Station 1
Respiratory

	Short case	Checked and updated as necessary for this edition by
1	Interstitial lung disease (fibrosing alveolitis)	Dr Omer Khair*
2	Pneumonectomy/lobectomy	Dr Omer Khair*
3	Chronic bronchitis and emphysema	Dr Omer Khair*
4	Bronchiectasis	Dr Omer Khair*
5	Dullness at the lung bases	Dr Omer Khair*
6	Rheumatoid lung	Dr Omer Khair*
7	Old tuberculosis	Dr Omer Khair*
8	Chest infection/consolidation/pneumonia	Dr Omer Khair*
9	Yellow nail syndrome	Dr Shireen Velangi*
10	Kyphoscoliosis	New short case for this edition by Dr Dev Banerjee*
11	Stridor	Dr Omer Khair*
12	Marfan's syndrome	Dr David Carruthers*
13	Carcinoma of the bronchus	Dr Omer Khair*
14	Klippel–Feil syndrome	Dr David Carruthers*
15	Kartagener's syndrome	Dr Omer Khair*
16	Lung transplant	Dr Omer Khair*
17	Cystic fibrosis	Dr Omer Khair*
18	Obesity/Pickwickian syndrome	Dr Omer Khair*
19	Pneumothorax	Dr Omer Khair*
20	Cor pulmonale	Dr Omer Khair*
21	Collapsed lung/atelectasis	New short case for this edition by Dr Rahul Mukherjee*
22	Superior vena cava obstruction	Dr Omer Khair*
23	Tuberculosis/apical consolidation	Dr Omer Khair*
24	Normal chest	Dr Bob Ryder

*All suggested changes by these specialty advisors were considered by Dr Bob Ryder and were accepted, edited, added to or rejected with Dr Ryder making the final editorial decision in every case.

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Case 1 | Interstitial lung disease (fibrosing alveolitis)

Frequency in survey: main focus of a short case or additional feature in 34% of attempts at PACES Station 1, Respiratory.

Record

The patient is breathless on minimal exertion (and may be on long-term oxygen therapy). There is bilateral *clubbing* of the fingers. There is evidence of steroid purpura peripherally, (may be) *cyanosis*, reduced but symmetrical expansion of the chest (there may be dullness to percussion at the bases) and *fine inspiratory crackles* at the bases.

The likely diagnosis is diffuse interstitial lung fibrosis.

Causes of diffuse interstitial lung disease*

Acute (less likely in the exam)

Vasculitis/haemorrhage (haemoptysis, falling haemoglobin)

Eosinophilic lung disease (drugs, fungi/parasites/allergic bronchopulmonary aspergillosis (ABPA))

Infection (immunosuppression)

Acute respiratory distress syndrome (ARDS) (complicating acute severe illness often with septicaemia)

Chronic (more likely in the exam)

Cryptogenic fibrosing alveolitis† (most common cause is usual interstitial pneumonia (UIP) (histological diagnosis) with insidious onset, dyspnoea and cough)

Rheumatoid lung disease (?hands, nodules)

Systemic sclerosis (?mask-like facies, telangiectasia, sclerodactyly; see Vol. 3, Station 5, Locomotor, Case 3)

*Over 200 separate entities falling within the spectrum of diffuse parenchymal lung disease have been described.

†The umbrella term of cryptogenic fibrosing alveolitis encompasses the most common group of interstitial lung diseases – the *idiopathic interstitial pneumonias* (IIPs) that present with progressive breathlessness, diffuse interstitial infiltrates on chest X-ray and fine end-inspiratory crepitations on auscultation. The separate IIPs have widely differing prognoses and have distinct radiological and pathological patterns. Consequently, in 2002 they were reclassified by a consensus committee of the American Thoracic Society (ATS) and European Respiratory Society. *Idiopathic pulmonary fibrosis* (IPF) is the most common IIP with histological lesions of UIP and poor prognosis (median survival 2.8–4 years).

Non-specific interstitial pneumonia (NSIP) is clinically indistinguishable from IPF except that it has a better prognosis (median survival 6–7 years). *Cryptogenic organizing pneumonia* (COP), previously also known as bronchiolitis obliterans organizing pneumonia, typically presents with rapidly progressive breathlessness coming on over a 3–4-week period, with lethargy, loss of appetite, low-grade fever and cough productive of clear sputum. Plain chest X-ray reveals consolidation that is impossible to distinguish from infection or malignancy and diagnosis is made by biopsy. COP is important because, unlike many of the other interstitial lung diseases, the majority of patients achieve full resolution of their disease with corticosteroids. *Acute interstitial pneumonia* (AIP) is an idiopathic form of acute respiratory distress syndrome (ARDS). It tends to affect individuals in the fourth or fifth decade of life. There is rapidly progressive breathlessness, usually proceeding to respiratory failure within a period of a few weeks. Prognosis is poor.

Systemic lupus erythematosus (?typical rash; see Vol. 3, Station 5, Locomotor, Case 16)
Polymyositis (?proximal muscle weakness and tenderness; see Vol. 3, Station 5, Locomotor, Case 15)
Dermatomyositis (?heliotrope rash on eyes/hands and polymyositis; see Vol. 3, Station 5, Skin, Case 6)
Sjögren's syndrome (?dry eyes and mouth)
Mixed connective tissue disease
Ankylosing spondylitis (?male with fixed kyphosis and stooped 'question mark' posture; see Vol. 3, Station 5, Locomotor, Case 6)
Sarcoidosis (?extrapulmonary features, e.g. lupus pernio; see Vol. 3, Station 5, Skin, Case 19)
Extrinsic allergic alveolitis also known as hypersensitivity pneumonitis (HP) (acute pulmonary and systemic symptoms occur 6 h following inhaled allergen – ?farmer, pigeon racer, etc.)
Asbestosis (?occupational history – lagger, etc.)
Silicosis (?occupational history – slate worker or granite quarrier, etc.)
Drug induced (e.g. bleomycin, busulphan, nitrofurantoin, amiodarone)
Radiation fibrosis
Chemical inhalation (e.g. beryllium, mercury)
Poison ingestion (e.g. paraquat)
ARDS (also called acute interstitial pneumonia)

Investigations include

Chest X-ray (bilateral interstitial shadowing, reticulonodular, loss of lung volumes)*
High-resolution CT (subpleural reticulation, traction bronchiectasis, basal honeycombing, ground-glass attenuation)†
Pulmonary function tests (restrictive defect, reduced lung volumes, impaired gas transfer)
Serology:
- Eosinophilia – Churg–Strauss syndrome, eosinophilic pneumonia
- Serum calcium – raised in 5–15% of cases of sarcoidosis
- Serum angiotensin converting enzyme (ACE) – may be raised in sarcoidosis
- Antineutrophil cytoplasmic antibody (ANCA) – raised in a cytoplasmic pattern in Wegener's granulomatosis and in a perinuclear pattern in Churg–Strauss syndrome
- Rheumatoid factor, and an autoimmune profile – connective tissue disease
- Serum precipitins – may be raised in hypersensitivity pneumonitis (HP). Inflammatory markers – frequently raised in interstitial lung disease

*The distribution of changes on chest X-ray may provide diagnostic clues:
upper zone – hypersensitivity pneumonitis, ankylosing spondylitis, radiation fibrosis, sarcoidosis
lower zone – idiopathic pulmonary fibrosis, NSIP, drug-induced, connective tissue, asbestosis

†A number of interstitial lung diseases have a characteristic HRCT appearance such that biopsy is not needed to make the diagnosis in the fourth or fifth decade of life. Rapidly progressive breathlessness usually proceeding to respiratory failure within a period of a few weeks. Prognosis is poor.

Histology – gold standard for diagnosis; depending on the suspected diagnosis, tissue samples can be obtained:
  Endobronchially, e.g. sarcoidosis
  Transbronchially, e.g. sarcoidosis, amyloid, cryptogenic organizing pneumonia (COP)
  Surgically, either by open biopsy or as is more usual by video-assisted thoracoscopic surgery (VATS)
Bronchoscopy with bronchoalveolar lavage (BAL) (characteristic patterns of macrophages, lympohocytes, eosinophils and neutrophils in different diseases)
Echocardiogram (to exclude coexistent pulmonary hypertension)
Six-minute walk (patients with IPF desaturating below 88% or who manage less than 200 metres fall into a poorer prognostic group)
Gallium scanning (sarcoidosis)
24-hour urine collection for hypercalciuria in sarcoidosis – may lead to the development of calculi

Case 2 | Pneumonectomy/lobectomy

Frequency in survey: main focus of a short case or additional feature in 13% of attempts at PACES Station 1, Respiratory. Additional feature in a further 1%.

Survey note: some candidates had to discuss the chest X-ray of their pneumonectomy short case. It would usually show a 'white out' on one side, deviated trachea and compensatory hyperinflation on the other side.

Record 1

There is a deformity of the chest with *flattening* on the R/L and a *thoracotomy scar* on that side. The trachea is *deviated* to the R/L and the apex beat is *displaced* in the same direction. On the R/L *expansion* is reduced, the percussion note is *dull* and the *breath sounds* are *diminished*. There is an area of bronchial breathing in the R/L upper zone (over the grossly deviated trachea).

These findings suggest a R/L pneumonectomy.

In the patient with lobectomy, as opposed to total pneumonectomy, the signs will be more confined. For example see *Record* 2.

Record 2

There is a *deformity** of the chest with the left lower ribs *pulled in** and a left-sided *thoracotomy scar.* The *trachea* is central (may be displaced) but the *apex beat* is displaced* to the left. The *percussion note is dull* over the left lower zone and *breath sounds* are *diminished* in this area.

These signs suggest a left lower lobectomy.*

Surgical resection and the lung

Surgery has little role in the management of *small cell* carcinoma. In others, after a full assessment which includes clinical examination, lung function tests, bone and liver biochemistry, isotope bone scan, ultrasound or CT scan of the liver, mediastinal CT scan and, if necessary, mediastinoscopy, 25% of *non-small cell* lung cancers will be suitable for attempted surgical resection. The operative mortality for lobectomy is about 2–4% and this rises to about 6% for total pneumonectomy, which may be required if the tumour involves both divisions of a main bronchus or more than one lobe.

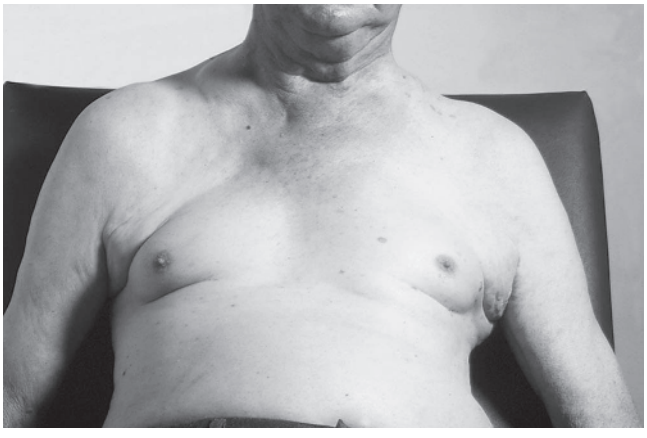
Surgical resection is often required for *solitary pulmonary nodules* of uncertain cause. The possibility of undiagnosed small cell cancer in this instance is not necessarily a reason for avoiding thoracotomy; resection of small cell lung cancer presenting as a solitary pulmonary nodule may have a 5-year survival comparable to that of other forms of nodular bronchogenic carcinoma treated surgically (approximately 25%).

Surgical resection is indicated in the treatment of *bronchiectasis* (see Station 1, Respiratory, Case 4) when other forms of treatment have failed to control symptoms, particularly if it is localized and if recurrent haemoptysis is present.

In the days before antituberculous chemotherapy, tuberculosis was sometimes treated surgically (see Station 1, Respiratory, Case 7).

*In lower lobectomy there may not be deformity (or it may be subtle) and the ribs may not be pulled in and apex beat not displaced because the remaining lung may expand to occupy the space.

(a)



(b)

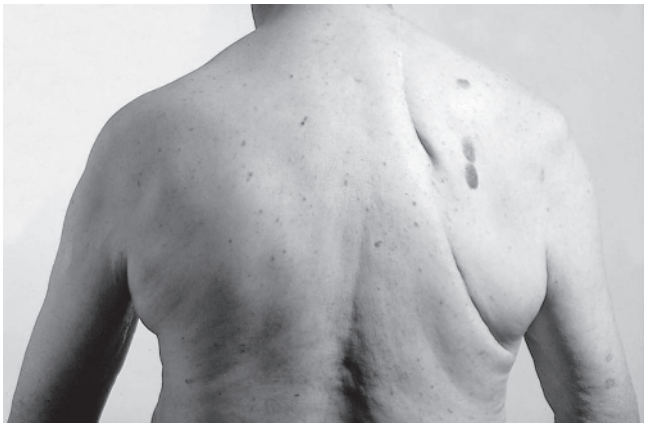


Figure C1.1 (a) Deformity of the right chest with flattening. (b) Right-sided thoracotomy scar on the back.

Case 3 | Chronic bronchitis and emphysema*

Frequency in survey: main focus of a short case or additional feature in 11% of attempts at PACES Station 1, Respiratory.

Survey note: usually the patients in the examination fall between the extremes of the classic *Records* below.

Record 1

This thin man (with an anxious, drawn expression) presents the classic 'pink puffer' appearance. He has *nicotine staining* of the fingers. He is tachypnoeic at rest with *lip pursing* during expiration, which is *prolonged*. The suprasternal notch to cricoid distance is reduced (a sign of hyperinflation; normally >3 finger breadths). His chest is *hyperinflated*, *expansion* is mainly *vertical* and there is a *tracheal tug*. He uses his *accessory muscles* of respiration at rest and there is *indrawing* of the *lower ribs* on inspiration (due to a flattened diaphragm). The percussion note is hyperresonant, obliterating cardiac and hepatic dullness, and the breath sounds are quiet (this is so in classic pure emphysema – frequently, though, wheezes are heard due to associated bronchial disease).

These are the physical findings of a patient with emphysema (inspiratory drive often intact).†

Record 2

This (male) patient (who smokes, lives in a foggy city, works amid dust and fumes, and has probably had frequent respiratory infections) presents the classic 'blue bloater' appearance. He has *nicotine staining* on the fingers. He is stocky and *centrally cyanosed* with suffused conjunctivae. His chest is *hyperinflated*, he uses his *accessory muscles* of respiration; there is *indrawing* of the *intercostal muscles* on inspiration and there is a *tracheal tug* (both signs of hyperinflation). His pulse is 80/min, the venous pressure is not elevated (may be raised with ankle oedema and hepatomegaly if cor pulmonale is present), the trachea is central, but the suprasternal notch to cricoid distance is reduced. *Expansion* is equal but *reduced* to 2 cm and the percussion note is resonant; on auscultation, the expiratory phase is prolonged and he has widespread *expiratory rhonchi* and (may be) coarse inspiratory crepitations. (His forced expiratory time (see Section B, Examination *Routine* 3) is 8 sec.) There is no flapping tremor of the hands (unless he is in severe hypercapnoeic respiratory failure in which case ask to examine the fundi – ?papilloedema).

These are the physical findings of advanced chronic bronchitis‡ (inspiratory drive often reduced) producing chronic small airways obstruction (and, if ankle oedema, etc., right heart failure due to cor pulmonale).

*Chronic obstructive pulmonary disease (COPD), which encompasses both chronic bronchitis and emphysema, includes the criteria of an obstructive spirometry (i.e. one can have chronic bronchitis only without an obstructive spirometry and hence such a person technically does not have COPD but only chronic bronchitis).

†Emphysema is, however, a histological or CT diagnosis.

‡Chronic bronchitis, though, is defined as sputum production (not due to specific disease such as bronchiectasis or TB) on most days for 3 months of the year for 2 consecutive years.

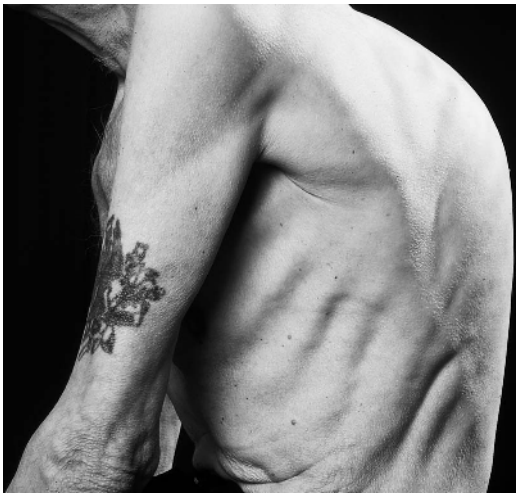


Figure C1.2 Hyperinflated rib cage. Note indrawing of intercostal muscles.

Causes of emphysema

Smoking (usually associated with chronic bronchitis; mixed centrilobular and panacinar)

α1-antitrypsin deficiency (?young patient; lower zone emphysema, panacinar in type; ?icterus, hepatomegaly, etc. of hepatitis or cirrhosis)

Coal dust (centrilobular emphysema – simple coal worker's pneumoconiosis – only minor abnormalities of gas exchange)

Macleod's (Swyer–James) syndrome – rare (unilateral emphysema following childhood bronchitis and bronchiolitis with subsequent impairment of alveolar growth; breath sounds diminished on affected side – more likely to meet this in the 'pictures' section of MRCP Part 2 written examination)

Record 1 (continuation)

The decreased breath sounds over the . . . zone of the R/L lung of this patient with emphysema raises the possibility of an emphysematous bulla.

Investigations include:

Pulmonary function tests:

- FEV_1 <80% predicted value for height, age and sex (diagnostic)
- FEV_1/FVC ratio <0.7 (diagnostic)
- TLC elevated
- RV elevated
- TLCO or KCO (transfer factor) reduced

Reversibility testing may be helpful in distinguishing COPD from asthma

Chest X ray:

- Hyperinflation (>7 posterior ribs visible)
- Flattened diaphragm
- Irregular distribution of lung vasculature
- Bullae

FBC – ?polycythaemia

Arterial blood gases

Pulse oximetry

α1-antitrypsin – young age or family history

High-resolution CT thorax:

- Bullae
- Destruction of normal lung parenchyma and architecture

ECG and echocardiography if possible cor pulmonale

Functional assessement – 6-min walking test with oximetry during exercise

Case 4 | Bronchiectasis

Frequency in survey: main focus of a short case or additional feature in 9% of attempts at PACES Station 1, Respiratory.

Record

This patient (who may be rather *underweight, breathless* and *cyanosed*) has *clubbing* of the fingers (not always present) and a frequent *productive cough* (the patient may cough in your presence;* there may be a *sputum pot* by the bed). There are (may be) *inspiratory clicks* heard with the unaided ear. There are *crepitations* over the . . . zone(s) (the area(s) where the bronchiectasis is) and (may be) widespread *rhonchi.*

The diagnosis could well be bronchiectasis. The frequent productive cough and inspiratory clicks are in favour of this. Other possibilities (clubbing and crepitations) are:

1 Fibrosing alveolitis* (marked sputum production and clicks are against this)
2 Sarcoidosis
3 Post TB
4 Lung abscess
5 Carcinoma of the lung (?heavy nicotine staining, lymph nodes, etc.).

Possible causes of bronchiectasis

Respiratory infection in childhood (especially whooping cough, measles, TB)

Cystic fibrosis (young, thin patient, may have malabsorption and steatorrhoea; see Station 1, Respiratory, Case 17)

Bronchial obstruction due to foreign body, carcinoma, granuloma (tuberculosis, sarcoidosis) or lymph nodes (e.g. tuberculosis)

Fibrosis (complicating tuberculosis, unresolved or suppurative pneumonia with lung abscess, mycotic infections or sarcoidosis)

Hypogammaglobulinaemia (congenital and acquired)

Allergic bronchopulmonary aspergillosis (proximal airway bronchiectasis)

Marfan's syndrome (?tall, long extremities, high-arched palate; see Station 3, Cardiovascular, Case 13)

Yellow nail syndrome (?excessively curled yellow nails with bulbous fingertips; lymphoedema of extremities; see Vol. 3, Station 5, Skin, Case 12)

Congenital disorders such as sequestrated lung segments, bronchial atresia and Kartagener's syndrome†

Associated with smoking-related chronic obstructive pulmonary disease (COPD)‡

Investigations include

Chest X-ray ('tramlines', indicating thickened airways, 'ring shadows', and segmental or lobar collapse)

High-resolution thin-section CT thorax (tramlines (non-tapering of bronchi), the 'signet ring' sign (end-on dilated bronchi larger than accompanying pulmonary artery), crowding of the bronchi with associated lobar volume loss, mucous plugging of

*It is worth asking the patient to 'give a cough' as it may help you differentiate bronchiectasis from fibrosing alveolitis.

†The features of Kartagener's syndrome are dextrocardia, situs inversus, infertility, dysplasia of frontal sinuses, sinusitis and otitis media. Patients have ciliary immotility.

‡The majority of bronchiectasis patients seen in chest clinics nowadays have no obvious cause but also have COPD from smoking. One study showed that a distinct proportion of COPD patients (up to 30%) also have bronchiectasis (as confirmed by CT).

dilated bronchi (flame and blob sign), thickening and plugging of small airways resulting in numerous nodular and V- or Y-shaped opacities)
Arterial blood gas
Exercise capacity
Pulmonary function tests (combined restrictive/obstructive features)
Sputum culture (NB: *Pseudomonas* colonization) including AFB
Total immunoglobulin levels of IgG, IgM, IgA, IgE
Specific antibodies to pneumococcus and tetanus antigens
Aspergillus radioallergosorbent test (IgE) and precipitins (IgG)
Rheumatoid factor
Protein electrophoretic strip
α1-antitrypsin
Sweat test, nasal potentials, cystic fibrosis genotyping
Cilia studies (if nasal mucociliary clearance is prolonged or nasal nitric oxide low, proceed to light microscopy of ciliary beat frequency and then electron microscopy)

Case 5 | Dullness at the lung base

Frequency in survey: main focus of a short case or additional feature in 7% of attempts at PACES Station 1, Respiratory.

Record

The pulse is regular and the venous pressure is not elevated. The trachea is central,* the expansion is normal, but the percussion note is *stony dull* at the R/L base(s), with *diminished* tactile *fremitus* and vocal *resonance*, and *diminished breath sounds*. There is (may be) an area of bronchial breathing above the area of dullness.

The diagnosis is R/L pleural effusion.†

Causes of pleural effusion

Exudate (protein content >30 g L^{-1})‡

Bronchial carcinoma (?nicotine staining, clubbing, radiation burns on chest, lymph nodes)

Secondary malignancy (?evidence of primary especially breast, lymph nodes, radiation burns)

Pulmonary embolus and infarction (?DVT; blood-stained fluid will be found at aspiration)

Pneumonia (bronchial breathing/crepitations, fever, etc.)§

Tuberculosis

Mesothelioma (asbestos worker, ?clubbing)

Rheumatoid arthritis (?hands and nodules)

Systemic lupus erythematosus (?typical rash)

Lymphoma (?nodes and spleen)

Transudate (protein content <30 g L^{-1})

Cardiac failure (?JVP ↑, ankle and sacral oedema, large heart, tachycardia, S_3 or signs of a valvular lesion)

Nephrotic syndrome (?generalized oedema, patient may be young; see Station 1, Abdominal, Case 20)

Cirrhosis (?ascites, generalized oedema, signs of chronic liver disease; see Station 1, Abdominal, Case 3)

*The trachea may be deviated if the effusion is very large. A large effusion without any mediastinal shift (clinically and on chest X-ray) raises the possibility of collapse as well as effusion.

†On your initial inspection there may be biopsy or aspiration needle marks, or the marks of a sticking plaster removed by the invigilator at the beginning of the day, as a clue that you are going to find a pleural effusion.

‡Although the protein content >30 g L^{-1} is not necessarily 100% sensitive and specific, it is still the most simple way of dividing exudate from transudate. Also, in exudates, the fluid to serum protein ratio is usually greater than 0.5, with an LDH of >200 IU and a fluid to serum LDH ratio of >0.6.

§Up to 57% of patients with pneumonia develop a pleural effusion and, of these, over 4% develop frank pleural infection. The associated mortality is about 20%. Diagnostic thoracentesis should be performed in all suspected cases, using image guidance if the effusion is small or heavily loculated. Aspiration of overt pus confirms empyema. About 40% of infected pleural effusions are culture negative and, in this situation, biochemical pleural fluid markers (pH, LDH, white blood cell count and glucose) are central in establishing a diagnosis. Pleural fluid pH <7.2 suggests pleural infection. Pleural fluid glucose concentration can be used if pH measurement is unavailable. The amplification of bacterial DNA from culture-negative fluid improves diagnostic sensitivity.

Other causes of pleural effusion

Meigs' syndrome (ovarian fibroma)
Subphrenic abscess (?recent abdominal disease or surgery)
Peritoneal dialysis
Hypothyroidism (?facies, pulse, ankle jerks)
Pancreatitis (more common on the left; fluid has high amylase)
Dressler's syndrome (recent myocardial infarction, ?pericardial friction rub)
Trauma
Asbestos exposure
Yellow nail syndrome (yellowish-brown beaked nails usually associated with lymphatic hypoplasia; see Vol. 3, Station 5, Skin, Case 12)
Chylothorax (trauma or blockage of a major intrathoracic lymphatic, usually by a neoplastic process)

Other causes of dullness at a lung base

Raised hemidiaphragm (e.g. hepatomegaly, phrenic nerve palsy)
Basal collapse
Collapse/consolidation (if the airway is blocked by, for example, a carcinoma there may be no bronchial breathing)
Pleural thickening (e.g. old TB or old empyema or asbestos-induced with or without mesothelioma)

Pleural biopsy

Biopsy is carried out using an 'Abram's biopsy needle'. The specificity for detecting TB and malignancy is better with a pleural biopsy than with a pleural aspiration on its own. Remember that any biopsies for TB cultures should be placed in normal saline and not formalin.

Thoracoscopy

This is a technique involving visual inspection of the pleural cavity for diagnostic and therapeutic purposes (e.g. pleurodesis).

Case 6 | Rheumatoid lung

Frequency in survey: main focus of a short case or additional feature in 4% of attempts at PACES Station 1, Respiratory.

Record

There is (may be) cyanosis (there may also be dyspnoea) and the principal finding in the chest is of *fine inspiratory crackles* (or crepitations – whichever term you prefer) on auscultation at both bases.

In view of the *rheumatoid* changes (see Vol. 3, Station 5, Locomotor, Case 1) in the *hands* (there may also be clubbing), the likely diagnosis is pulmonary fibrosis associated with rheumatoid disease (rheumatoid lung).

Classic fibrosing alveolitis develops in 2%* of patients with rheumatoid arthritis and has a poor prognosis. It may progress to a honeycomb appearance on chest X-ray, bronchiectasis, chronic cough and progressive dyspnoea. Pulmonary function tests show reduced diffusion capacity, diminished compliance and a restrictive ventilatory pattern.

Gold, used in the therapy of rheumatoid arthritis, can also induce interstitial lung disease; it is indistinguishable from rheumatoid pulmonary fibrosis except that the gold-induced disease may reverse when the drug is discontinued.

Other pulmonary manifestations of rheumatoid disease

Pleural disease.† Though frequently found at autopsy, rheumatoid pleural disease is usually asymptomatic. The rheumatoid patient may have a pleural rub or pleural effusion but only occasionally would the latter be of sufficient size to cause respiratory limitation. The pleural fluid at diagnostic aspiration is never blood-stained and often contains immune complexes and rheumatoid factor; it is high in protein (exudate) and LDH and low in glucose, C3 and C4. The white count in the pleural fluid is variable but usually <5000/μL.

Intrapulmonary nodules. Single or multiple radiological nodules may be seen in the lung parenchyma before or after the onset of arthritis. They are usually asymptomatic but may become infected and cavitate. As they have a predilection for the upper lobes and can cause haemoptysis, they can resemble tuberculosis or even carcinoma. They can rupture into the pleural space, causing a pneumothorax. Massive confluent pulmonary nodules may be seen in rheumatoid lungs in association with pneumoconiosis (*Caplan's syndrome*).

Obliterative bronchiolitis. Rarely, small airways obstruction may develop into a necrotizing bronchiolitis, classically associated with dyspnoea, hyperinflation and a high-pitched expiratory wheeze or 'squawk' on auscultation. This complication may also result from therapy with gold or penicillamine.

Two other manifestations are *pulmonary arteritis* (reminiscent of polyarteritis nodosa) and *apical fibrobullous disease*.

*Though fibrosing alveolitis becomes overt in only 2% of patients, 25% of patients with rheumatoid arthritis show interstitial changes on chest X-ray and 50% have reduced diffusion capacity, suggesting that subclinical fibrosing alveolitis is common. There is no relationship between the extent of lung disease and the titre of rheumatoid factor.

†Though our surveys have not thrown up a case of pleural effusion and rheumatoid hands, it would nevertheless be worth a glance at the hands of a patient with a pleural effusion for possible rheumatoid changes as well as for clubbing.

Case 7 | Old tuberculosis

Frequency in survey: main focus of a short case or additional feature in 3% of attempts at PACES Station 1, Respiratory.

Record 1

The trachea is *deviated* to the R/L. The R/L upper chest shows *deformity* with *decreased expansion, dull percussion* note, *bronchial breathing* and *crepitations*. The apex beat is (may be) *displaced* to the R/L. There is a *thoracotomy sca*r posteriorly with evidence of rib resections.

The patient has had a R/L thoracoplasty for treatment of tuberculosis before the days of chemotherapy.

Record 2

The tracheal deviation to the R/L and the diminished expansion and crackles at the R/L apex suggest R/L apical fibrosis.

Old tuberculosis is the likely cause.

Record 3

Expansion is diminished on the R/L with dullness and reduced/absent breath sounds at the R/L lung base. There is a R/L supraclavicular scar (there may also be crepitations).

The patient has had a phrenic nerve crush for TB before the days of chemotherapy.

Treatment of pulmonary tuberculosis*

First 2 months (intensive phase†):

- isoniazid
- rifampicin
- pyrazinamide
- ethambutol

Four-month continuation phase:

- rifampicin
- isoniazid

*Treatment does not have to be initiated in hospital and patients do not need to be kept in hospital if their treatment is initiated there. Treating patients at home does not put their co-habitants at increased risk.

†The aim of the intensive phase is to render the patient non-infectious. Ninety percent of the mycobacteria are killed within the first week. With fewer organisms, the risk of secondary drug resistance falls until after 2 months it is safe to reduce to two bactericidal drugs.

Case 8 | Chest infection/consolidation/pneumonia

Frequency in survey: main focus of a short case or additional feature in 3% of attempts at PACES Station 1, Respiratory.

Record

There is reduced movement of the R/L side of the chest. There is *dullness* to percussion over . . . (describe where) with *bronchial breathing*, *coarse crepitations*, *whispering pectoriloquy* and a *pleural friction rub.*

These features suggest consolidation (say where).

Most common causes of consolidation

Bacterial pneumonia (pyrexia, purulent sputum, haemoptysis, breathlessness)

Carcinoma (with infection behind the tumour; ?clubbing, wasting, etc; see Station 1, Respiratory, Case 13)

Pulmonary infarction (fever less prominent, sputum mucoid, occasionally haemoptysis and blood-stained pleural effusion)

Causes of community-acquired pneumonia in hospital studies*

Microbe	Percentage
Streptococcus pneumoniae	39
Chlamydia pneumoniae	13.1
Mycoplasma pneumoniae	10.8
Haemophilus influenzae	5.2
Legionella spp	3.6
Chlamydia psittaci	2.6
Staphylococcus aureus	1.9
Moraxella catarrhalis	1.9
All viruses	12.8

Investigations may include

Chest X-ray (consolidation, air bronchograms, cavitation, parapneumonic effusions)

Full blood count and inflammatory markers

Renal and hepatic indices (derangement may indicate increased severity or a multisystem involvement of atypical pneumonias – *Mycoplasma*, *Chlamydia*, *Legionella*)

Oximetry

Arterial blood gases

Sputum and blood cultures

***Recommended antimicrobial therapy if microbe identified**

Streptococcus pneumoniae	Amoxicillin
Chlamydia pneumoniae	Clarithromycin
Mycoplasma pneumoniae	Erythromycin
Haemophilus influenzae	Co-amoxiclav
Legionella	Clarithromycin +/– rifampicin
Chlamydia psittaci	Tetracycline
Staphylococcus aureus	Flucloxacillin +/– rifampicin
Methicillin-resistant *Staph. aureus*	Vancomycin
Pseudomonas aeruginosa	Ceftazidime + aminoglycoside

Urinary antigen tests for pneumococcus and *Legionella* infections
Paired serological tests for other atypical pneumonias
Diagnostic pleural tap

The CURB-65 score

Confusion
Urea >7 mmol/L
Respiratory rate >30/min
Blood pressure (BP); systolic BP <90 mmHg or diastolic BP <60 mmHg
65 years and above

One or fewer of the above is associated with a low mortality (1.5%) and perhaps suitability for home treatment, whereas three or more features are suggestive of a severe pneumonia with a higher mortality (22%) and the advisability of consideration of ICU support.

Case 9 | Yellow nail syndrome

Frequency in survey: main focus of a short case or additional feature in 2% of attempts at PACES Station 1, Respiratory.

Survey note: see Vol. 2, Section F, Anecdotes 89 and 90.

Yellow nail syndrome is dealt with in Vol. 3, Station 5, Skin, Case 12.

Case 10 | Kyphoscoliosis

Frequency in survey: main focus of a short case or additional feature in 2% of attempts at PACES Station 1, Respiratory.

Survey note: see Vol. 2, Section F, Anecdotes 91 and 92.

Record

On inspection of the chest from the side (of this patient whom you have been told has been referred for investigation of *breathlessness*), I note an *increase in thoracic curvature*. There is no suggestion of any prominent angular features indicative of a gibbus and no evidence of any reversal of the normal lumbar lordosis. Further inspection of the patient when bending forwards shows restriction in the mobility of the spine. Inspection of the back shows no evidence of neurofibromatosis, spina bifida (hairy patch), thoracotomy scars, or spinal surgery scars. Whilst sitting down and *bending forwards*, the *curvature remains*, suggesting that the scoliosis is fixed. There is a *rib hump*. Palpation of the spine reveals no tenderness. Sliding the fingers down the spine reveals no evidence of a palpable step at the lumbo-sacral junction (feature of spondylolisthesis). Whilst the patient is standing, he demonstrates that he cannot touch his toes on flexion, and there is reduced extension when asked to bend back, whilst keeping the pelvis steady. There is evidence of reduced lateral flexion and rotation. There are no other features suggestive of old poliomyelitis or any obvious muscle atrophy in any of the limbs. The patient is (may be) *cyanosed*. *Chest expansion* is *reduced* but percussion and breath sounds are normal.

The patient demonstrates a scoliosis, most likely idiopathic in origin. The breathlessness is likely to be due to hypoventilation secondary to the deformity.

Scoliosis is a lateral curvature of the spine. Deformity of the spine will suggest a structural scoliosis rather than a non-structural scoliosis where the vertebrae are normal and the curvature can be due to compensatory reasons, e.g. tilting of the pelvis, sciatic due to unilateral muscle spasm or postural. In structural scoliosis, the deformity cannot be altered by a change in posture.

Kyphosis is the term used to describe the increased forward curvature. Therefore kyphoscoliosis describes an abnormal curvature of the spine in both coronal and sagittal planes. There may be varying degrees of kyphosis and scoliosis in an individual patient.

Causes of kyphoscoliosis include

1 Congenital, e.g. hemivertebra, fused verterbrae or absent/fused ribs

2 Paralytic secondary to the loss of supportive action of the trunk and spinal muscles, e.g. anterior poliomyelitis

3 Neuropathic as a complication of neurofibromatosis, spina bifida, cerebral palsy, syringomyelia, Friedreich's ataxia

4 Myopathic, e.g. muscular dystrophy (Duchenne's muscular dystrophy, arthrogryposis)

5 Metabolic, e.g. cystine storage disease, Marfan's syndrome
6 Idiopathic, the most common (approximately 65% of all cases of scoliosis)

Prognosis depends on age of onset, the level of the spine affected (the higher the level, the worse the prognosis), the number of primary curves, the type of structural scoliosis (congenital versus idiopathic).

Causes of kyphosis *only* include

1 Postural
2 Degenerative spine (e.g. osteoporosis)
3 Scheuermann's disease
4 Congenital
5 Nutritional (vitamin D deficiency)
6 Post tuberculosis of the spine (gibbus)
7 Post traumatic (vertebral fractures)

Investigations include

X-ray of spine and chest – concavity, with displacement of the spine and narrowing of the pedicles. On convexity there will be widening of the rib spaces. Angular deformity can be measured more accurately using the Cobb method*

MRI of the spine may be considered to look at the spinal cord

Spirometry – restrictive picture with a reduced FEV_1 and FVC. FVC of under 1 L will increase the risk of hypercapnic respiratory failure secondary to hypoventilation

Non-invasive ventilation

The need for domiciliary NIV should be assessed in the presence of breathlessness, poor sleep quality, and type II respiratory failure from blood gas analysis. Long-term follow-up by NIV specialists is necessary. Pulmonary hypertension is common in untreated respiratory failure.

*The Cobb angle, named after the American orthopaedic surgeon John Robert Cobb (1903–1967), was originally used to measure coronal plane deformity on anteroposterior plain radiographs in the classification of scoliosis and has subsequently been adapted to classify sagittal plane deformity. It is defined as the angle formed between a line drawn parallel to the superior endplate of one vertebra above the deformity and a line drawn parallel to the inferior endplate of the vertebra one level below the deformity.

Case 11 | Stridor

Frequency in survey: main focus of a short case or additional feature in 1% of attempts at PACES Station 1, Respiratory.

Record

The patient is comfortable at rest. From the *bedside* I can hear a noisy, *high-pitched sound* with each inspiration. Her respiratory rate is 12/min. Chest expansion is normal, resonance is normal and auscultation reveals *normal vesicular breath sounds* and no added sounds. There is (may be) a *healed tracheostomy scar* present.

In view of the tracheostomy scar, it is likely that the inspiratory stridor is due to tracheal stenosis following prolonged ventilatory support via a tracheostomy.

Inspiratory stridor usually implies upper airways obstruction and tracheal narrowing (extrathoracic).

Causes of inspiratory stridor

1 Acute (infective epiglottitis, croup)
2 Trauma (foreign body, smoke inhalation)
3 Chronic (neoplastic, tracheal stenosis)

Expiratory stridor is usually found with lower intrathoracic obstruction.

Causes of expiratory stridor

1 Foreign body
2 Intraluminal mass/neoplasm
3 Lower tracheal stenosis
4 Bronchial stenosis

Causes of tracheal stenosis

1 Congenital (webs, tracheomalacia), *or*
2 Acquired (tracheostomy or intubation)
3 Post trauma, *or*
4 Post infections (e.g. TB)
5 Neoplasia

Management would include referral to thoracic surgeon who would consider rigid bronchoscopy with possible dilation and/or stent insertion. Primary reconstruction may be considered as definitive treatment.

Case 12 | Marfan's syndrome

Frequency in survey: main focus of a short case or additional feature in 1% of attempts at PACES Station 1, Respiratory.

Survey note: see Vol. 2, Section F, Experience 27 and Anecdote 88.

Marfan's syndrome is dealt with in Vol. 3, Station 5, Locomotor, Case 9.

Case 13 | Carcinoma of the bronchus

Frequency in survey: main focus of a short case or additional feature in 0.8% of attempts at PACES Station 1, Respiratory.

Survey note: candidates reported a variety of signs. The three *records* given are typical.

Record 1

There is *clubbing* of the fingers which are *nicotine-stained.* There is a hard *lymph node* in the R/L supraclavicular fossa. The pulse is 80/min and regular, and the venous pressure is not raised. The trachea is central, chest expansion normal, but the percussion note is *stony dull* at the R/L base and *tactile fremitus, vocal resonance* and *breath sounds* are all *diminished* over the area of dullness.

The likely diagnosis is carcinoma of the bronchus causing a *pleural effusion.*

Record 2

The patient is *cachectic*. There is a *radiation burn* on the R/L upper chest wall. There is *clubbing* of the fingers which are *nicotine-stained*. The pulse is 80/min, venous pressure is not elevated and there are no lymph nodes. The *trachea* is *deviated* to the R/L and *expansion* of the R/L upper chest is *diminished*. *Tactile vocal fremitus* and *resonance* are *increased* over the upper chest where the *percussion note* is *dull* and there is an area of *bronchial breathing*.

It is likely that this patient has had radiotherapy for carcinoma of the bronchus which is causing *collapse* and *consolidation of the* R/L *upper lung.*

Record 3

There is a *radiation burn* on the chest. There are *lymph nodes* palpable in the R/L axilla. The trachea is central. I did not detect any abnormality in expansion, vocal fremitus, vocal resonance or breath sounds, but there is *wasting* of the *small muscles* of the R/L *hand*, and *sensory loss* (plus pain) over the *T1** dermatome. There is a R/L *Horner's syndrome* (see Station 3, CNS, Case 41).

The diagnosis is *Pancoast's syndrome* (due to an apical carcinoma of the lung involving the lower brachial plexus and the cervical sympathetic nerves).

Other complications of carcinoma of the bronchus

1 Other local effects such as:
Superior vena cava obstruction (?oedema of the face and upper extremities, suffusion of eyes, fixed engorgement of neck veins and dilation of superficial veins, etc; see Station 1, Respiratory, Case 22)
Stridor (often associated with superior vena cava obstruction; dysphagia may occur)

2 Metastases and their effects (pain, ?hepatomegaly, neurological signs, etc.)

*The weakness, sensory loss and especially pain may be more widespread (C8, T1, 2).

3 Non-metastatic effects such as:

Hypertrophic pulmonary osteoarthropathy (?clubbing plus pain and swelling of wrists and/or ankles – subperiosteal new bone formation on X-ray)

Neuropathy (peripheral neuropathy – sensory, motor or mixed; cerebellar degeneration and encephalopathy; proximal myopathy, polymyositis, dermatomyositis, reversed myasthenia – Eaton–Lambert syndrome)

Endocrine (inappropriate antidiuretic hormone, ectopic ACTH, ectopic parathormone or parathormone-related peptide,* carcinoid)

Gynaecomastia (if rapidly progressive and painful may be due to a HCG-secreting tumour)

Thrombophlebitis migrans (?DVT)

Non-bacterial thrombotic endocarditis

Anaemia (usually normoblastic; occasionally leucoerythroblastic from bone marrow involvement)

Pruritus

Herpes zoster (see Vol. 3, Station 5, Skin, Case 32)

Acanthosis nigricans (grey-brown/dark brown areas in the axillae and limb flexures, in which skin becomes thickened, rugose and velvety with warts; see Vol. 3, Station 5, Skin, Case 48)

Erythema gyratum repens (irregular wavy bands with a serpiginous outline and marginal desquamation on the trunk, neck and extremities)

Record 4 (lobectomy)

There is a R/L *thoracotomy scar*. The *trachea* is *deviated* to the R/L. On the R/L side *chest expansion* is *diminished*, percussion note more resonant and breath sounds are harsher. The patient has had a R/L lobectomy to remove a tumour, resistant lung abscess or localized area of bronchiectasis.

Treatment for lung cancer

1 Surgical resection for limited disease

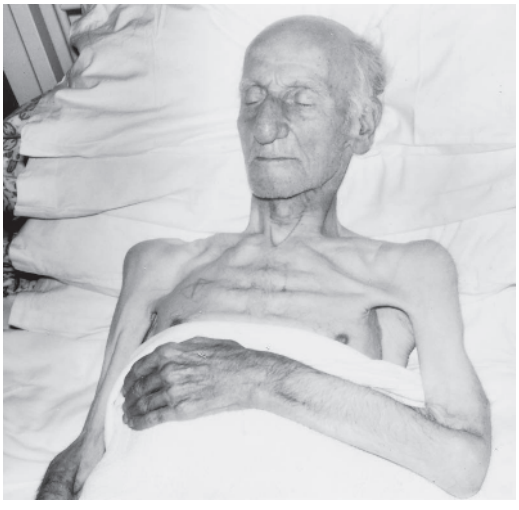
2 Chemotherapy/radiotherapy or combination

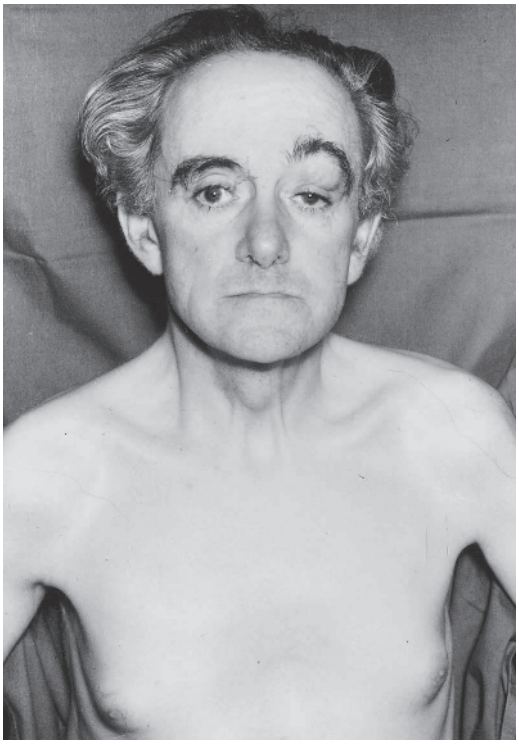
3 Radiofrequency ablation (alternative to surgery in early stages)

4 Palliative care

*Hypercalcaemia may also be due to bone secondaries.

(a)





(b)

Figure C1.3 (a) Cachexia due to carcinoma of the bronchus (note radiotherapy ink marks). (b) Pancoast's tumour (note gynaecomastia and left Horner's syndrome).

Case 14 | Klippel–Feil syndrome

Frequency in survey: main focus of a short case or additional feature in 0.8% of attempts at PACES Station 1, Respiratory.

Survey note: see Vol. 2, Section F, Anecdote 91.

Klippel–Feil syndrome is dealt with in Vol. 3, Station 5, Other, Case 7.

Case 15 | Kartagener's syndrome

Frequency in survey: main focus of a short case or additional feature in 0.7% of attempts at PACES Station 1, Respiratory.

Record

This patient (who may be rather *underweight, breathless* and *cyanosed*) has *clubbing* of the fingers (not always present) and a frequent *productive cough* (the patient may cough in your presence;* there may be a *sputum pot* by the bed). There are (may be) *inspiratory clicks* heard with the unaided ear. There are *crepitations* over the . . . zone(s) (the area(s) where the bronchiectasis is) and (may be) widespread *rhonchi*.

The apex beat is *not palpable on the left side*, and the heart sounds can barely be heard there but can be heard instead on the *right* side. The patient has dextrocardia.†

The findings in the chest are in keeping with bronchiectasis and this in association with dextrocardia suggests that the diagnosis is Kartagener's syndrome.

Features of Kartagener's syndrome

1 Dextrocardia
2 Bronchiectasis
3 Situs inversus
4 Infertility
5 Dysplasia of frontal sinuses
6 Sinusitis
7 Otitis media

Patients with Kartagener's syndrome have ciliary immotility.

*It is worth asking the patient to 'give a cough' as it may help you differentiate bronchiectasis from fibrosing alveolitis.
†Consider the possibility of this diagnosis if you cannot feel the apex beat and then have difficulty hearing the heart sounds. As you gradually move the stethoscope towards the right side of the chest, they get louder.

Case 16 | Lung transplant

Frequency in survey: main focus of a short case or additional feature in 0.7% of attempts at PACES Station 1, Respiratory.

Record 1

This *young* man (who has had a *major operative procedure* for a *severe chronic respiratory problem*) is not breathless at rest. His respiratory rate is 12/min. He is bilaterally clubbed. He has a *mid-sternotomy scar*. Expansion is equal and normal both sides. Percussion is normal and *breath sounds* are *vesicular* with no added sounds in both lungs.

In view of his age I suspect that the chronic respiratory problem requiring a surgical procedure was cystic fibrosis and I suspect that he has had a double lung transplantation that has been successful.

Record 2

This middle-aged man (who had a right lung transplant and has been increasingly breathless in recent months) has an *increased respiratory rate* of 18/min. He is taking *oxygen* 2 L min^{-1} via nasal cannulae. He is bilaterally clubbed. He has features of *Cushing's* syndrome. There is a *right thoracotomy scar*. He has reduced expansion in both lungs. The left base is dull to percussion. There are fine inspiratory crackles to the mid-zones in the left lung and a few scattered *inspiratory squeaks* in the right lung.

The findings in the left lung suggest that the lung transplant was for pulmonary fibrosis. It may be that he has had recurrent episodes of acute rejection during recent months and has now developed bronchiolitis obliterans syndrome (BOS) for which he takes large doses of steroids and is on continuous oxygen therapy. (He will have a spirometry recording book to demonstrate the fall in FEV_1 and FVC over the last 12 months and may be on the active retransplant list – look for the bleeper.)

Indications for lung transplantation

1 Pulmonary vascular: primary pulmonary hypertension, pulmonary hypertension secondary to systemic disease and Eisenmenger's syndrome
2 Restrictive pulmonary diseases: idiopathic pulmonary fibrosis, fibrosis secondary to connective tissue disease, sarcoidosis and chronic allergic alveolitis
3 Obstructive diseases: emphysema with or without α1-antitrypsin deficiency, Langerhans cell granulomatosis and lymphangioleiomyomatosis
4 Suppurative disease including cystic fibrosis and bronchiectasis

Complications of lung transplantation

1 Perioperative, e.g. dehiscence of graft
2 Infection: viral, especially cytomegalovirus, but also herpes simplex; bacterial; fungal – *Candida*, *Aspergillus*; other opportunistic infections, e.g. pneumocystis
3 Rejection: may be hyperacute (within hours) or acute; most patients experience one or two episodes during the first 6 months. *Bronchiolitis obliterans syndrome* is progressive airways obstruction with rapid progression and poor survival. Acute

rejection is a major prognostic factor. It is characterized by non-productive cough, dyspnoea and malaise. Pulmonary function shows irreversible airflow obstruction, reduced total lung capacity and gas transfer. No effective treatment is available. A regimen of immunosuppressives is commonly employed. Retransplantation may be considered.

4 Side-effects of drugs, e.g. azathioprine, cyclosporin, corticosteroids

Case 17 | Cystic fibrosis

Frequency in survey: main focus of a short case or additional feature in 0.7% of attempts at PACES Station 1, Respiratory.

Record

This young patient (who is usually *underweight*, of *short stature* and rather pale, but may also be *breathless* and *cyanosed*) has *clubbing* of the fingers (often present) and a *productive cough* (there may be a *sputum pot* by the bed). There are (may be) *inspiratory clicks* and *expiratory wheeze* (heard with the unaided ear). There are *crepitations* over . . . (the area of bronchiectasis – state where). There is (may be) widespread *polyphonic expiratory wheeze.*

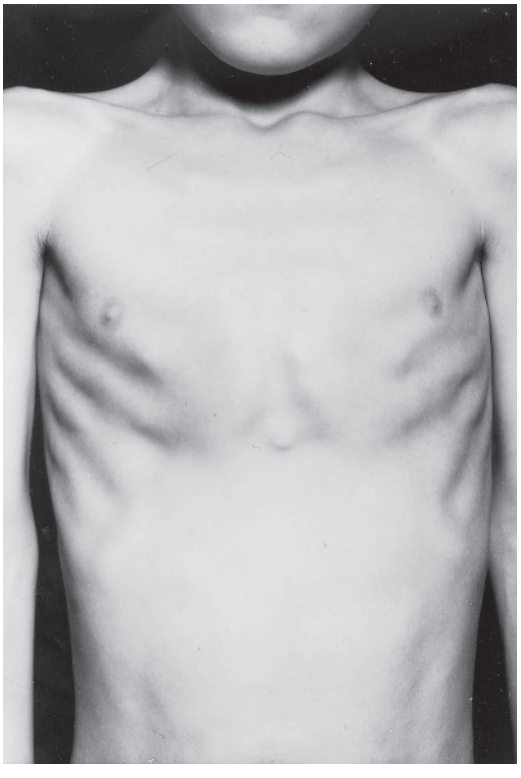
These features suggest *bronchiectasis* (see Station 1, Respiratory, Case 4) and as this is a young patient, this suggests that the underlying disorder is cystic fibrosis. If so, the patient is also likely to have *pancreatic insufficiency* and *malabsorption*.* The diagnosis can be confirmed by the *sweat sodium test.*†

Some patients have well-developed *cor pulmonale* with *cyanosis*, *ankle oedema* and *right heart failure.*

Cystic fibrosis is a genetic disorder (autosomal recessive) and a child born to two heterozygote carriers has a 25% chance of having the disease. The disorder usually occurs in Caucasians; Africans and Asians are seldom affected.

*In most cases there is a history of frequent large foul stools which are difficult to flush down. These patients are underachievers in weight and height for their age; they have a good appetite, steatorrhoea and a protuberant abdomen. Hepatic symptoms are relatively uncommon but there may be *jaundice*. Sometimes there may be *portal hypertension*, glycosuria, biliary cirrhosis, cholelithiasis, intussusception and aspermia.

†A history of recurrent respiratory infections and of gastrointestinal symptoms, especially recurrent abdominal pain and faecal impaction, is highly suggestive of cystic fibrosis. The sodium and chloride levels are in excess of $70\,mmol\,L^{-1}$ in the sweat, and these levels do not fall after the administration of aldosterone ($0.1\,mg\,kg^{-1}$) for a week.



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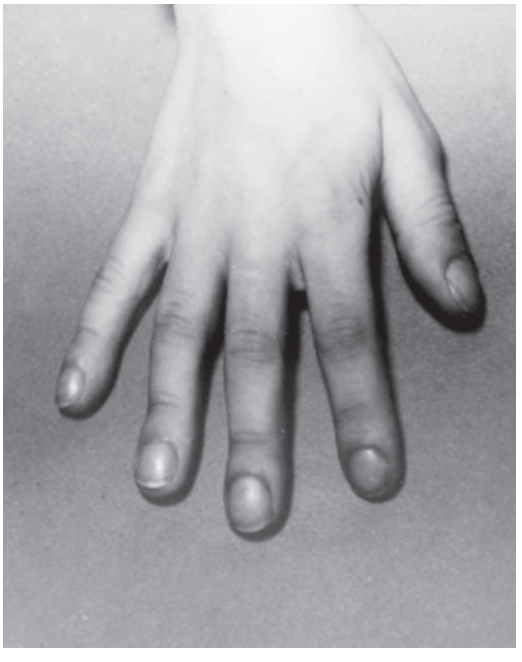


(b)

Figure C1.4 (a) Hyperinflated rib cage with rib recession in an undernourished patient. (b) Clubbing of cyanosed fingers.

Case 18 | Obesity/Pickwickian syndrome

Frequency in survey: main focus of a short case or additional feature in 0.5% of attempts at PACES Station 1, Respiratory.

Survey note: most cases revolved around features of the Pickwickian syndrome, though there was one case with an apronectomy scar and small testes in which Klinefelter's was suggested.

Record

The patient is *massively obese* and *cyanosed*. He has (may have) rapid and shallow breathing (or hypoventilating), his *venous pressure is elevated* and there is *ankle oedema.*

These features suggest *cor pulmonale* secondary to the extreme obesity – the Pickwickian syndrome.*

Respiratory problems associated with obesity

Severe obesity leads to increased demand for ventilation, increased breathing workload, respiratory muscle inefficiency, decreased functional reserve capacity and expiratory reserve volume. There is alveolar hypoventilation and reduced ventilatory sensitivity to CO_2. Peripheral lung units can close, resulting in a ventilation–perfusion mismatch. The overall result is chronic hypoxaemia with cyanosis and hypercapnia; the end-stage is the *Pickwickian syndrome* in which nocturnal obstructive apnoea† and hypoventilation are so marked that the patient can only have undisturbed sleep when upright (more usually sitting than standing as in the original description!*), often in the daytime.

Pulmonary hypertension occurs, there are usually morning headaches and impotence and there may be polycythaemia. Eventually cardiac failure supervenes.

Sleep apnoea is very common in the severely obese. The most obese are not necessarily the most severely affected. It may be obstructive or central.† Daytime somnolence is common and is partly due to the hypoxia and partly from the continual disturbance of sleep at night – the patient tends to wake after each episode of sleep apnoea (cessation of breathing for 10 sec or longer).

Body Mass Index (BMI) = weight (kg)/height $(m)^2$. As a rule of thumb, health risks increase as BMI increases above 25; however, BMI normally increases with age and one study found that the BMI associated with the lowest mortality was approximately: 19.5 at age 20, 21 at age 30, 22.5 at age 40, 24.5 at age 50, nearly 26 at age 60 and 27.5 at age 70.

*The term is derived from the character in Charles Dickens' *Pickwick Papers* and was first applied by Osler. The character, Joe, kept beating the door even after hearing a response from inside the room, because if he stopped, he would fall asleep standing on his feet! (See *American Journal of Medicine* 1956, **21**: 811–18 to read Dickens' wonderful description.)

†*Obstructive sleep apnoea* – the upper pharyngeal cavity collapses due to the negative intrathoracic pressure and the process is aided by a short neck and large accumulations of fat often in combination with micrognathia and enlarged tonsils. There are vigorous thoracoabdominal movements but no air entry into the lungs. The obstruction leads to hypoventilation and hypoxia which somehow trigger apnoeic episodes, making the hypoxia and hypercapnia worse. Weight loss and sometimes surgical removal of the obstructive tissues may help. *Central sleep apnoea* – cessation of ventilatory drive from the brain centres so that diaphragmatic excursions stop for periods of 10–30 sec. There are no thoracoabdominal movements and there is no activity. It is not known why the obese are prone to this.

Body fat can be estimated by measuring skinfold thickness with callipers at the biceps, triceps, subscapular and suprailiac regions.

Adipocytes increase in size and then number as necessary to accommodate excess nutrient calories; but once formed, though they can decrease in size with weight loss, their total number does not decrease (the 'ratchet effect'). *Lipoprotein lipase* (LPL) generates free fatty acids (FFA) from circulating chylomicrons and VLDL, and the FFA can then enter adipocytes. LPL activity is high in obese people and rises with initial weight loss and this may be a factor in the accelerated weight regain of many patients. Maintained weight loss, however, is associated with a decrease in LPL activity. Fat cells from the upper body are probably different in responsiveness to testosterone and oestrogens than lower body fat cells leading to:

Android fatness: fat distributed in upper body above the waist,

Gynaecoid fatness: fat predominantly in lower body – lower abdomen, buttocks, hips, thighs.

Android fatness carries a greater risk for hypertension, cardiovascular disease, hyperinsulinaemia, diabetes, gallbladder disease, stroke and a higher mortality than does gynaecoid fatness. A waist:hip (circumference) ratio greater than 0.85 for women and 1.0 for men is abnormal.

Other clinical manifestations of obesity

Insulin resistance (enlarged adipocytes less sensitive to the antilipolytic and lipogenic actions of insulin; decreased number of insulin receptors as well as post-receptor defects; liver and muscle also less sensitive to insulin; basal and stimulated hyperinsulinaemia results*)

Diabetes mellitus (type 2 diabetes approximately three times higher in the overweight; 85% of type 2 patients in the USA are obese; though the development of type 2 diabetes requires the appropriate genetic legacy, obesity by enhancing insulin resistance tends to unmask and exacerbate the underlying propensity)

Hypertension* (prevalence three times higher in the obese; mechanism is uncertain – hyperinsulinaemia* leading to increased tubular reabsorption of sodium may be a factor; weight loss by dieting lowers blood pressure even without dietary salt restriction)

Cardiovascular disease (in obesity increased blood volume, stroke volume, left ventricular end-diastolic volume and filling pressure result in high cardiac output; this leads to left ventricular hypertrophy and dilation, the former being exacerbated by hypertension; the result is greater risk of congestive heart failure and sudden death)

Lipid abnormalities (obesity is associated with low HDL cholesterol;* LDL may be elevated; hypertriglyceridaemia* is more prevalent, possibly because the insulin resistance and hyperinsulinaemia* cause increased hepatic production of triglycerides; the hypertriglyceridaemia tends to improve with weight loss; if a true genetic lipoprotein disorder coexists, more intensive therapy may be required)

Venous circulatory disease (severe obesity is often associated with varicose veins and venous stasis; congestive cardiac failure adds to the dependent oedema; increased propensity for thrombophlebitis and thromboembolism)

Cancer (obese women have a higher incidence of endometrial cancer, postmenopausal breast cancer, and cancer of the gallbladder and the biliary system; obese men have a higher mortality from cancer of the colon, rectum and prostate, for unknown reasons)

Gastrointestinal disease (cholesterol gallstones leading to cholecystitis; obesity may be associated with fatty liver with modest abnormalities of liver function tests)

Arthritis (osteoarthritis due to excess stress placed on the joints of the lower extremities and back; multifactorial elevation in uric acid levels in the obese)

Skin (intertrigo in redundant folds of skin; fungal and yeast infections; *acanthosis nigricans*, which should always be looked for in obese patients, may be associated with severe insulin resistance; see Vol. 3, Station 5, Skin, Case 48)

Increased mortality (obesity itself may make an independent contribution to mortality, though the effect generally occurs through linkage with factors such as hypertension, diabetes and hyperlipidaemia.*

*NB: the metabolic syndrome. This refers to the clustering of insulin insensitivity, hyperinsulinaemia, varying degrees of glucose intolerance, hypertension, increased triglycerides and decreased HDL, a clustering which may predispose to vascular disease, in particular coronary artery disease. It is postulated that insulin insensitivity is the underlying factor.

Endocrine causes of obesity

(<1% of obese patients)

Hypothyroidism (thickened and coarse facial features, dry skin, non-pitting swelling of subcutaneous tissues, hoarse voice, thinning hair, slow pulse, slow relaxing ankle jerks; see Vol. 3, Station 5, Endocrine, Case 5)

Polycystic ovarian syndrome (hirsutism, oligo- or amenorrhoea, excess androgen production of ovarian origin, ultrasound scan may show polycystic ovaries; patients with polycystic ovaries are often obese and insulin resistant)

Hypothalamic disease (damage to hypothalamic appetite systems and tracts by surgery, trauma, inflammation, craniopharyngioma or other tumours may lead to hyperphagic obesity)

Cushing's (truncal obesity, moon face, purple striae, proximal muscle weakness; see Vol. 3, Station 5, Endocrine, Case 6).

Rare genetic diseases associated with obesity include

Prader–Willi syndrome (obesity may be massive, almond-shaped eyes, acromicria, mental retardation, diabetes, hypogonadism; see Vol. 3, Station 5, Endocrine, Case 12)

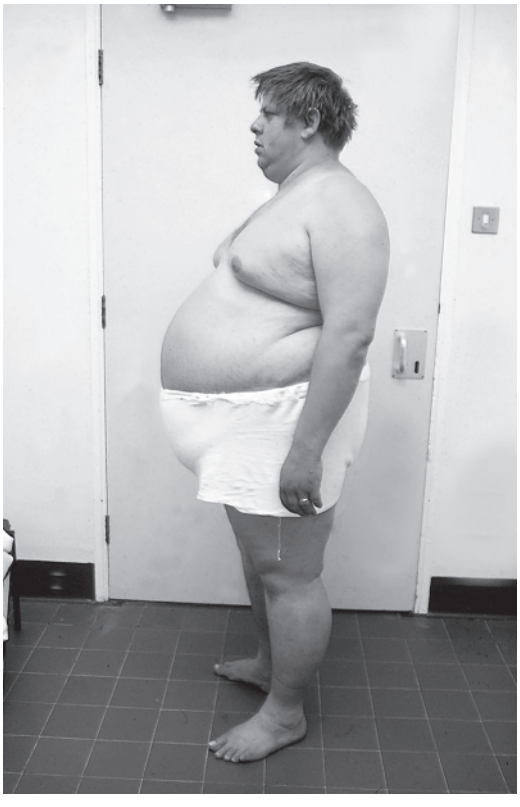
Laurence–Moon–Bardet–Biedl syndrome (?retinitis pigmentosa, hypogonadism, dwarfism, mental retardation, polydactyly; see Vol. 3, Station 5, Eyes, Case 19)

Alström syndrome (see Vol. 3, Station 5, Eyes, Case 19)

Cohen's syndrome (microcephaly, mental retardation, short stature, facial abnormalities and obesity)

Carpenter's syndrome (see Vol. 3, Station 5, Eyes, Case 19)

Blount's disease (bowed legs, tibial torsion, obesity).



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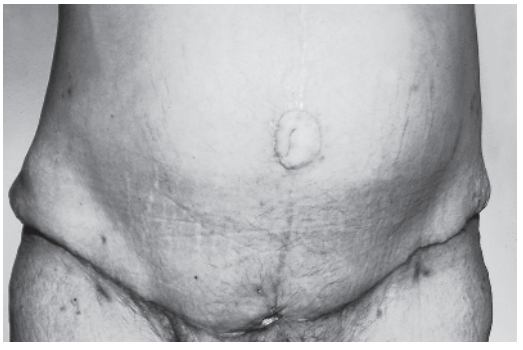


(b)

Figure C1.5 (a) Pickwickian syndrome. (b) Apronectomy scar.

Case 19 | Pneumothorax

Frequency in survey: main focus of a short case or additional feature in 0.5% of attempts at PACES Station 1, Respiratory.

Record

The R/L *side* of the chest (of this tall, thin, young adult male – old patients are usually bronchitic) *expands poorly* compared with the other side. Though the *percussion note* on the R/L side is *hyperresonant*, the tactile fremitus, vocal resonance and *breath sounds* are all *diminished* (large pneumothorax of one side may push the *trachea* and apex beat to the opposite side).

These findings suggest a pneumothorax of the R/L side.

Male-to-female ratio is 6/1.

A 'crunching' sound in keeping with the heart beat may be heard when the pneumothorax is small.

Treatment is not required in a healthy individual with a small pneumothorax (i.e. if only a quarter of one side is affected). Drainage is indicated for:

- Larger pneumothorax associated with dyspnoea, increasing in size or not resolving after 1 week
- Tension pneumothorax
- Pneumothorax complicating underlying severe chronic bronchitis with emphysema
- Pneumothorax exacerbating acute severe asthma (hence a chest X-ray is mandatory in acute severe asthma).

When drainage is indicated, simple aspiration (with a plastic cannula, syringe and three-way tap so that aspirated air can be removed) should usually be attempted before resorting to intercostal drainage via a tube attached to an underwater seal. Tension pneumothorax should be released urgently by stabbing an intravenous cannula through the chest wall at the second intercostal space, mid-clavicular line, pending the insertion of an intercostal drain.

Recurrent spontaneous pneumothorax is treated by obliteration of the pleural space (pleurectomy; insertion of irritating substances into the pleural cavity; scarification of the pleura followed by intrapleural suction).

Causes of pneumothorax

Traumatic

Penetrating chest wounds

Iatrogenic (chest aspiration, intercostal nerve block, subclavian cannulation, transbronchial biopsy, needle aspiration lung biopsy, positive pressure ventilation)

Chest compression injury (including external cardiac massage)

Spontaneous

Primary (a common cause in young men*)

Secondary

chronic obstructive pulmonary disease

asthma

congenital cysts and bullae

pleural malignancy

rheumatoid lung disease (see Station 1, Respiratory, Case 6)

bacterial pneumonia (see Station 1, Respiratory, Case 5)

tuberculosis

*The risk of a second pneumothorax in a young adult following the first episode is of the order of 25%. After a second episode, the risk increases to the order of 50%.

cystic fibrosis (see Station 1, Respiratory, Case 17)
tuberous sclerosis (see Vol. 3, Station 5, Skin, Case 9)
endometriosis of the pleura
Marfan's syndrome (see Station 3, Cardiovascular, Case 13)
sarcoidosis
histiocytosis X
whooping cough
oesophageal rupture
Pneumocystis carinii pneumonia

Case 20 | Cor pulmonale

Frequency in survey: main focus of a short case or additional feature in 0.3% of attempts at PACES Station 1, Respiratory.

Record

The patient's fingers are *nicotine-stained* and there is *central cyanosis*. There is (may be) *finger clubbing* (if associated with pulmonary fibrosis). The pulse is regular, the *venous pressure is raised* (give height) with prominent small *a* waves and giant *v* waves (if there is secondary tricuspid incompetence), and there is *ankle* and *sacral oedema*. *Expiration is prolonged and noisy.* The *accessory muscles* of respiration are in use at rest, and there is a *tracheal tug.* The trachea is central, expansion is equal, the percussion note is resonant, and tactile fremitus and vocal resonance are normal. There is a *left parasternal heave* and a palpable second heart sound* (?pansystolic murmur of tricuspid incompetence (rare)). The heart sounds are often difficult to hear due to hyperexpansion of the lungs. There are (may be) widespread *expiratory rhonchi* and coarse inspiratory crepitations and the forced expiratory time (see Section B, Examination *Routine* 3) is 8 sec. (There is no *flapping tremor* of the hands – if there were, you would want to examine the fundi for papilloedema.)

These findings suggest cor pulmonale due to chronic bronchitis and emphysema. (Right heart failure is often precipitated by acute infection.)

The auscultatory cardiac signs of pulmonary hypertension, some of which may be audible,* are:

Loud pulmonary second sound
Pulmonary early systolic ejection click
Right ventricular fourth heart sound
Pansystolic murmur of functional tricuspid incompetence (giant *v* waves)
Early diastolic murmur of functional pulmonary incompetence (Graham Steell murmur).

Causes of pulmonary hypertension

COPD (with or without emphysema; by far the most common cause; see Station 1, Respiratory, Case 3)

Recurrent pulmonary emboli (signs of pulmonary hypertension without clinical evidence of other lung disease; ?DVT)

Primary pulmonary hypertension (signs of pulmonary hypertension without clinical evidence of other lung disease; usually a female)

Non-pulmonary causes of alveolar hypoventilation (kyphoscoliosis, obesity (see Station 1, Respiratory, Case 18), neuromuscular weakness)

Lung diseases which only occasionally result in cor pulmonale include:

Progressive massive fibrosis (?coal dust tattoos on the skin; chronic bronchitis is the most common cause of cor pulmonale in miners)

Bronchiectasis (especially cystic fibrosis; ?clubbing, cyanosis, full sputum pot, productive cough, crepitations; see Station 1, Respiratory, Case 4)

Cryptogenic fibrosing alveolitis (?clubbing, cyanosis, basal crackles; see Station 1, Respiratory, Case 1)

Systemic sclerosis (hands, facies; see Vol. 3, Station 5, Locomotor, Case 3)

Sarcoidosis (?lupus pernio; see Vol. 3, Station 5, Skin, Case 19)

Asthma (severe and chronic; may be missed if the reversibility is not checked in chronic small airways obstruction).

*These findings, which may be prominent in cor pulmonale due to other causes, may be difficult to elicit in cor pulmonale where a barrel-shaped chest and hyperinflation are present, and the heart is enfolded by overinflated lungs.

Case 21 | Collapsed lung/atelectasis

Frequency in survey: main focus of a short case or additional feature in 0.2% of attempts at PACES Station 1, Respiratory.

Survey note: see Vol. 2, Section F, Anecdote 93.

Record 1

There is no clubbing. On inspection of the chest from the front and the back, there is a *decrease* in *right-sided chest expansion*. This is confirmed on *palpation for chest expansion* and there is *displacement of cardiac apex* to the right. The percussion note is *dull* below the scapula. Tactile vocal fremitus is unreliable except in large pleural effusions; hence I have replaced it with auscultation for *vocal resonance*,* which is *increased* below the scapula on the right where *bronchial breath sounds* are also present.

This all suggests a diagnosis of right†-sided lower lobe lung collapse, most likely to be due to focal chronic lung pathology such as postpneumonic scarring. (Check the *sputum pot* is empty as copious sputum production is compatible with chronic infection of a collapsed pulmonary lobe). I would like to investigate it further to rule out a proximal obstructive lesion of the large airways, most importantly a neoplasm, with a *chest X-ray* in the first instance proceeding, if required, to a *CT scan* of the thorax and/or a *bronchoscopy*.

Record 2

There is no clubbing. On inspection of the chest from the front and the back, there is a decrease in *right-sided chest expansion*. This is confirmed on *palpation for chest expansion* and there is *tracheal deviation* to the right. The percussion note is *dull* over the anterior upper chest on the right. Tactile vocal fremitus is unreliable except in large pleural effusions; hence I have replaced it with auscultation for *vocal resonance*,* which is *increased* over the anterior upper chest on the right; *bronchial breath sounds* are also present.

This all suggests a diagnosis of right†-sided upper lobe lung collapse, most likely to be due to focal chronic lung pathology such as postpneumonic scarring. (Check the *sputum pot* is empty as copious sputum production is compatible with chronic infection of a collapsed pulmonary lobe). I would like to investigate it further to rule out a proximal obstructive lesion of the large airways, most importantly a neoplasm, with a *chest X-ray* in the first instance proceeding, if required, to a *CT scan* of the thorax and/or a *bronchoscopy*.

*__A note on the examination for vocal resonance:__ Early German physicians (18th century: pre-stethoscope era) asked patients to say *neun-und-neunzig* to evoke fremitus over the thorax, the English translation of which is *ninety-nine*. However, it is recommended that patients use the sound 'oy' (as in 'boy') because normal lung is believed to better transmit low-pitched vibrations than '99' when auscultating for vocal resonance. This improves the detection of not only increased resonance but also distortion of the transmitted sound to a bleating nature ('aegophony') in the presence of consolidation, which is often associated with collapsed lung. Eliciting vocal resonance is of paramount importance when trying to distinguish between the causes of dullness to percussion: a reduced vocal resonance suggesting pleural effusion and an increased vocal resonance suggesting collapse/consolidation.

†You should be able to work out from this what the equivalent signs would be on the left.

Causes of collapsed lung

Atelectasis is a collapse of lung tissue affecting part or all of one lung. In clinical practice the acute and subacute causes are common but less likely to be presented in an examination setting.

Acute:

1 Mucous plugging, particularly in asthma patients with sudden deterioration
2 Inhaled foreign body (mainly in children)
3 Chest wall injury/rib fractures causing atelectasis due to locally reduced movement

Subacute:

1 Proximal obstructing lesion of the large airways (e.g. neoplasm)
2 Postoperative patients/prolonged bed rest (much more common in patients with chronic lung disease who undergo specific risk assessment for postoperative atelectasis as part of their anaesthetic assessment for thoracoabdominal surgery in many centres)

Chronic:

1 Postpneumonic focal fibrosis
2 Bronchiectasis (usually associated with recurrent infection/excessive secretions)
3 Chronic aspiration (usually associated with recurrent infection/excessive secretions)
4 Diaphragmatic palsy
5 Pulmonary fibrosis
6 Trapped lung due to pleural thickening

Case 22 | Superior vena cava obstruction

Frequency in survey: main focus of a short case or additional feature in 0.1% of attempts at PACES Station 1, Respiratory.

Record

There is (may be) stridor. The face and upper extremities are *oedematous* (puffy) and *cyanosed*, and the eyes are *suffused*. The *superficial veins* over these areas are *dilated* and there is *fixed engorgement* of the *neck veins.* The undersurface of the tongue is covered with multiple venous angiomata. There is (may be) a radiation burn on the chest wall.

The diagnosis is superior vena cava obstruction, most likely due to carcinoma* of the bronchus, particularly small cell carcinoma (?lymph nodes, chest signs, clubbing, etc; see Station 1, Respiratory, Case 13). It has been treated by radiotherapy.†

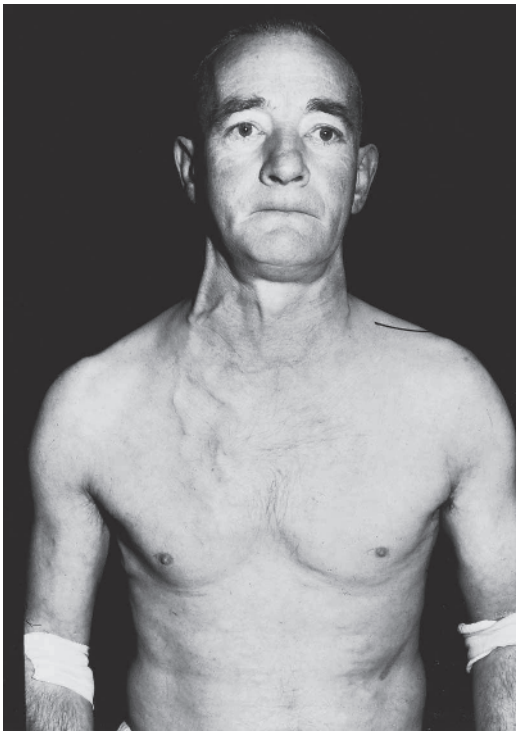
The patient may complain of headaches (may be severe on coughing), difficulty in breathing, dysphagia, dizziness or blackouts. Physical signs are frequently absent or minimal.

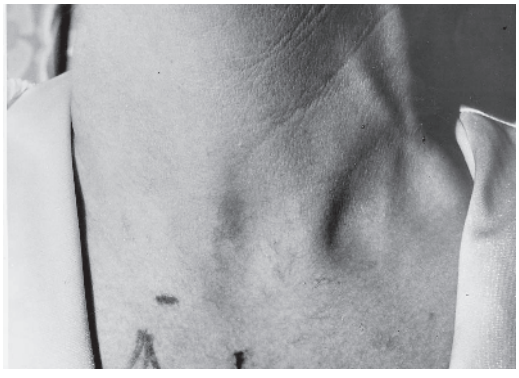
Other causes of superior vena cava obstruction

Lymphoma
Aortic aneurysm
Mediastinal fibrosis
Mediastinal goitre

*The compression may either be by the tumour or by involved lymph nodes.
†Radiotherapy, or chemotherapy, is required urgently in this condition. A stent can sometimes be placed in the superior vena cava as a palliative procedure. Dexamethasone is also used.

(a)





(b)

Figure C1.6 (a,b) Superior vena cava obstruction. Note the radiotherapy ink marks in (b).

Case 23 | Tuberculosis/apical consolidation

Frequency in survey: main focus of a short case or additional feature in 0.1% of attempts at PACES Station 1, Respiratory.

Record

The trachea in this Asian patient is central (may be deviated*) and the expansion is normal (may be reduced at the apex*). The percussion note is *dull* at the R/L apex with *diminished tactile fremitus*. There is *bronchial breathing* with *inspiratory crackles* over the area of dullness.

The diagnosis is R/L apical consolidation, with tuberculosis being a serious contender as the underlying cause.†

Principal varieties of tuberculosis

Primary pulmonary tuberculosis. The first infection with the tubercle bacillus (primary TB) usually includes involvement of the draining lymph node (the Ghon focus). All other TB lesions are regarded as post primary and are not accompanied by major involvement of the draining lymph nodes (in Europeans), though sometimes in immigrants gross enlargement of the lymph nodes may be seen. *Erythema nodosum*, *phlyctenular conjunctivitis* and *pleural effusion* may accompany primary pulmonary tuberculosis.

Miliary tuberculosis. Acute dissemination of tubercle bacilli via the bloodstream may occur if the initial infection is an overwhelming one or the patient's defences are poor due to malnutrition, corticosteroid or immunosuppressive drug therapy, HIV or intercurrent disease. This condition should be borne in mind in at-risk groups (see below).

Tuberculous meningitis. May occur at any age but is particularly common in small children as a complication of the primary infection.

Postprimary pulmonary tuberculosis. This form may arise as direct progression of a primary lesion, reactivation of an old lesion, haematogenous spread or from contact with a patient with *open* (sputum-positive) TB. The predisposing factors for reactivation are malnutrition, poor and overcrowded housing conditions, silicosis and other occupational diseases, alcoholism and cigarette smoking, immunosuppressive drugs, and diseases associated with impaired cellular immunity (e.g. Hodgkin's disease, leukaemia, lymphoma, AIDS). Complications of postprimary pulmonary TB include *empyema*, *laryngitis*, *aspergillomata* (colonization of a cavity), *amyloidosis*, *TB of the organs* and *adult respiratory syndrome.*

Bone and joint tuberculosis. There is a high rate in immigrants of Asian origin. Usually of haematogenous origin. The most common site for skeletal TB is the spine followed by the weight-bearing joints. AFB may be obtained from synovial fluid or bone but diagnostic exploration may have to be undertaken. Patients with TB can also have a reactive arthritis known as *Poncet's disease*; this usually settles with control of the TB.

Urinary tract tuberculosis. Results from haematogenous spread to the kidney with subsequent spread

*In the examination setting there are usually elicitable signs, even though in clinical practice one often encounters patients with pulmonary TB, sometimes with excessive radiological changes, who have no physical signs. The patient may have signs of fibrosis (e.g. deviated trachea), as seen in advanced cases, but the candidate should consider the diagnosis of pulmonary TB when there is only a dull percussion note, or a few crepitations at the apex of the lung.

†The differential diagnosis should include *carcinoma of the bronchus*, atypical pneumonia especially due to *Klebsiella pneumoniae*, pulmonary infarction and fungal infection.

down the ureteric tract. The patient may present with dysuria, nocturia, loin pain or may have painless haematuria, though many patients with positive urine cultures are asymptomatic. A history of recurrent urinary tract infection or *pyuria with negative bacterial cultures* should be regarded with suspicion for urinary tract TB.

Genital tuberculosis. A large majority of patients have evidence of tuberculosis at extragenital sites. Females present with infertility, pelvic inflammatory disease or amenorrhoea. Adnexal masses are palpable on pelvic examination in about half the cases.

Tuberculous peritonitis. Usually haematogenous. Often associated with weight loss, abdominal pain and gross ascites. Diagnosis can be made at laparoscopy when the peritoneum studded with whitish granulomata can be seen. Peritoneal fluid is rarely positive for AFB by stained smear and even by culture is positive in somewhat less than 50% of cases. May occur in the alcoholic with cirrhosis (see Footnote, Station 1, Abdominal, Case 7).

Tuberculous lymphadenitis. Mostly seen in patients of Afro-Asian origin. The patient may present with painless swelling of cervical lymph glands, or sometimes with pyrexia and lymphadenopathy. Untreated swelling may form a 'cold' abscess or sinus.

Cutaneous tuberculosis. This may present in one of many ways including a *primary complex* (an ulcerating papule on the face), *miliary* TB (particularly in immunocompromised children), *verrucous* TB (warty lesions as an occupational hazard in patients working with infected material), *scrofuloderma* (breakdown of skin over a tuberculous focus) and *lupus vulgaris* (see Vol. 3, Station 5, Skin, Case 38).

At-risk groups

Contacts – should be screened by tuberculin testing and chest X-ray

Immigrants from the Asian subcontinent have a high notification rate

Inhabitants of some institutions – prisons, lodging houses, hostel dwellers and mental institutions

Nursing homes – outbreaks of TB among the elderly in nursing homes have been reported

Medical laboratory workers – the incidence is high among staff in hospital pathology departments

Other groups – doctors, dentists, hospital employees, schoolteachers and those carers who work with children are potentially exposed to the risks for contracting TB

Treatment (see also Station 1, Respiratory, Case 7)

Most patients can be treated at home. Patients are advised to avoid making new contacts for 2 weeks. Treatment regimens should last for 6 months except in those who have tuberculous meningitis; they should be treated for 12 months.

Drug therapy should be given as combination tablets to aid compliance. The initial phase of 2 months should include four drugs (see Station 1, Respiratory, Case 7). Treatment should be continued for 4 more months with rifampicin and isoniazid. Regular checks by nurses and health visitors are necessary to ensure compliance. The rifampicin in the combination tablets produces a pink/orange discoloration of the urine which will aid these checks.

Case 24 | Normal chest

Frequency in survey: has still not occurred in our surveys of PACES Station 1, Respiratory.

The College have made it clear that 'normal' is an option in PACES. To have no findings on clinical examination is common in real clinical medicine and so this must be a possibility in the exam. In terms of the practical reality of the exam, in order for PACES to proceed there must be a chest case in Station 1, Respiratory. If, at the last minute, neither of the scheduled chest cases turns up on the day, or if in the middle of a carousel the only one who did turn up decides not to continue or is too ill to continue, a substitute case has to be found at short notice. In this situation, one option is to proceed with a patient with a chest which is normal and make up an appropriate scenario. One simply has to imagine oneself as the invigilating registrar to think what that might be. One would first look amongst any surplus cases in the other stations for a patient with something relating to the chest. Failing that, one might look for someone who is a smoker or a member of the nursing, portering or other support staff who smokes and come up with a scenario such as:

> 'You have been asked to see this . . . -year-old smoker for insurance purposes. Please examine the chest . . .'

There may be a clue in the case scenario and the fact that the scenario has been hurriedly hand-scribbled. As it turns out, we have never yet heard through our surveys of this happening in the case of PACES Station 1, Respiratory. This is likely to be because at any one time, there are so many chest cases in the hospital that the case found at short notice is more likely to be one with COPD. Nevertheless, it could happen if time were short.

From our surveys, it is clear that the most common reason for finding no abnormality is missing the physical signs that are present (see probably both Anecdotes 1 and 2, Station 3, Cardiovascular, Case 24; possibly Anecdote 5, Station 1, Abdominal, Case 11; and Vol. 2, Section F, Experience 158). Other reasons for cases of 'normal' will be either because the physical signs are no longer present by the time the patient comes to the examination (see Anecdote 1, Station 3, CNS, Case 28) or that the examiners and candidate disagree with the selectors of the cases about the presence of physical signs (this may have happened in Anecdote, Vol. 3, Station 5, Eyes, Case 21; see also Vol. 2, Section F, Experience 198 and Anecdote 303).