common as the ciliated cells lining the airways regenerate and thus an effective mucociliary escalator results in increased mucous clearance. Hallucinations are a symptom of either a medical (e.g. epilepsy), neurological, or mental disorder, with certain recreational drugs including amphetamines and cocaine, hallucinogens (e.g. lysergic acid diethylamide or LSD), phencyclidine (PCP), and cannabis or marijuana or withdrawal from alcohol and sedatives. Weight gain of 1 to 4Kg is common after stopping cessation.

**3.**

**a. True. b. True. c. True. d. False. e. True.**

All nicotine replacement agents are licensed and recommended by FDA and NICE. Pharmacotherapy is recommended in anyone attempting to quit smoking. Consider a combination of nicotine patches and another form of NRT (e.g. gum, inhalator, lozenge or nasal spray) to people who show a high level of dependence on nicotine or who have found single forms of NRT inadequate in the past. Varenicline or bupropion may be offered to people with unstable cardiovascular disorders, subject to clinical judgement. However, neither varenicline or bupropion should be offered to young people under 18 nor to pregnant or breastfeeding women. With the exception of bupropion and nortriptyline, other antidepressants are not effective smoking cessation agents.

**4.**

**a. False. b. False. c. True. d. True. e. False.**

Quit attempts made around special dates are more likely to be successful in the short term. Even a single cigarette may lead to a relapse. Setting a quit date is essential, and preparing for this includes informing family and friends of the intention to quit, and even finding a quit buddy has been shown to increases the chances of a successful quit attempt. Alcoholism is a negative prognostic factor, and even drinking in moderation can be problematic, due to the association of drinking and smoking. Coffee consumption has no impact on a quit attempt.

**5.**

**a. False. b. False. c. True. d. True. e. False.**

Contrary to the general belief that a history of repeated unsuccessful quit attempts precludes being able to stop smoking, cessation history has consistently been shown to predict smoking cessation. Both the number and duration of previous cessation attempts are important predictors of eventual long-term cessation. Attempts lasting for more than 5 days indicate more likelihood to succeed. Previous cessations should be used to boost motivation, because if a smoker has managed to quit before, it is more likely that they will be successful in the future. It is essential to review all previous failed attempts, in order to identify and anticipate barriers to the next attempt. NICE recommends a 6 month interval before another quit attempt. If one NRT has been unsuccessful, there is no point in using the same delivery route again, except if used in combination therapy or at a higher dose.

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# CYSTIC FIBROSIS

Kiran Bundhoo and Daniel Glassman

**A 1-year-old boy is brought by his mother to see his GP with a persistent cough, wheeze and foul smelling stools.**
**Good Clinical Care.**

A 1-year-old boy is brought by his mother to see his GP with a persistent cough, wheeze and foul smelling stools. The mother reports a history of recurrent chest infections since birth and the GP notes that the child is below the appropriate centile for weight gain. How would you further assess and manage this patient?

## Introduction

Cystic fibrosis (CF) is a commonly occurring genetic disorder affecting multiple organs through alteration of mucus production from exocrine glands, classically causing broncho-pulmonary and pancreatic dysfunction. It is an autosomal recessive condition that predominantly affects Caucasians. In the United Kingdom, 1 in 25 of the population are carriers of the genetic abnormality and in total approximately 8,000 people have the disease[1].

## Pathophysiology

The most common genetic abnormality causing CF is due to a mutation affecting a gene in the long arm of chromosome 7 that specifically encodes for the cystic fibrosis transmembrane regulator (CFTR) protein. The CFTR protein is involved in the cAMP regulated transport of sodium and chloride channels at cell surface membranes. The most common defect in the Caucasian population is a mutation causing deletion of a phenylalanine amino acid in codon 508. The resulting defective CFTR protein causes reduced chloride secretion and increased sodium and water reabsorption across the cell surface epithelium. Whilst this is the most common defect in Caucasians, more than 300 additional CFTR gene defects have been identified, some of which are more predominant in other ethnic groups. Within this number at least 230 mutations have been found to be associated with clinical disease[2]. The resultant increased water reabsorption causes the secretion of thicker and more viscous mucus within the respiratory system, gastrointestinal tract, pancreatic ducts and other exocrine glands. These viscoid secretions are more difficult to mobilise and increase the likelihood of bacterial colonisation, giving rise to infection and inflammation.

## What are the clinical features of CF?

As previously mentioned, CF has the potential to affect multiple systems within the body. Some of the most frequent presentations of CF are related to the effect of the disease on the respiratory system. As a result of the abnormal mucus secretions, patients suffer from a chronic cough that is productive of muco-purulent sputum. Recurrent episodes of pneumonia, or bronchiolitis in children, are often a common presenting feature and there is colonisation of the lungs with differing pathogenic bacteria. Initially, this takes the form of gram negative organisms, such as *Klebsiella* and *Escherichia coli*, and other common respiratory pathogens including *Staphylococcus aureus* and *Haemophilus influenza.* As disease progression occurs one develops mucous gland hypertrophy due to viscoid mucous hypersecretion. This results in recurrent inflammation within the lung parenchyma giving rise to a different environment colonised by other bacteria, such as *Pseudomonas aeruginosa* and *Stenotrophomonas maltophilia.* These organisms give rise to multi-drug resistance and are pathognomic of CF. Fungal colonisation also commonly occurs and infection with *Aspergillus fumigatus* has the potential to cause allergic bronchopulmonary aspergillosis. In the latter stages of CF, the chronic accumulation of viscoid secretions and inflammatory changes within the lung causes permanent bronchial dilatation and thus bronchiectasis. The inflammatory effects also increase airflow limitation leading to haemoptysis, further shortness of breath with respiratory failure, pulmonary hypertension and eventual cor pulmonale. Chronic damage to the lungs can also lead to spontaneous pneumothoraces and finger clubbing is an important clinical sign that is present in the majority of CF sufferers.

Gastrointestinal features of CF are also an important manifestation of disease. This is primarily a result of pancreatic insufficiency caused by mucus secretions blocking pancreatic exocrine duct function. The resulting enzyme deficiencies produce malabsorption of fat, protein and other nutrients leading to a consequential growth and development delay in children and therefore a failure to thrive. Clinically, there is steatorrhoea, abdominal distension, recurrent cramping abdominal pain and flatulence. The fat-soluble vitamins (A, D, E and K) are also not absorbed causing additional systemic manifestations of cystic fibrosis. One such manifestation is CF related bone disease caused by the failure to absorb vitamin D. Vitamin D is vital in the regulation of calcium and phosphorus in bone mineralization and therefore a deficiency may lead to bone pain and eventual rickets in children or osteomalacia in adults. Pancreatic endocrine function is also occasionally compromised leading to a form of diabetes mellitus resulting from blockage of insulin secretion from islets of Langerhans cells within the pancreas.

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An increased viscosity of meconium in neonates can lead to the more severe, but less common complication of meconium ileus, which involves the failure to pass motions due to impaction of stool within the distal ileum. A related condition that occurs in later life is distal intestinal obstruction syndrome, whereby stools adhere to the intestinal wall causing obstruction. Symptoms of this include abdominal pain, constipation and a palpable mass in the right lower quadrant on abdominal examination. Rectal prolapse may also arise in neonates due to bulky stools. Occasionally one can develop an appendiceal mass due to mucoid impaction and CF may be diagnosed upon the histological finding of a larger proportion of goblet cells with excess mucus within the mass. Mucoid impaction can also act as a lead point for intussusception. Hepatobiliary effects are commonly seen in CF with an increased level of cholestasis resulting in the formation of cholesterol gallstones. Hepatic duct secretions may also be thicker leading to obstruction of the bile ducts causing damage to hepatocytes and eventual hepatic cirrhosis.

CF can also cause infertility in men and sub-fertility in women. In the former, there is a congenital absence of the vas deferens or atresia of vas deferens leading to an obstructive azoospermia. In this situation, the production of spermatozoa is not usually affected, however, transport from the testes to penile ejaculatory ducts is impaired due to absent or defective bilateral vas deferens. Women are often sub-fertile due to a thickened cervical mucous causing an inhospitable environment for fertilisation. Amenorrhoea may also be a manifestation of CF due to severe malnutrition causing a disruption of ovulatory function.

| **Infancy** |
|---|
| • meconium ileus in newborn period |
| • prolonged neonatal jaundice |
| • failure to thrive |
| • recurrent chest infections |
| • malabsorption, steatorrhoea. |
| **Young child** |
| • bronchiectasis |
| • rectal prolapse |
| • nasal polyps |
| • sinusitis |
| **Older child and adolescent** |
| • diabetes mellitus |
| • cirrhosis and portal hypertension |
| • distal intestinal obstruction |
| • pneumothorax or recurrent haemoptysis |
| • aspergillosis |
| • sterility in males |

**Table 1: Summary of clinical features with relation to age[3].**

## What diagnostic investigations could be performed?

A variety of diagnostic investigations to confirm CF are available and these are sometimes used in combination. Commonly used investigations are: immunoreactive trypsin (IRT) levels in neonates; the sweat test; nasal potential difference testing; and genotype identification.

The pancreatic enzyme IRT is elevated in CF within the first few weeks of life and is a good screening test in neonates. By performing a neonatal heel prick test one can measure serum levels of the enzyme. Elevated serum titres suggest a diagnosis of CF and the test may be repeated to confirm this. A negative result, however, is disregarded after 8 weeks of life as the effects of CF causing pancreatic insufficiency begins to develop, which alters the accuracy of the test.

The sweat test remains the definitive investigation for diagnosing CF. Intradermal pilocarpine is injected and sweat is induced by iontophoresis. The chloride and sodium concentrations are then chemically determined. New techniques such as the macroduct system have been developed in order to improve the ease of testing. In macroduct testing, a closed capillary collecting system is applied to the skin of the forearm after the induction of sweating via the previously described method. This method greatly decreases the amount of sweat needed for testing and a formal diagnosis can be made from as little as 50µl of sweat[4]. Chloride concentrations greater than 60mmol/L and sodium greater than 70mmol/L is indicative of CF. One needs to be aware that elevated chloride and sodium sweat concentrations are also associated with: coeliac disease, adrenal insufficiency, hypothyroidism, anorexia nervosa and severe malnutrition. False-positive results may occur in 10-15% of cases and are usually attributed to inaccurate methodology when performing the test. In approximately 1% of patients with CF, often those with unusual genotypes, normal sweat chloride concentrations may be observed[5].


**Photo 1: Macroduct sweat testing using the closed capillary collecting system. Sweat is seen entering the tubing stained blue[4].**

# CYSTIC FIBROSIS

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Nasal potential difference testing is another diagnostic technique. This is usually only performed if the diagnosis of CF proves difficult by other methods, for example, due to cutaneous abnormalities deeming sweat testing to be technically complicated and the patient is too old to perform IRT testing. The nasal epithelium is particularly sensitive for measuring cell membrane electrolyte transport, with CF individuals recording greater transepithelial potential differences compared to individuals without CF. This is due to the difference in sodium and chloride transport across cell membranes as a result of CFTR protein mutations.

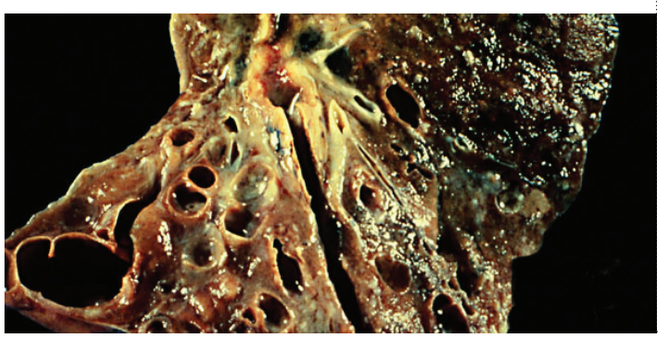
Genotype testing may be performed: prenatally, for example, in children from parent carriers of CF, if diagnosis is suspected but presentation is atypical, or diagnosis is still uncertain despite performing the above investigations. Over 1,600 CF mutations have been recognised[6]. These mutations are identified on the CFTR gene by nucleotide sequence analysis performed using varying methods. Due to the wide variety of genotype abnormalities, a negative analysis does not necessarily exclude the diagnosis of CF; however, a positive result is significant.

| What investigations would you perform to diagnose CF? |
|---|
| • Sweat testing |
| • Immunoreactive trypsin testing |
| • Nasal potential difference testing |
| • Genotype testing |

**Table 2**

| What other investigations could be performed to assess someone with CF? |
|---|
| • Chest radiograph (see Photo 2): hyperinflation; signs of bronchiectasis (peribronchial wall thickening, ring shadows); signs of pneumonia (lobar consolidation). |
| • Lung function testing. |
| • Sputum microscopy and culture. |
| • Aspergillus RAST and IgE levels. |
| • Malabsorption screening. |
| • Oral glucose tolerance test. |

**Table 3**



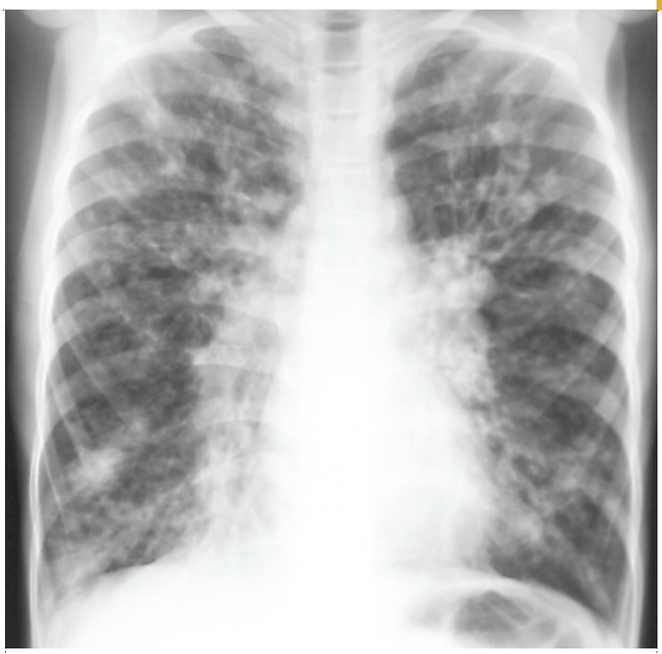


**Photo 2: A chest radiograph demonstrating diffuse interstitial disease, bronchiectasis and nodular densities indicating mucoid impaction in a patient with CF[7].**

## How would you manage a patient with CF?

The mainstay of effective CF management is via a multidisciplinary approach so as to achieve control of symptoms, particularly respiratory and nutritional aspects, limit progression of the disease and thus maintain a good quality of life. A range of different medical professionals are therefore required for optimal management of CF. These include: a general practitioner; paediatrician; dietician; nurses; physiotherapist; counsellors; and adult respiratory/nutritional specialists after childhood.

The treatment of respiratory disease is a fundamental part of managing CF patients and the most important aspect of this is dislodging accumulated secretions within the airways. Bronchial clearance is performed primarily by manual chest percussion and postural drainage, which should be conducted multiple times daily. This is initially carried out by physiotherapists, however, patients and their families are encouraged to learn and perform the techniques themselves. These methods all serve to mobilise and aid expectoration of mucus secretions. Frequent exercise is recommended as this has the ability to improve intercostal muscle strength and reduce the rate of mucus build up.

# CYSTIC FIBROSIS

Kiran Bundhoo and Daniel Glassman

Antibiotic therapy in the management of early stage CF is used as definitive treatment for acute lower respiratory tract infections. However, as the disease progresses and infections become more frequent, often regular prophylactic antibiotics are required. In these cases, acute infections are treated aggressively with additional intravenous antibiotics. Initially, as previously discussed, the pathogens are of the normal spectrum seen in patients without underlying CF. As the condition worsens, colonisation is predominantly by alternative organisms which are more difficult to treat and cause a more severe respiratory illness. Sputum samples are taken at regular intervals so as to monitor any change in respiratory pathogens and thus enable an appropriate alteration in therapy. With increasing age, Pseudomonas aeruginosa is the main colonising bacteria and coupled with the development of worsening bronchiectasis and deterioration in lung function, acute infections become more frequent. The necessity for regular intravenous antibiotic treatment can occasionally result in the placement of a subcutaneous port linked to a central venous catheter. This allows the self-administration of intravenous therapy for treatment of acute infection without the need for hospital admission. This in turn facilitates independence on the part of the patient and ensures an improved quality of life. With increasing obstruction of the airways, treatment with inhaled bronchodilators and steroids can be beneficial. Another treatment option that can be utilised are mucolytics, which can aid the expectoration of mucus. In severe cases where respiratory failure and cor pulmonale develop, medical intervention is of limited use and often surgical measures, such as heart-lung transplantation must be explored

As previously described, CF often causes severe pancreatic insufficiency and therefore nutritional supplementation is of the utmost importance. This should be managed by dieticians, paediatricians and nutritional specialists with appropriate education about their importance given to the patient and their family.

Enteric-coated capsules containing pancreatic supplements are taken with meals containing high doses of enzymes, such as lipase and trypsin. The capsule coating enables avoidance of acid degradation in the stomach. Therefore, the supplements can effectively reach the duodenum unchanged, where they would normally be secreted by exocrine ducts of a functioning pancreas. Prior to the specially designed enteric coating of capsules, H2 antagonists were prescribed in combination to help avoid stomach acid breakdown of the nutritional supplements. The doses of these supplements are managed by the multidisciplinary team according to the clinical response of the patient. One monitors a variety of factors and may decide to decrease dosage if, for example, there is appropriate weight gain (measured using weight centile charts in children) and reduction of steatorrhoea. The occurrence of of fibrosing colonopathy has been reported and is thought to be as a direct result of high dose pancreatic supplementation. One, therefore, has to be aware of this complication and give high doses with caution[8]. Fat soluble vitamin tablets (vitamins A, D, E and K) are also taken by patients to replace their deficit as a result of an insufficient pancreas. Due to the increased energy expenditure and malabsorptive state of CF patients, a high calorie diet overseen by a dietician is recommended.

## What are the other issues that must be discussed with regards to this patient?

The psychological aspects of CF must be considered alongside the physical effects of the illness. CF is a chronic disease requiring much daily intervention that may disrupt normal lifestyle causing much emotional distress for both the patient and their family. Counsellors and psychologists may therefore play an important role in disease management.

Parents with a child affected by CF should be offered genetic counselling and possibly prenatal diagnosis for future pregnancies. Due to the autosomal recessive inherited nature of the disorder, parents should be educated that there is a 1 in 4 probability of any future child also having the disease. Siblings without CF should also be made aware that they have a 2 in 3 chance of being a carrier of the genetic abnormality for any possible future pregnancy. Newborn testing for CF is offered routinely as part of the NHS national screening programme[9]. In addition, parents with either a family history of CF or an existing child with the disease may be offered prenatal screening. This is important as it enables diagnosis of CF to be made and early interventional management to be provided. This can take the form of appropriate early nutritional support and methods to delay the deterioration in respiratory function with a view to decreasing future morbidity.

## Questions

**1. By what mode is CF inherited?**

**a.** Autosomal dominant.
**b.** Autosomal recessive.
**c.** X-linked recessive.
**d.** X-linked dominant.
**e.** Mitochondrial inheritance.

**2. In the UK, what is the carrier frequency of the genetic abnormality resulting in CF?**

**a.** 1 in 4.
**b.** 1 in 10.
**c.** 1 in 25.
**d.** 1 in 100.
**e.** 1 in 1000.

**3. Clinical Features of CF. The following statements are true or false:**

**a.** Finger clubbing is a common sign.
**b.** The initial respiratory pathogen is *Pseudomonas aeruginosa.*
**c.** There is frequently deficiency of water soluble vitamins.
**d.** Meconium ileus is common in neonates.
**e.** All females are infertile.

# CYSTIC FIBROSIS

Kiran Bundhoo and Daniel Glassman

**4. Investigations in CF. The following statements are true or false:**

**a.** A negative pancreatic IRT result excludes diagnosis.

**b.** In the sweat test, a chloride concentration >60mmol/L is indicative of CF.

**c.** In the sweat test, sweat is induced by iontophoresis.

**d.** Chest radiograph often reveals bilateral pleural effusions.

**e.** A negative genotype analysis excludes a diagnosis.

**5. Treatment of CF. The following statements are true or false:**

**a.** Bronchial clearance is an important treatment.

**b.** Intravenous antibiotic treatment of respiratory infections is always required.

**c.** Heart–lung transplantation is commonly performed.

**d.** Pancreatic enzyme replacement tablets are degraded and absorbed within the stomach.

**e.** v. High dose pancreatic enzyme replacement can be associated with fibrosing colonopathy.

## Answers

**1. b.**

CF is an autosomal recessive condition common amongst Caucasians.

**2. c.**

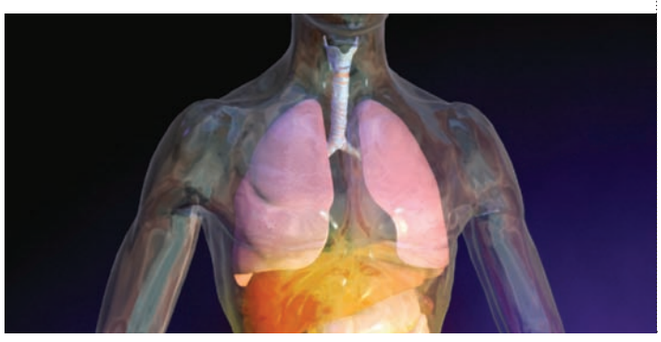
1 in 25 of the population are carriers of the genetic abnormality and in total approximately 8000 people have the disease in the United Kingdom

**3. a. – True, b. – False, c. – False, d. –True, e. – False.**

Finger clubbing is almost universal in chronic suppurative lung diseases. Pseudomonas aeruginosa is the colonising pathogen in the latter stages of disease. There is a deficiency of fat soluble vitamins due to a failure of absorption secondary to pancreatic duct dysfunction. Meconium ileus is a common presenting feature in neonates affected with CF. Almost all males are infertile, however, females demonstrate sub-fertility.

**4. a. – False, b. – True, c. – True, d. – False, e – False.**

After 8 weeks, a negative pancreatic IRT result does not necessarily exclude a diagnosis of CF as pancreatic insufficiency develops. In the sweat test, sweat is stimulated by iontophoresis. A chloride concentration of >60mmol/L and a sodium concentration of >70mmol/L is indicative of CF. Chest radiograph can show hyperinflation or signs of bronchiectasis (peribronchial wall thickening, ring shadows) and signs of pneumonia (lobar consolidation). A negative genotype does not always exclude a diagnosis due to the huge variety of possible mutations.



**5. a. – True, b. – False, c. – False, d. – False, e. – True.**

Bronchial clearance and physiotherapy plays a vital role in the expectoration of mucus secretions. Intravenous antibiotic treatment is only required in severe, acute lower respiratory tract infections. Heart-lung transplantation is only considered in severe, end-stage disease and due to a lack of suitable donors occurs infrequently. Pancreatic enzyme replacement supplements are administered in the form of enteric-coated capsules in order to protect from gastric degradation and ensure passage into the duodenum. There have been associations of fibrosing colonopathy with high dose pancreatic enzyme supplementation and this must be used cautiously.

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# AN UNUSUAL INFECTION PRESENTING AS A CHEST WALL MASS

Maria Silveira & Sue Wakeford

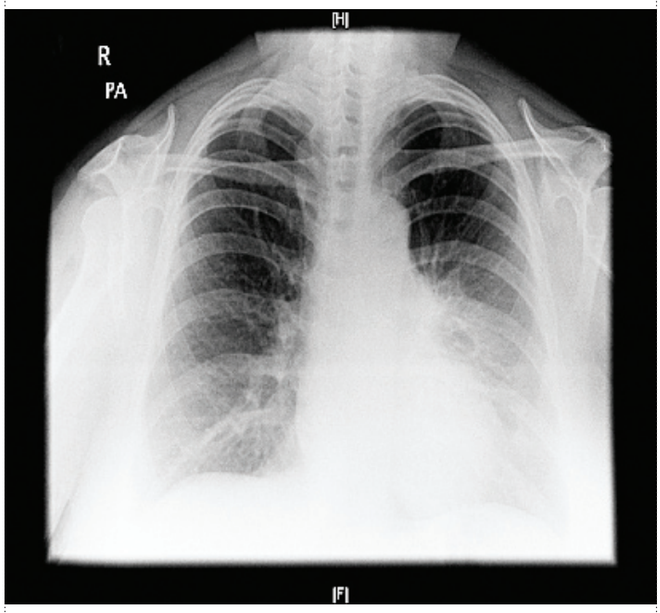
**A 54-year-old smoker was referred by her GP with a history of a painful lump in the left breast of 4 days duration.**
**Good Medical Practice.**

A 54-year-old smoker was referred by her GP with a history of a painful lump in the left breast of 4 days duration.

Past medical history consisted of type 2 diabetes, hypertension, chronic back pain and irritable bowel syndrome.

Examination revealed a tender hard fixed left chest wall mass 10cm x 5cm. Lung examination was clear bilaterally with no crackles or wheeze.

The chest X-ray shows a hazy opacity in the left hemithorax with obliteration of some of the pulmonary vessels.




Blood tests showed a white cell count of 15.4 with a neutrophilia of 9.3; a haemoglobin of 13.2; CRP of 121; LFTs were normal; and she was HIV negative.

CT of neck, chest and abdomen showed an 8cm lesion straddling the chest wall, just below the left sternoclavicular joint, and ''streakiness'' extending back down to the hilum. There was also some mediastinal lymphadenopathy.

CT guided biopsy was performed; there was no solid tissue within the lesion, but 8ml of thick green pus was aspirated.

Microscopy showed WBC+++. Gram positive bacilli on staining.

**Culture:** *1) actinomyces meyeri*
*2) Aggregatibacter aphrophilus*

Sensitive to amoxicillin and penicillin.

Surgical incision and drainage of the abscess was performed and, as the patient was allergic to penicillin, she was treated with clotrimoxazole and clindamycin for 2 weeks, and subsequently doxycycline for 12 months.

CT scan 2 months later showed the lesion had significantly reduced in size, with a track out to the chest wall forming a sinus.

## Discussion

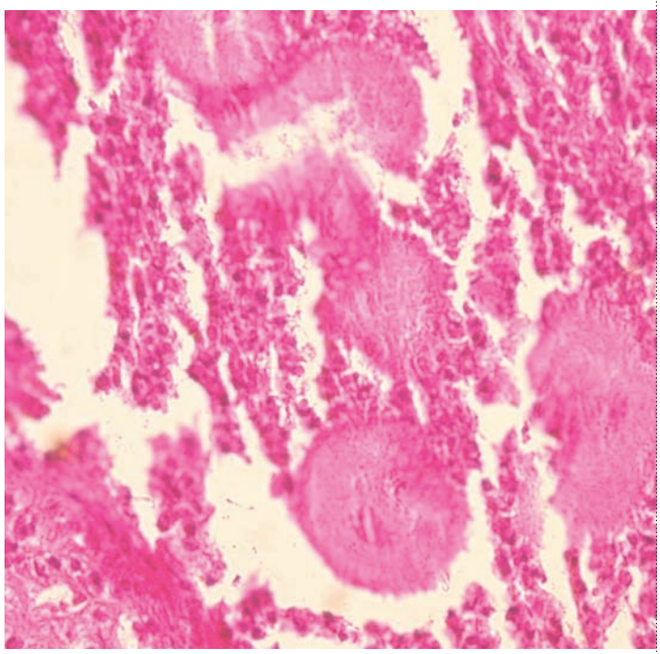
Actinomycosis is a chronic, suppurative, granulomatous infection caused by actinomyces species, most commonly *A.israelii.* The first case report in humans was in 1857, followed by the first case report of thoracic actinomycosis in 1882. The most common presentation is cervicofacial, presenting as a jaw mass, with thoracic actinomycosis accounting for between 15–50% of cases. The disease occurs in a 3:1 male to female ratio, possibly because of higher incidence of facial trauma. There is bimodal age distribution of 11–20 years of age, and 4th–5th decades of life. In the United Kingdom, patients with COPD and alcoholics, as well as those with poor oral hygiene are predisposed to actinomycosis. Actinomycosis organisms can be found in dental plaque and in the GI tract and aspiration and aerosolisation have been proposed as mechanisms of pulmonary disease.

# AN UNUSUAL INFECTION PRESENTING AS A CHEST WALL MASS

Maria Silveira & Sue Wakeford

The difficulty in diagnosis lies in the rarity of the disease, its radiological resemblance to malignant diseases, and the difficulty in identifying or culturing the organism (<50% success rate). The presence of sulphur granules, which are actually colonies of actinomyces, is suggestive of infective actinomycosis. Laboratory findings are non-specific, consisting of leukocytosis and elevated inflammatory markers. Common radiological findings of thoracic actinomycosis include wavy periostitis, pulmonary fibrosis, cavitation and pleural thickening. Pulmonary osteoarthropathy may also occur. Fine needle aspiration or Trucut biopsy can be used for tissue diagnosis. Video-assisted thoracoscopy may be useful for the diagnosis of peripheral lung or thoracic actinomycosis.

The first line treatment is medical with extended courses of intravenous penicillin for 2–6 weeks followed by oral penicillin or amoxicillin for 6–12 months. Doxycycline can be used as a substitute in penicillin allergic patients, according to National guidelines. Surgery has a role in managing complications, including abscess, empyema, fistula and haemoptysis. Haemoptysis due to actinomycosis has been found to have a 36.4% rate of rebleeding within 6 months of hospital discharge.



Early treatment leads to an excellent outcome with low mortality, but overall, pulmonary actinomycosis has a poorer prognosis than the disease in other locations.

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# BRONCHIECTASIS – NOT ALL COUGHS AND DYSPNOEA'S ARE COPD!

Ahsen Nazir, Nazir Khan & Damian John McKeon

**The first patient is Mrs IG, a 72-year-old lady, lifelong non–smoker. She was referred by her GP to the chest clinic with a history of a long-standing cough productive of large amount of muco-purulent phlegm and wheeze.**
**Good Medical Practice.**

We are presenting two different patients with similar pulmonary condition but with different underlying aetiologies. In one patient, the condition was diagnosed and then managed. The other patient was already known to have the condition and presented with one of its complications.

## Case reports

The first patient is Mrs IG, a 72-year-old lady, lifelong non–smoker. She was referred by her GP to the chest clinic with a history of a long-standing cough productive of large amount of muco-purulent phlegm and wheeze. Her past medical history was non-significant. Clinical examination and chest radiograph (chest X-ray) at that time was also unremarkable. Bronchiectasis was suspected and a high resolution computer tomography scan (HRCT) of her chest was organised. That showed widespread bronchiectasis. She was then investigated to find out the underlying cause for this condition. Her total IgE, RAST to aspergillus and immunoglobulin level were normal. Spirometry showed mixed obstructive and restrictive picture (FEV1 1.48, FVC 2.43, 50% and 63% predicted, FEV1/FVC ratio 0.60). Around the same time, she developed symptoms and signs that suggested rheumatoid arthritis, which is a recognised cause for bronchiectasis. Her anti-CCP antibodies (antibodies against cyclic citrullinated peptide is the most reliable biomarker for rheumatoid arthritis) were found to be positive at the level of 5.9U/ml. The rest of the autoimmune screen was unremarkable. In terms of management of her bronchiectasis, she was commenced on inhaled bronchodilator therapy and was referred to a chest physiotherapist. Her sputum culture at that time showed moderate growth of *Haemophilus Influenza* and *Pseudomonas Aeurginosa*. She was ill enough to need a couple of hospital admission during the last year because of infective exacerbation of her bronchiectasis that was successfully treated with intravenous antibiotics directed against pseudomonas. She was commenced on maintenance therapy in the form of colomycin nebuliser and oral flucloxacillin because she was colonised with *Pseudomonas Aeurginosa* and *Staphylococcal Aureus*. During this time she had frequent flare ups of her rheumatoid. She was tried on various combination therapies of "Disease Modifying Anti-Rheumatoid Drugs" (DMARDs). The choice was limited to DMARDs only as there is a significant risk of infection in her case with anti-TNF alpha agents due to immunosupression. She finally responded to a combination of lefluonamide and tacrolimus. She is under regular follow-up in chest and rheumatology clinic. Both of her conditions are well controlled at the moment.

The other patient is Mrs RG, a 40-year-old female, non-smoker, with a past medical history of obstructive airways disease and bronchiectasis (diagnosed on HRCT in 2004, which at that time showed bronchiectsis involving all lobes). The cause behind her bronchiectasis was recurrent lower respiratory infections in her childhood. She is known to have pseudomonal colonisation in her sputum. She presented with a couple of months history of tiredness; lethargy; increase in sputum volume; low appetite; weight loss of around 1 stone; and feeling hot at night. Clinical examination revealed early clubbing, widespread coarse crackles and around 1cm palpable left supraclavicular lymph node. Spirometry showed FEV1 0.94 and FVC 1.62 (36% and 53% respectively), which was a marked deterioration compared with her previous spirometry. Chest X-ray did not show any significant change in comparison to her previous X-rays. WCC was found to be raised at 20.4 _ $10^9$/L with marked neutrophilia and CRP was 49mg/L. The working diagnosis of infective exacerbation of bronchiectasis was made. The possibility of opportunistic mycobacterium and fungal infection was also kept in differential diagnosis. She was started on antibiotics directed against pseudomonas in the form of IV gentamicin and ceftazidime along with regular chest physiotherapy nebulised bronchodilator and oral steroids. Sputum samples were sent for Acid Fast Bacilli, bacterial and fungal culture. The Mantoux test (a most common used skin test to detect latent or recent TB infection) and quantiferon TB gold test (a blood test which measures Interferon-gamma (IFN-Ð) secreted from stimulated T cells previously exposed to *M. tuberculosis* to assess TB infection) was also done. Her sputum culture showed moderate growth of *Pseudomonas Auerignosa* sensitive to gentamycin and *Staphylococcus Aureus* sensitive to flucloxacillin, which was added to the treatment. The patient clinically responded well to antibiotics and CRP also came down according to its half-life of 19 hours. The other investigation's results did not show any evidence of TB or fungal infection. She was well enough to be discharged after 14 days of IVs.

We take this opportunity to discuss in detail the condition that both of our patients had.

## Bronchiectasis

Bronchiectasis is defined as irreversible abnormal dilatation of one or more bronchi with chronic airway inflammation. The exact prevalence of broncіectasis is unknown but is probably falling due to vaccinations and the improved and earlier treatment of childhood infections, such as whooping cough and measles.

# BRONCHIECTASIS – NOT ALL COUGHS AND DYSPNOEA'S ARE COPD!

Ahsen Nazir, Nazir Khan & Damian John McKeon

## Pathophysiology

An initial, usually infectious, insult damages the airways leading to secretion stagnation and secondary infection causing ongoing inflammation and further airway damage. Macroscopic appearance of bronchiectatic lung reveals permanent dilatation of subsegmental airways that are inflamed tortuous and often partially or totally obstructed with secretions. The process also includes bronchioles and at the end stage there may be marked fibrosis of small airways. In *Allergic Bronchopulmonary Aspergillosis,* the changes are predominately in proximal airways. Bronchiectasis caused by the most common inherited genetic disease to affect Caucasians, cystic fibrosis is likely to be more marked in the upper airways.

There is a spectrum of disease that ranges from cylindrical (where there is uniform dilatation) to saccular (where there is gross terminal dilatation of the bronchi). An intermediate form is known as varicose bronchiectasis.

| Causes Of Bronchiectasis | |
|---|---|
| Genetic | Cystic fibrosis<br>Alpha 1 antitrypsin deficiency |
| Congenital/ developmental defects | Williams Campbell syndrome – deficiency of the bronchial wall<br>Pulmonary sequestration<br>Mounier-kuhn syndrome – trachiobronchomegaly |
| Post-infective | Tuberculosis<br>Whooping cough<br>Measles<br>Bordetella pertusis |
| Immune deficiency | Primary-hypogammaglobulinemia<br>Secondary-HIV, CLL |
| Excessive immune response | Allergic bronchopulmonary aspergillosis<br>Post-lung transplant |
| Mucociliary clearance defects | Primary ciliary dyskinesia<br>Kartagner syndrome<br>Cystic fibrosis<br>Young's syndrome |
| Mechanical obstruction | Intrinsic endobronchial tumour obstruction<br>Extrinsic lymph node compression |
| Toxic insults | Aspiration<br>Inhalation of toxic gases or chemical (e.g. ammonia) |
| Associations | Rheumatoid arthritis<br>Inflammatory bowel disease<br>Chronic sinusitis<br>Yellow nail syndrome<br>Connective tissue diseases<br>(e.g. SLE, Sjogren's syndrome) |

**Table 1: Causes of bronchiectasis.**

## History taking in bronchiectasis

**Bronchiectasis should be suspected if there is history of:**

- Persistent cough productive of mucopurulent or purulent sputum throughout the year.
- Intermittent hemoptysis.
- Breathlessness some time associated with wheeze.
- Intermittent pleuritic pain usually with exacerbations.
- Lethargy and malaise.

**Past medical history, ask about:**

- Recurrent lower respiratory tract infection especially childhood measles, whooping cough and tuberculosis.
- Any concomitant condition which could be associated with bronchiectasis (e.g. rheumatoid arthritis).
- Previous history or family history of diseases associated with immune suppression or immune suppressing medication.

Signs: look for finger clubbing, coarse inspiratory and expiratory, crackles and wheeze, however, examination often unremarkable.

## Investigations leading to diagnosis

Chest X-rays carry about 50% sensitivity to pick up bronchiectasis and are, therefore, often unremarkable. Typical findings are thickened and dilated bronchi producing tram line and ring shadows.

High resolution CT of Chest (HRCT) is a thin slice CT scan, highly sensitive to detect even minor lung parenchymal changes. It is the gold standard test for the diagnosis of bronchiectasis. It has replaced the invasive investigation of bronchography and is now widely used. The diagnostic criteria for brochiectasis on HRCT is to find dilated and thickened bronchi (dilatation being present if the diameter of the affected bronchus is greater than the accompanying pulmonary artery giving a "signet ring" sign) and there is failure of tapering as the bronchus courses towards the periphery.

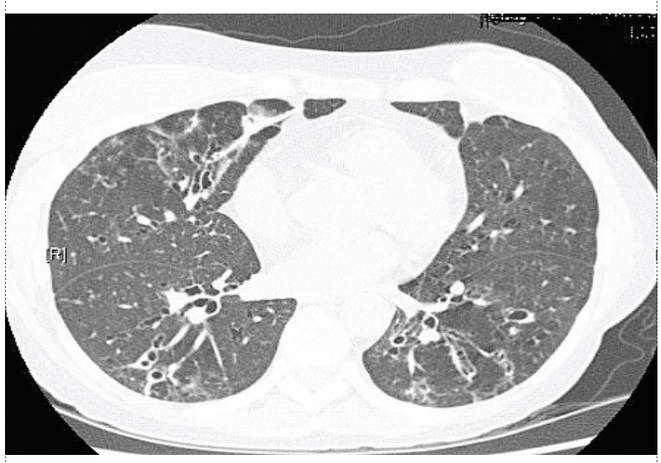



**Figure 1: CT scan of a patient with bronchiectasis showing dilated thick walled bronchi and characteristic signet-ring sign.**

# BRONCHIECTASIS – NOT ALL COUGHS AND DYSPNOEA'S ARE COPD!

Ahsen Nazir, Nazir Khan & Damian John McKeon

## Investigations to assess current disease status

High resolution CT of the chest (HRCT) to assess extent of bronchiectasis – single or multi-lobular involvement.

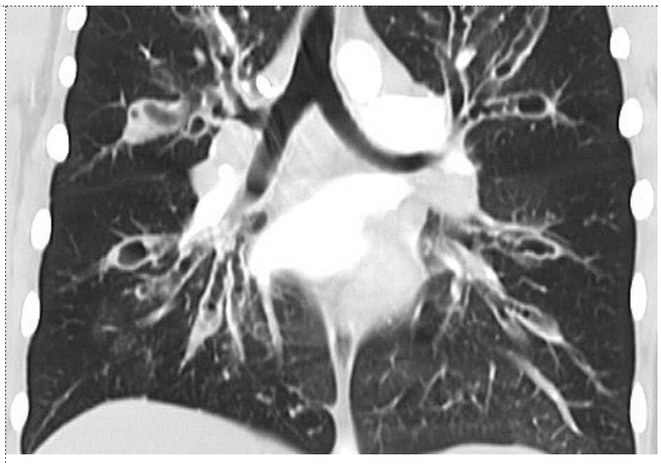
Pulmonary function test to look for associated obstructive airways disease and extent of loss of airway function. Patients also often exhibit reversibility to bronchodilators.

Sputum culture to assess colonising micro-organism including aspergillus and non-tuberculous mycobacterium. The usual colonising organisms are *haemophilus influenza; streptococcus pneumoniae; pseudomonas aeruginosia; and staphylococcus aureus.*

Inflammatory markers to assess patient's current inflammatory status.

| Investigations To Assess Underlying Aetiology For Bronchiectasis | |
|---|---|
| Bronchoscopy | If CT suggest bronchial obstruction - to establish whether tumour or foreign body. BAL/ bronchial washings for microscopy and culture to establish opportunistic infection i.e. Non Tuberculous Mycobacterium now known as environmental mycobacteria/ fungal infection |
| Full assessment of humoral, innate and Ab- mediated immunity including immunoglobulins and IgG subclasses | To identify immunodeficiency |
| Alpha 1 antitrypsin level | To identify alpha 1 antitrypsin deficiency |
| Autoimmune screen | To identify associated autoimmune disease |
| Aspergillus skin testing, IgE and RAST to aspergillus | To identify ABPA (allergic bronchopulmonary aspergillosis |
| Cystic fibrosis genetic and skin test | To exclude most common genetic cause, cystic fibrosis |
| Saccharin screening test Nasal nitric oxide Measure ciliary beat frequency | As evidence of primary ciliary dyskinesia |

**Table 2: Investigations to assess underlying aetiology for bronchiectasis.**



## General management

Patients needs good general education about bronchiectasis. Patients with an established disease should be told that bronchiectasis will always be present and the strategy developed for management will need to be flexible and lifelong. It is for the patient to decide after being informed by the health professional about the goals of treatment considering all of the evidence and professional recommendations.

Sputum clearance is important by employing physiotherapy with postural drainage and chest percussion. A handheld flutter device is also effective in clearing secretions.

Inhaled bronchodilator therapy is indicated in patients with variable air flow obstructions.

Mucolytics - the use of recombinant human DNAse is only recommended in cystic fibrosis. The use of N-acetyl cystine and carbocystine may be helpful in chronic, thick sputum producers.

Antimicrobial therapy and high dose oral antibiotics should be given for at least 14 days based on the colonising micro organisms and sensitivity in an acute exacerbation, and the response to therapy should be monitored by assessing the fall in sputum volume and changes in the sputum from mucopurulent to mucoid. Patients who repeatedly fail to respond to oral therapy should be treated with IV antibiotics for 10–14 days.

Maintenance therapy - the use of long-term nebulizer antibiotic is established and common in cystic fibrosis, but long-term oral antibiotic in non-CF bronchiectasis is controversial and may involve rotational treatment.

Surgery is indicated if the bronchiectasis is limited to a single lobe and if there is uncontrolled hemoptysis or failure of aggressive antimicrobial therapy to treat infection.

Lung transplant is usually indicated for cystic fibrosis-related bronchiectasis. It should also be considered in a severe non-CF-related disease. It always requires a bilateral transplantation.

# BRONCHIECTASIS – NOT ALL COUGHS AND DYSPNOEA'S ARE COPD!

Ahsen Nazir, Nazir Khan & Damian John McKeon

| Specific Therapy For Individual Causes | |
|---|---|
| Panhypogammaglobulinemia | Intravenous immunoglobulins with guidance from immunologist |
| Primary ciliary dyskinesia | Intense physiotherapy |
| Allergic bronchopulmonary aspergillosis | Corticosteroids and antifungal therapy |
| Associated conditions (e.g. rheumatoid arthritis, inflammatory bowel disease) | Treat both bronchiectasis and associated active condition |
| Gastro-oesophageal reflux and aspiration | Trial of proton pump inhibitors |

**Table 3: Specific therapy for individual causes.**

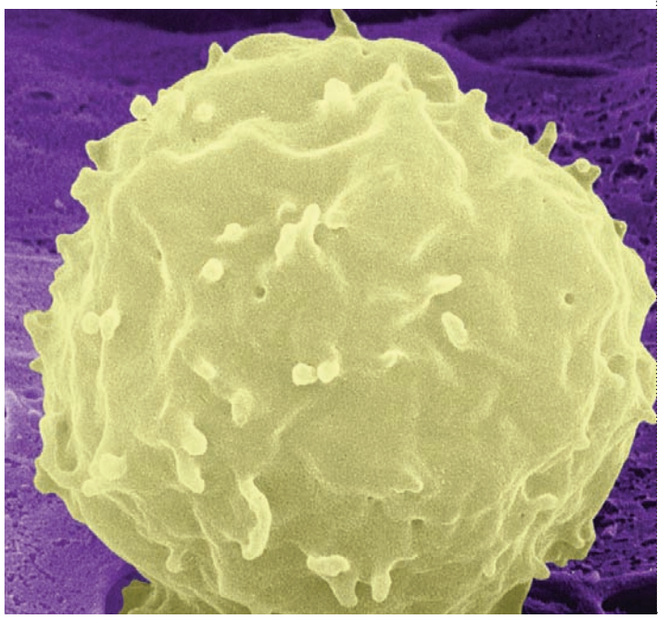
## Complications

Infective exacerbations are the most common complications to precipitate hospital admissions. Minor hemoptysis is common but the massive hemoptysis requiring embolisation or surgery is rare. Empyema, metastatic spread of infection and amyloidosis are all rare now days.

## Discussion

Identifying the underlying cause for bronchiectasis can have major implication for management as we have seen in our first case. All efforts should be made to identify the underlying cause by taking a good history and looking for other systemic features that might point to an underlying multi-system disease.

Long term outlook for bronchiectatic patients is good, with exception of patients with cystic fibrosis, if the disease is diagnosed early and treated aggressively, as is the example of our second case. Each patient requires a tailored management plan which should be agreed between the patient and the physician. This also facilitates patient education and self-management.



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# MANAGEMENT OF OBSTRUCTIVE SLEEP APNOEA

David E Simcock & Himender K Makker


**A 46-year-old shopkeeper, originally from Bangladesh, presents to the sleep clinic after referral by the ENT surgeons.**
**Good Medical Practice.**

## Clinical case

A 46-year-old shopkeeper, originally from Bangladesh, presents to the sleep clinic after referral by the ENT surgeons. His GP had originally referred him to ENT following complaints from his wife about loud snoring, to which he is oblivious. ENT examination was unremarkable. He is a smoker, drinks alcohol each evening and has no previous medical history, however, his blood pressure is under surveillance in the community and he has recently had some blood tests to look at his blood glucose and lipids. His wife is upset that her husband has changed, is becoming withdrawn and she has had to move to sleep in another room in the house. He feels un-refreshed after a night's sleep and his wife also reports finding him asleep on the sofa when she return from work in the evening. His BMI is 30, oxygen saturations of 96% and normal spirometry.

**What questions would you ask next?**

**What is the cause of the symptoms?**

**What investigations would you order to confirm the correct diagnosis?**

**What advice would you give?**

**How would you treat this patient?**

## Case discussion

The above represents a typical case presentation to the sleep clinic. The patient has a number of predisposing factors for sleep disordered breathing, such as age, sex, race and a number of salient features suggestive of the diagnosis. The history needs to be expanded to include details of sleep quality and symptoms, and subjective scores of daytime sleepiness. The crux of consultation is to determine if this man is a simple snorer, and likely to come to no harm, or has obstructive sleep apnoea (OSA) syndrome and is at risk of its many sequelae. Treatment of OSA is rapidly effective and immediately apparent to the individual and it is unfortunate that this remains one of the most unrecognised and untreated disorders in medicine. The answers to the above points raised form the basis of this article and are detailed below.

## Introduction

Sleep disordered breathing (SDB) describes a group of conditions characterised by an abnormality of respiratory pattern, such as pauses in breathing or the quality of ventilation during sleep. The most common form of SDB is OSA and will be the main focus of this article. The last 15 years has seen a dramatic rise in the number of obese adults worldwide and epidemiological studies have identified bodyweight as the strongest risk factor for OSA. This represents a significant public health problem and there is an increasing demand for sleep service facilities due to the high prevalence and growing public awareness of sleep disorders. It is conservatively estimated that OSA affect 4% of middle-aged men and 2% of middle-aged women in the UK[1]. Despite considerable progress in the field of sleep medicine, most of those affected remain undiagnosed and half of those with a diagnosis do not have access to the specialist treatment they need.

## Obstructive sleep apnoea/hypopnoea syndrome

OSA is a chronic condition characterised by repetitive narrowing or collapse of the pharyngeal airway during sleep leading to brief arousal, sleep fragmentation and excessive daytime sleepiness. The functional consequence of intermittent airflow obstruction leads to loud snoring, which is often the reason for specialist referral. Snoring itself is extremely common, affecting 30% of the middle-aged population, but since most of these individuals are not sleepy ("simple snorers"), they are unlikely to have OSA or suffer any adverse effects. Airway obstruction during sleep may be complete or partial despite ongoing respiratory effort. Complete occlusion leading to cessation of airflow for at least 10 seconds is known as apnoea; partial obstruction leading to a reduction in airflow by at least 50% for at least 10 seconds is known as hypopnoea. These apnoea/hypopnoea events are associated with oxygen desaturation and arousal from sleep. Since apnoeas and hypopnoeas frequently coexist, this form of SDB and associated sleepiness is referred to as the obstructive sleep apnoea/hypopnoea syndrome (OSAHS). The consequences of untreated OSAHS are considerable to the individual with significant morbidity and mortality and should not be underestimated. Patients frequently experience a poor quality of life, difficulties in their personal relationships, lose their jobs and have an increased risk of accidental injury including a three to sevenfold increased risk of road traffic accidents[2]. Recurrent nocturnal hypoxaemia results in raised sympathetic output mediating elevated blood pressure (BP); risk of stroke; arrhythmia; pulmonary hypertension; cardiac failure; and sudden cardiac death[3]. It is also suggested that OSAHS is an independent risk factor for developing insulin resistance and type 2 diabetes mellitus[4]. A number of factors predispose to developing OSAHS.

# MANAGEMENT OF OBSTRUCTIVE SLEEP APNOEA

David E Simcock & Himender K Makker

**Predisposing factors are:**

- Increasing age
- Male>female
- Race (highest incidence in Indians)
- Obesity and changes in body composition (e.g. collar size >17)
- Sedative drugs
- Smoking and alcohol consumption
- Familial/genetic factors
- Craniofacial anomalies (e.g. retrognathia)
- Medical co-morbidities (e.g. acromegaly, stroke, hypothyroidism).

## Pathophysiology

The underlying pathological mechanism is likely to vary considerable between individuals. The upper airway consists of a collapsible portion extending from the hard palate to the larynx in order to permit swallowing and speech. This feature also provides the opportunity for collapse on inspiration. An anatomically narrow airway is more prone to collapse than a normal one. MRI studies have demonstrated that the cross sectional area of the upper airway is likely to be reduced in those with OSAHS. In the awake state protective reflexes acting via upper airway dilator muscles maintain the integrity and patency of the airway. This is seen in OSAHS patients by increased activity in the genioglossus muscle compared to healthy controls. It is hypothesised that this dilating reflex is lost during sleep resulting in airway narrowing caused by the unopposed negative pressure generated on inspiration in predisposed individuals reliant on elevated genioglossal tone. Muscular tone is particularly reduced during REM sleep and apnoea/hypopnoeas are consequently more pronounced and associated with greater hypoxaemia compared with non-REM sleep in OSAHS. This resulting airway occlusion leads to snoring and reduced airflow. Apnoea/hypopnoea and ensuing hypoxia episodes terminate with arousal from sleep, muscular tone returns and airway patency returns. This cortical arousal may be observed on electroencephalogram (EEG) and indicated by an increased sympathetic drive resulting in cardio-acceleration and a rise in blood pressure (and cardiovascular consequences). Such arousals and interruptions to breathing can happen hundreds of times a night leading to fragmented sleep, loss of REM and slow wave sleep. The repetitive oxygen desaturation results in brain hypoxia and has a profound effect of neuropsychological functioning. Cortical arousal is not, however, essential for restoration in muscular tone since studies have shown increased inspiratory flow in the absence of arousal in 22% of instances. This suggests an additional contribution from other stimuli, such as hypoxia, hypercapnoea and negative airway pressure/muscle loading, in maintaining airway patency. It is also suggested that those with OSAHS have a degree of instability of ventilatory control, where the ventilatory sensors and effectors are uncoupled leading to a robust ventilatory response to an equivalent respiratory stimulus leading to airway collapse. Such increased respiratory effort may lead to arousal and not be associated with recordable apnoea/hypopnoea. This phenomenon is known as respiratory-related arousal and may lead to a separate entity or "upper airway resistance syndrome" (UARS). UARS helps to explain the poor correlation between daytime symptoms and apnoea/hypopnoea episodes recorded by sleep studies (see Figure 1).

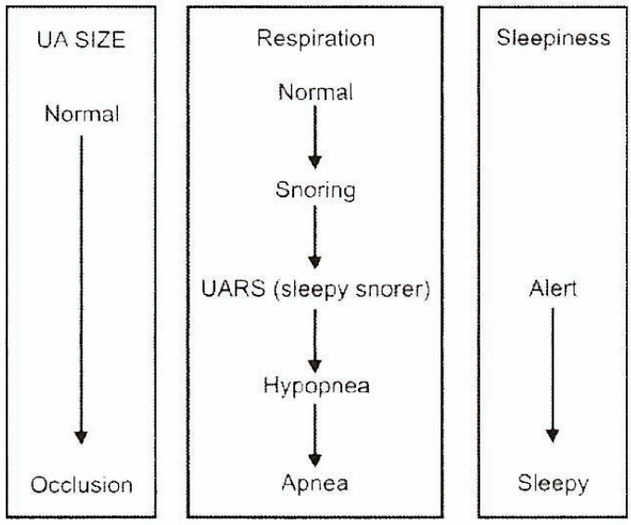
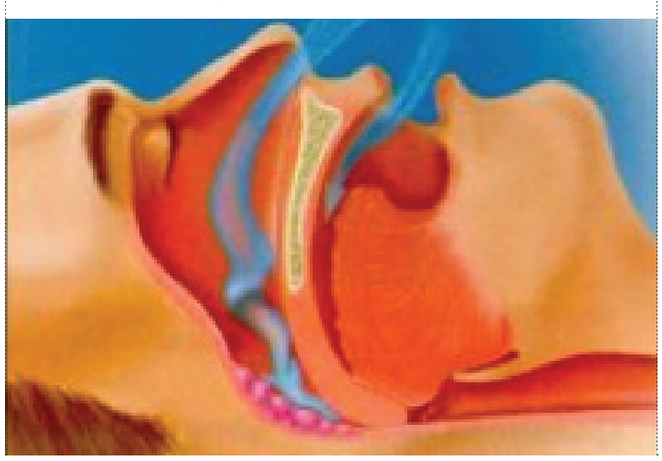



**Figure 1: Effect of upper airway size on breathing during sleep and daytime sleepiness.**

## Clinical features

It is desirable to interview the patient with their partner who may be a valuable source of information based on their observations of the patient when asleep. Snoring is the most frequently reported symptom in OSAHS and reflects airway obstruction during sleep. A diagnosis of OSAHS is unlikely in the absence of a snoring history and patients are frequently unaware of their snoring. The snoring is most apparent to partners who also suffer disturbed sleep and may sleep separately as a result. Partners may notices pauses in breathing during sleep and such witnessed apnoeas are a good diagnostic predictor of OSAHS. Many patients report waking at night with a choking sensation that can be terrifying and usually passes within seconds of waking. Patients report symptoms of excessive daytime sleepiness (EDS) and symptoms secondary to sleep fragmentation, such as fatigue; poor memory and concentration; daytime napping/falling asleep; changes in personality; depression; loss of libido; impotence and nocturia (see below). There are many causes of EDS with 30 to 50% of the population reporting sleepiness symptoms. EDS is not a useful discriminating feature alone and, in addition, does not correlate with severity of OSAHS. [1, 5]



# MANAGEMENT OF OBSTRUCTIVE SLEEP APNOEA

David E Simcock & Himender K Makker

**Symptoms of OSAHS:**

- Excessive daytime sleepiness
- Snoring
- Witnessed apnoeas
- Impaired memory/concentration
- Un-refreshed sleep
- Choking episodes in sleep
- Restless sleep
- Personality change/mood/depression
- Nocturia and enuresis
- Reduced libido
- Morning nausea/headache
- Sleep maintenance insomnia.

## Physical characteristics[1, 5]

Obesity (BMI >30) is a consistent finding in OSAHS; particularly central obesity and the patient's body mass index (BMI) should be calculated. Clinical experience suggests that many patients prior to presentation have noticed considerable, and recent, weight gain. Neck circumference is a strong positive predictor and a value >17≤/43cm is associated with an increased probability of OSAHS. Various craniofacial features, such as retrognathia; micrognathia; tonsilar hypertrophy; and macroglossia are associated with OSAHS, however, non-specific airway narrowing of the upper airway, due to excess fat in the parapharyngeal space, is the most consistent finding. The oral cavity should be inspected and a Mallampati score should be obtained in assessing the airway size based on the visibility of the uvula, tonsils and fauces (see Table 1). Mallampati grade 3 and 4 airways are most often associated with OSAHS. Patients may have clinical signs consistent with predisposing causes, such as acromegally or hypothyroidism. The link between hypertension and OSAHS is now well established and persistent or resistant hypertension is suggestive of the diagnosis. Measurements of FEV1 and FVC should be made to identify spirometric abnormalities that may point towards an underlying airway, chest wall or neuromuscular abnormality[1, 5].

| | |
|---|---|
| **Grade 1** | Complete visualisation of uvula, tonsils and arches. |
| **Grade 2** | Complete visualisation of uvula, tonsils and arches partially visible. |
| **Grade 3** | Only soft and hard palate visible. The uvula is hidden. |
| **Grade 4** | Only hard palate visible. |

**Table 2: Epworth Sleepiness Scale.**

Objective assessments of the patient's sleepiness are not usually available outside large sleep centres and include the Multiple Sleep Latency Test (MSLT), which measures the time to fall asleep (by EEG criteria). An average MSLT time of <7 minutes is regarded as pathological sleepiness. An alternative is the Maintenance of Wakefulness Test (MWT), which tests the ability to stay awake. A MWT <20 minutes is regarded as abnormal[1, 5].

## Sleep studies

The purpose of the sleep study (SS) is to confirm the diagnosis of OSAHS and make an assessment of severity and effect of treatment. In those with the appropriate clinical features, the diagnostic test must be able to demonstrate or exclude recurrent breathing pauses during sleep. The "gold standard" investigation for diagnosis of OSAHS is full polysomnography (PSG). PSG records a number of variables including EEG; electromyography; electrooculography respiratory flow; thoraco-abdominal movement oxygen saturation; ECG; snoring; and movement, and is carried out overnight in a specialist sleep centre with the aid of sleep technicians. The full PSG is time consuming, resource and labour intensive and studies have suggested that a number of the variables, while useful, do not contribute significantly to the diagnosis. Increasingly "limited sleep studies" are being adopted. Limited studies vary from overnight oximetry alone to multichannel recordings of movement, snoring, respiratory and oxygenation patterns without the neurophysiologic data (see Figure 2). Limited studies focused on cardiac and respiratory variables are often carried out at home to minimise patient inconvenience and cost.

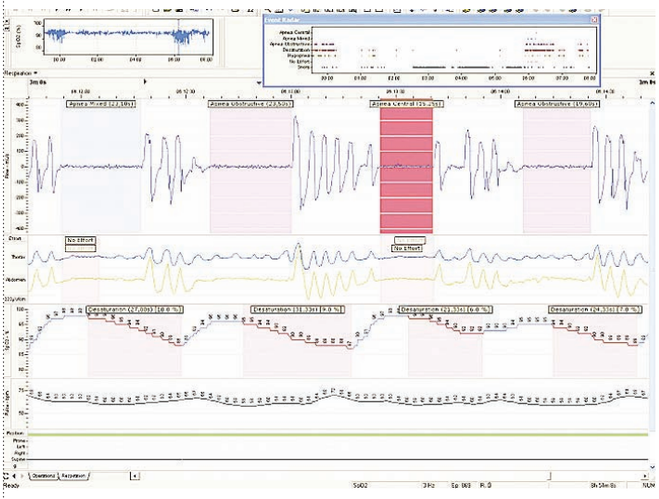


**Figure 2: Multi-channel home sleep study showing obstructive apnoea associated with oxygen desaturation.**

# MANAGEMENT OF OBSTRUCTIVE SLEEP APNOEA

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Overnight pulse oximetry is the simplest method of evaluating suspected OSAHS and is often sufficient alone by recording the characteristic pattern of repetitive desaturation (a 4% dip in oxygen saturation represents a 10 second obstruction to airflow) especially in more severe disease. Brief apnoeas and hypopnoeas may fail to cause desaturation and oximetry must be used with caution in mild cases. Visilab SS involves an 8-hour sleep observation period with video, audio and oximetry recordings. The video signal is used to assess sleep and sleep disturbance, the audio signal monitors breathing and snoring, and a 4% dip in oxygen saturation is used as an index of airflow obstruction. In expert hands, the data obtained from limited studies are adequate and highly effective in making the correct diagnosis and the ability of the reporter of the SS is likely to be more important than the choice of device. It is sensible to use limited SS, such as overnight home oximetry, as the first line investigation, particularly in those who the clinical suspicion of OSAHS is either high or low. More complicated in-hospital studies should be reserved for those in which the diagnosis is unclear or in those with atypical features suggesting an alternative diagnosis, such as central sleep apnoea or periodic leg movement. The SS records the frequency of apnoeas and hypopnoeas per hour of recording and is used to diagnose and assess the severity of OSAHS and need for treatment. This is known as the apnoea/hypopnoea index (AHI) and OSAHS may be subdivided depending on the AHI (see Table 3)[1, 5].

| a. Diagnosis |
|---|
| AHI >5 associated with unexplained daytime sleepiness or any of two symptoms. See symptoms of OSAHS, as above |
| **b. Severity** |
| Mild: AHI 5–14 hour<br>Moderate: AHI 15–30 hour<br>Severe: AHI >30 hour |

**Table 3: Diagnosis and severity of OSAHS based on AHI index.**

The relationship between AHI and EDS scores is not strong and it is essential to take into consideration the patients symptoms before assigning a diagnosis of OSAHS. As mentioned previously, this is likely to be due to unrecorded arousal events not related to AHI.

## Treatment of OSAHS

The aim of treatment is to reduce daytime sleepiness by reducing the number of apnoea/hypopnoeas experienced during sleep. Treatment depends on the severity of OSAHS and, to an extent, patient choice. Evidence from randomised controlled trials and national guidelines (NICE) advise that those individuals with moderate or severe OSAHS (AHI >15 or 4% desaturation rate >10/hr) are likely to benefit from treatment[7]. Those with mild OSAHS (AHI <5) are recommended to receive treatment if they have symptoms that affect their quality of life or daily activities. Treatment options are broadly divided into three groups: lifestyle changes; non-surgical options, such as continuous positive airway pressure (CPAP) and mandibular advancement splint (MAS); and surgical options.

## Lifestyle changes

Overweight patients are encouraged to lose weight. Weight loss of 10% or more is desirable and has been shown to improve markers of OSAHS. Those with gross obesity that are unable to lose weight can be considered for bariatric surgery. Patients should avoid alcohol, that increases caloric intake and decreases upper airway dilating muscle tone. Likewise sedative drugs should be avoided and patients should stop smoking. These measures alone may be sufficient in treating simple snorers or those with mild OSAHS[6].

## CPAP

CPAP is the first choice treatment for those with moderate and severe OSAHS and provides instant relief from symptoms[1,7]. CPAP therapy works by blowing the airway open throughout all phases of sleep breathing, usually with pressures of 5–10cm$H_2$0. In addition to acting as a "pneumatic splint", CPAP may work by reducing upper airway mucosal oedema and increasing lung functional residual capacity (see Figure 3).

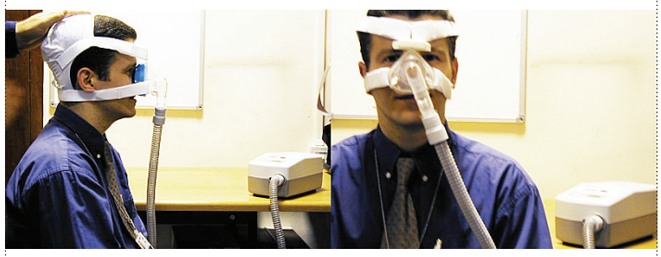

**Figure 3: Continuous Positive Airway Pressure (CPAP) treatment: compressor pump, long tubing and nasal mask held in place by head straps.**

# MANAGEMENT OF OBSTRUCTIVE SLEEP APNOEA

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It has been shown in randomised placebo controlled trials to improve breathlessness during sleep; sleep quality; sleepiness scores; Bp; cognition; mood; quality of life; and driving ability. CPAP operates by means of a flow generator that generates a positive pressure conducted via air tubing to a nasal or facemask worn overnight. Initiation of CPAP treatment includes finding an agreeable and comfortable mask and trying it out in the clinic for at least 30 minutes before overnight use. An overnight monitored trial of CPAP is used to identify the pressure required to splint the airway open. The CPAP pressure is then titrated accordingly and an individualised pressure is prescribed and set. Auto-titrating CPAP (A-CPAP) machines adjust the pressure delivered depending on the amount of airway resistance and degree of obstruction. These intelligent machines can be used to establish the pressure required for later home use. A-CPAP are more comfortable for the patient since they allow lower pressures to be delivered at times of low airway resistance and in theory produce better outcomes and compliance than fixed pressure machines. Studies comparing CPAP to A-CPAP have not shown differences between the two modalities in terms of AHI, subjective sleepiness or compliance with therapy. The efficacy of CPAP is dependant on the patient's willingness to use it for at least 4 hours each night. Non-adherence (<4/hrs) is reported in 46 to 83% of patients treated for OSAHS. CPAP is rather cumbersome and an unusual addition to peoples bedroom routines and takes acclimatising to. Side effects of CPAP include: nasal congestion; dryness and epistaxis; sinusitis; nose bridge ulceration; discomfort; claustrophobia; and bloating. Heat humidification and topical nasal steroids help improve some of these minor problems and increase patient comfort and acceptance. It is said that patients prefer A-CPAP, however, there is no trial data showing increased compliance with this mode of delivery. Similarly, other modes of CPAP that vary pressure between inhalation and exhalation (flexible-CPAP) and bi-level-PAP have not been shown to increase adherence to CPAP. Patients should have access to a dedicated sleep nurse or technician who can help with mask changes and adjustments, initiation and titration of CPAP and provide ongoing patient support. This last point is particularly important since the early pattern of CPAP use during the first few days/weeks determine and predict long-term compliance.

## Mandibular advancement splints (MAS)

The oral devices work by mechanically holding the lower jaw and tongue in a forward position and open the pharyngeal airway (see Figure 4). MAS have proven efficacy in relieving symptoms of snoring, however, their ability to reduce AHI is variable and not as effective as CPAP, particularly with more severe disease. MAS are recommended as treatment for simple snorers and for patients with mild OSAHS without daytime sleepiness and represent an alternative to those unable to tolerate CPAP[8].

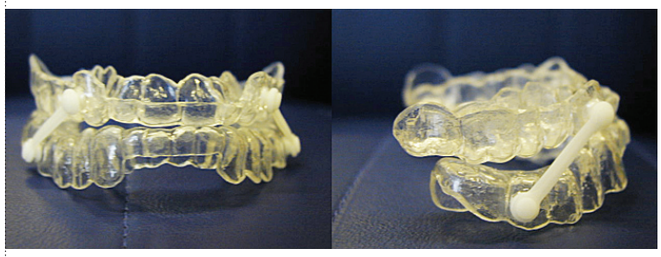
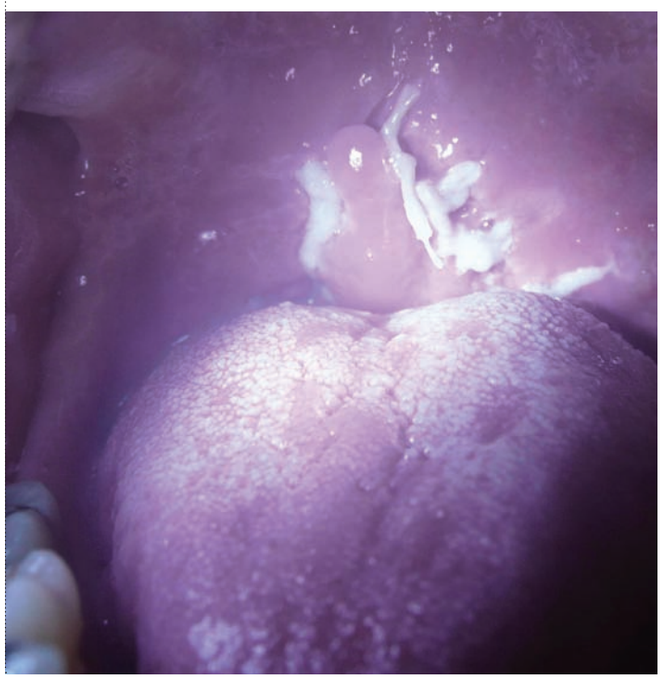


**Figure 4: Front and side view of a custom made mandibular advancement splint.**

## Surgery

Uvulopalatopharyngoplasty (UPPP) and laser-assisted UPPP were previously used to treat OSAHS. Such measures were only effective in a minority of individuals and clinical outcomes were variable. Neither procedure is currently recommended for treatment of OSAHS. Specific surgical interventions, such as tonsillectomy, nasal polypectomy and mandibular advancement, remain viable options to correct anatomical anomalies in certain individuals. Tracheostomy completely bypasses the upper airway and should only be considered when all other options have failed.



# MANAGEMENT OF OBSTRUCTIVE SLEEP APNOEA

David E Simcock & Himender K Makker

## Good Clinical Care Assessment Questions

**1. The site of upper airway obstruction in patients with obstructive sleep apnoea is at the level of:**

**a.** Nose.
**b.** Pharynx.
**c.** Larynx.
**d.** Trachea.
**e.** Main bronchus.

**2. The following symptoms suggest diagnosis of obstructive sleep apnoea:**

**a.** Breathlessness on exertion.
**b.** Disturbed sleep.
**c.** Loud disruptive snoring.
**d.** Early morning headaches.
**e.** Excessive daytime sleepiness.

**3. Patients with obstructive sleep apnoea usually have the following features on clinical examination:**

**a.** Obesity.
**b.** Collar size of >17.
**c.** Inspiratory stridor.
**d.** Reduced chest expansion.
**e.** Crowded oral cavity.

**4. The treatment of choice for patient with OSA is:**

**a.** Inhaled bronchodilator.
**b.** Tonsillectomy.
**c.** CPAP.
**d.** Laser assisted palatoplasty.
**e.** Bariatric surgery.

**5. The diagnosis of obstructive sleep apnoea syndrome is based on the following:**

**a.** Restrictive ventilatory defect on spirometry.
**b.** Excessive daytime sleepiness on Epworth Sleepiness Score.
**c.** Apnoea–hypopnoea index of >5 /hour on sleep study.
**d.** Type 2 ventilatory failure on arterial blood gas analysis.
**e.** Oxygen desaturation dip of 4% >10 /hour on overnight pulse oximetry.

## Answers

**1.** b.
**2.** b, c, d, e.
**3.** a, b, e.
**4.** c.
**5.** b, c, e.

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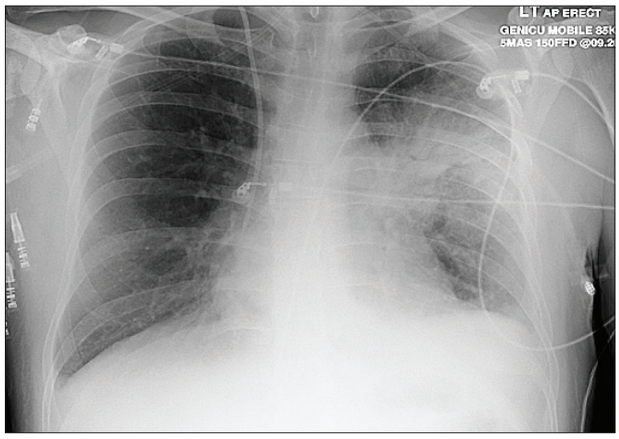
# CHEST X-RAY QUIZ

Adrian Draper

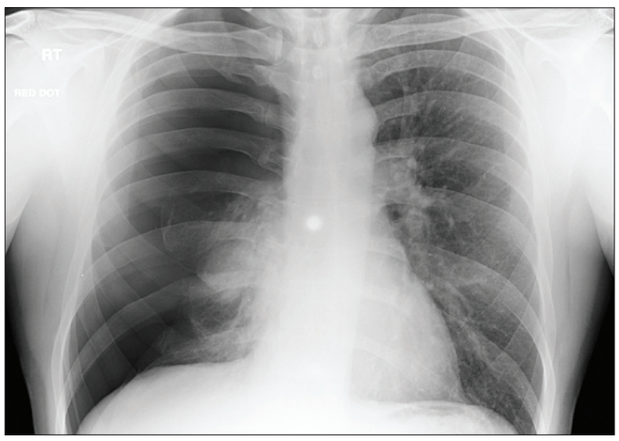
**A great time to test your knowledge.**
**Picture Quiz.**

This patient presents with a 3-day history of fever, cough and breathlessness.

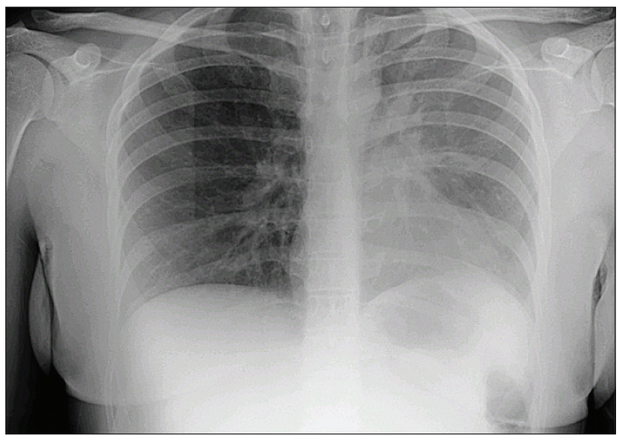



**1. What does the X-ray show?**
**2. What treatment should be given?**

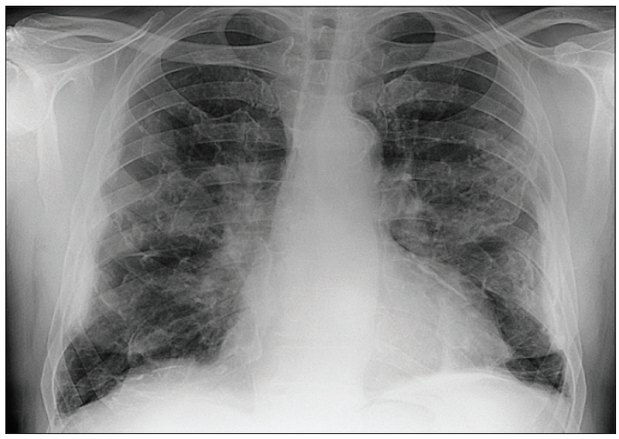
This patient presents with sudden onset chest pain and breathlessness.




**3. What does the X-ray show?**
**4. What treatments should you consider?**



**5. What does the X-ray show?**

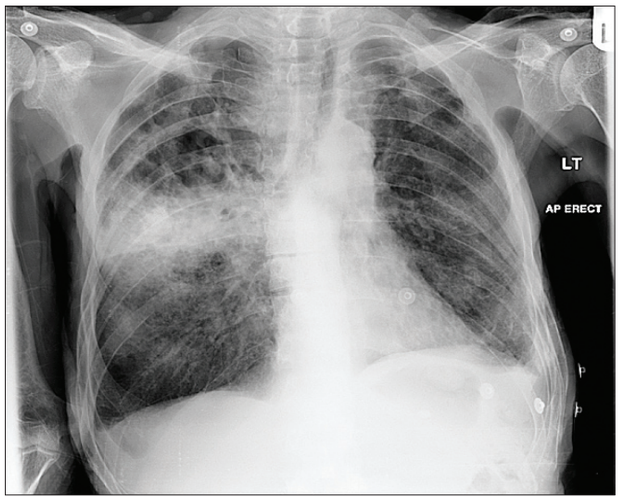


**6. What is the main abnormality shown on this chest X-ray?**
**7. Does this patient require regular follow-up in clinic and why?**

# CHEST X-RAY QUIZ

Adrian Draper

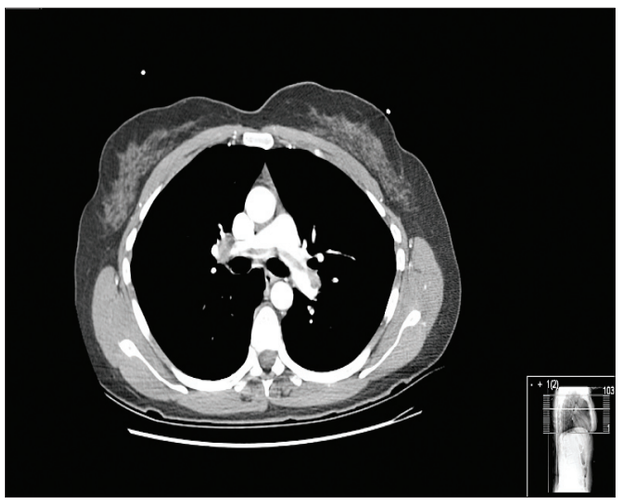
This patient presents with a productive cough, fever and night sweats.



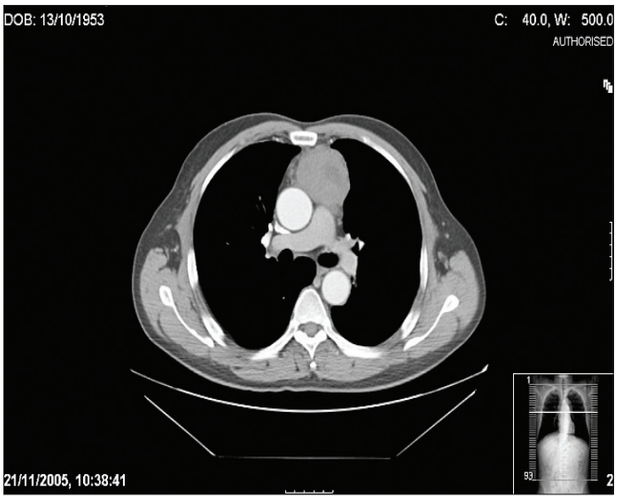

**8. What does the X-ray show?**
**9. What test would you request?**

This patient presented with shortness of breath, syncope and hypotension.

**10. What investigation has been performed?**

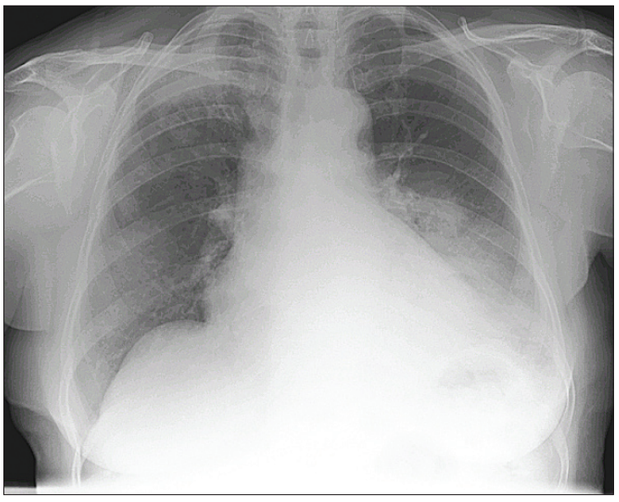


**11. What does the scan show?**
**12. What treatment might be indicated?**




**13. What does this CT scan show?**
**14. Give four possible causes of the abnormality shown on this CT slice.**

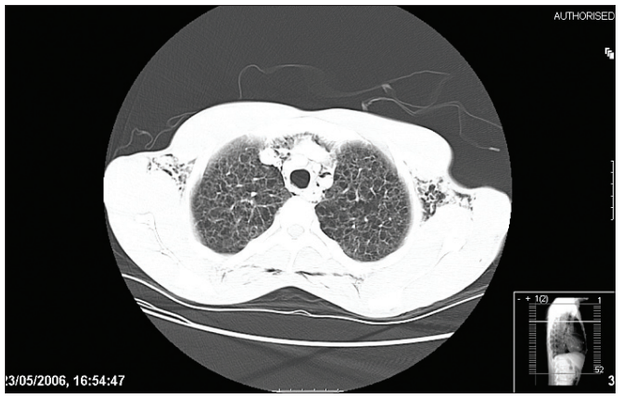
This patient presented with general malaise, cough, fever, weight loss, elevated inflammatory markers and abnormal liver function tests. All microbiological cultures were negative and the patient did not respond to antibiotic therapy.



**15. What is the abnormality on this chest X-ray and CT?**
**16. Give some possible causes.**

# CHEST X-RAY QUIZ

Adrian Draper




**17. What two main abnormalities are present on the CT scan?**
**18. Give a possible diagnosis?**

## Answers

**Answer 1.**
Left midzone consolidation with characteristic air bronchograms, right triple lumen jugular line in situ, monitoring electrodes and oxygen tube.

**Answer 2.**
Treat ABC first so high flow oxygen therapy. Assess pneumonia severity (CURB65 score). This patient is ill judging by monitoring equipment seen on the X-ray so will warrant an intravenous penicilllin and macrolide treatment.

**Answer 3.**
Large right-sided pneumothorax, without mediastinal shift.

**Answer 4.**
Pneumothoraces will reabsorb at 1.25% of volume per day – this can be increased with supplemental oxygen therapy.

If the patient is symptomatic pleural aspiration can be attempted using a cannula and syringe via 4th intercostal space mid axillary line or 2nd intercostal space mid clavicular line.

If aspiration is unsuccessful an intercostal drain will be needed.

**Answer 5.**
This X-ray shows the veil sign of left upper lobe collapse. There is generalised opacification of the left lung field with obscuration of the left heart border and elevation of the left hemidiaphragm.

**Answer 6.**
Calcified pleural plaques.

**Answer 7.**
No, these are benign and will not undergo malignant transformation. However, prior asbestos exposure does render the patient at higher risk (depending on amount of exposure) of developing lung cancer or malignant mesothelioma separately so information about warning signs should be given.

**Answer 8.**
Cavitation right upper lobe and bilateral apical abnormalities.

**Answer 9.**
An urgent sputum sample with acid alcohol fast stain. This patient had smear positive tuberculosis and patients with suspected TB should be isolated immediately if admitted to hospital.

**Answer 10.**
CT pulmonary angiography.

**Answer 11.**
Saddle embolus in pulmonary bifurcation.

**Answer 12.**
A large pulmonary embolus associated with shock (hypotension) is an indication for thrombolytic therapy. This patient was successfully treated with tissue plasminogen activator (TPA).

**Answer 13.**
Anterior mediastinal mass.

**Answer 14.**
Remember the 4 "Ts":
**a.** Retrosternal Thyroid
**b.** Thymoma
**c.** Terrible Lymphoma
**d.** Teratoma (Germ cell tumours)

# CHEST X-RAY QUIZ

Adrian Draper

**Answer 15.**
There is peripheral multifocal consolidation in right upper lobe and lingular areas.

**Answer 16.**
**a.** Multifocal infection
**b.** Cryptogenic organising pneumonia
**c.** Eosinophilic pneumonia
**d.** Multifocal bronchoalveolar cell carcinoma
**e.** Primary lymphoma of the lung

This patient had cryptogenic organising pneumonia, a rare but well described clinical entity, the patient had a good response after treatment with oral corticosteroids.

**Answer 17.**
**a.** apical cystic changes.
**b.** pneumomediastinum with subcutaneous emphysema.

**Answer 18.**
Pulmonary Langerhans Cell Histiocytosis.

This very rare condition that occurs in smokers presents in adult life with nodular and cystic diseases with sparing of the lung bases. It is often associated with pneumothoraces. This patient underwent an open lung biopsy that confirmed the presence of histiocytes that stained with S-100 protein and monoclonal antibody CD-1a . No treatments have been tested in clinical trials for this condition and some patients improve spontaneously – particularly with smoking cessation which must be implemented.

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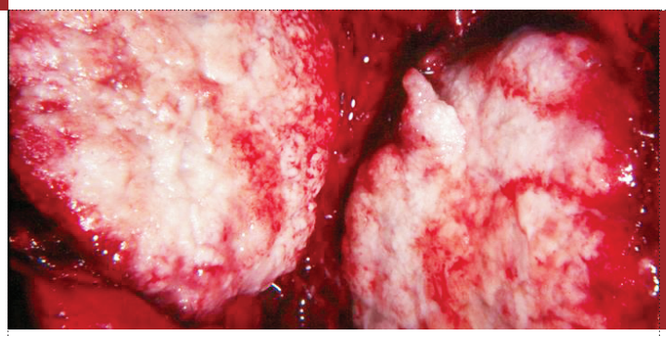
# THE UNILATERAL WHITE LUNG

Arshad Ali Khan, Sandip Mandal and Ashis Banerjee



**An 80-year-old woman had been previously diagnosed with non-small cell lung cancer. Patient Management.**

## Case report

An 80-year-old woman had been previously diagnosed with non-small cell lung cancer. She attended the emergency department 14 months after the initial diagnosis, with increased shortness of breath, lethargy, reduced appetite and generalised weakness. She had a past medical history of type 2 diabetes mellitus, currently requiring insulin, hypertension and ischaemic heart disease.

On examination, she was found to be anaemic. Her vital signs were: temperature 36.9 degrees Centigrade, heart rate: 100 beats per minute; supine blood pressure 160/60mmHg; respiratory rate 22 per minute; and Sp02 96% on oxygen via nasal cannula at a flow rate of 2 litres per minute.

Clinical examination revealed reduced expansion, percussion dullness and absent breath sounds over the left hemithorax. The trachea was felt to be central. Examination of other systems did not reveal any notable abnormality, apart from a palpable liver edge. She had a haemoglobin of 8.2%. A chest X-ray was obtained (see Figure 1).

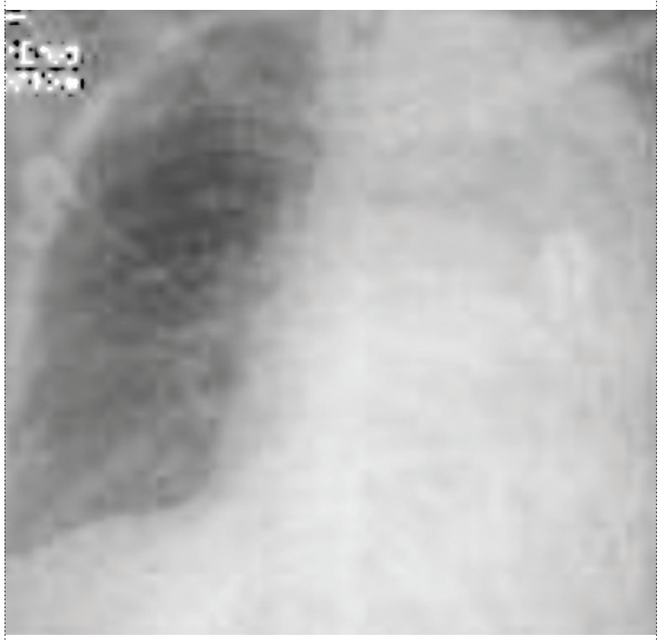


**Figure 1.**

## Questions

**1. How would you describe and analyse the chest X-ray findings?**

**2. Based on the clinical examination and radiological findings, what is your differential diagnosis?**

**3. How would you further manage her presenting condition?**

## Answers

**1. A chest X-ray showed a homogeneous, total opacification of the entire left lung. Further analysis of the chest X-ray should focus on determination of the presence or absence of expansion in the volume of the opaque hemithorax. Expansion causes mediastinal shift to the opposite side, widening of the intercostal spaces and depression of the hemidiaphragm. Reduction in volume causes mediastinal shift to the same side, narrowing of the intercostal spaces, and elevation of the hemidiaphragm[1].**

Based on systematic radiological analysis of a unilateral opaque hemithorax on the chest X-ray, the following possibilities emerge.

**Mediastinal shift away from opaque side:**

- Massive pleural effusion, including chylothorax; haemothorax; empyema. With a pleural effusion, some aerated lung is usually visible at the apices on an erect film. A mediastinally-based retro-cardiac density is caused by herniation of a fluid-filled azygo-oesophageal recess.
- Diaphragmatic hernia (fluid filled).
- Tumour.

**Central mediastinum:**

- Extensive pulmonary consolidation.
- Pleural thickening (e.g. mesothelioma)[2].
- Massive pleural effusion, with a fixed mediastinum (from malignant pleural infiltration) or with obstructive collapse, usually because of a bronchial carcinoma in the underlying lung[3].

# THE UNILATERAL WHITE LUNG

Arshad Ali Khan, Sandip Mandal and Ashis Banerjee

**Mediastinal shift towards opaque side:**

- Complete left lung collapse secondary to central obstructing tumour. Whole lung atelectasis is often associated with herniation of the opposite lung.
- Central mucus plug (postoperative).
- Pulmonary hypoplasia.
- Previous pneumonectomy: elevated gastric bubble; leftward shift of mediastinum; surgical clips in the left hemithorax in the vicinity of the left main stem bronchus; compensatory hyperinflation of the opposite lung.
- Restrictive pleural disorder with lung collapse.

**2. The relevant differential diagnoses in this patient should include extensive consolidation and tumour, alongside massive pleural effusion.**

**3. Previous chest X-rays should be reviewed. In particular, previous attempts at pleural drainage should be noted, along with their outcome. The radiological abnormality needs further categorisation, with an ultrasound being a useful screening tool for pleural fluid, which appears dark and anechoic on ultrasound . Furthermore, large unilateral chest tumours that are hidden by pleural fluid collections, can be identified and characterised by ultrasound[4]. In these circumstances, the co-existence of narrowing of the carinal angle in patients with mediastinal shift is helpful in confirming the diagnosis. CT scanning, with or without bronchoscopy, allows for recognition of the underlying lesion. Pleural drainage should only be attempted after a confident diagnosis of pleural fluid.**

Repeated attempts at intercostal drainage were unsuccessful. With ultrasound guidance on the day after admission, a drain was inserted. Small amounts of haemorrhagic fluid were obtained, but there was a sensation of resistance to needle insertion. Repeat chest radiology after insertion did not show any change (see Figure 2).

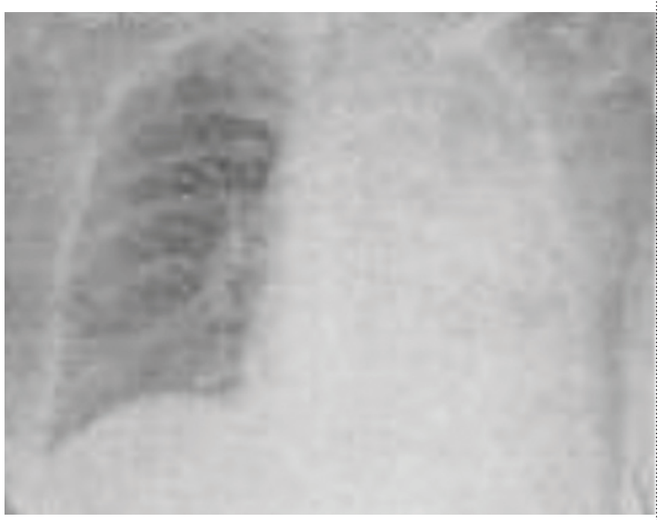


**Figure 2**

CT scanning was advised, but the patient declined any further intervention.

**Learning points:**

- There is a wide range of differential diagnoses for the "white lung".
- In all cases, volume changes including mediastinal shift, should be looked for.
- Further imaging is often required.
- Repeated attempts at intercostal drainage for a presumed pleural effusion are contraindicated.

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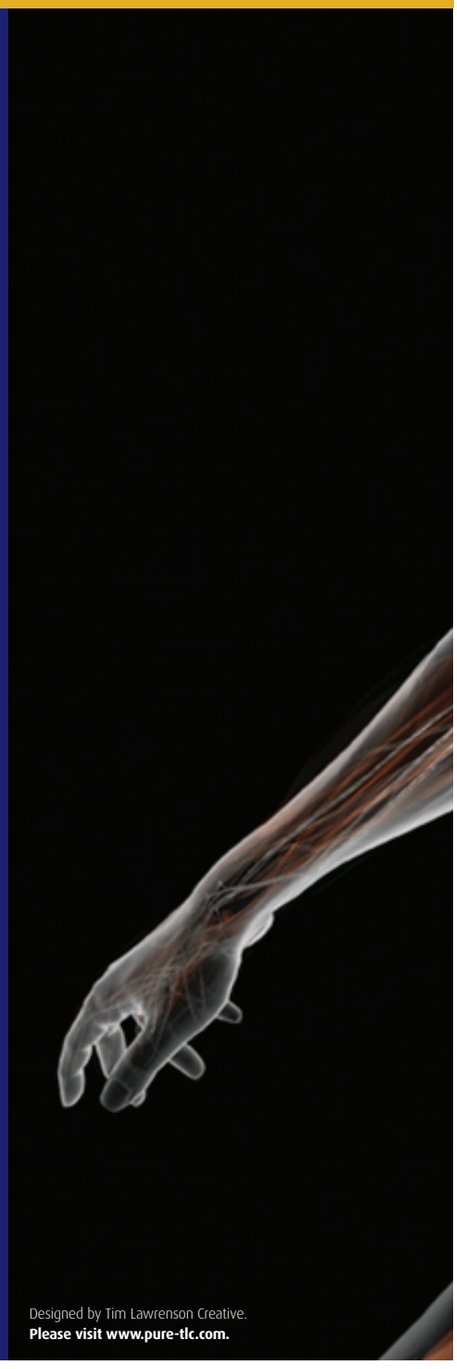

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