Respiratory symptoms and signs

Suveer Singh

Abstract

The year 2016 marks the 200th anniversary of Laënnec's invention of the stethoscope, with the subsequent publication of auscultatory sounds for clinical diagnosis in 1819. Today, history and examination remain pivotal to accurate diagnosis. The hypotheticodeductive method of diagnosis based on Bayes' theorem requires a detailed history and examination skills to elicit symptoms and signs. The key symptoms of respiratory disease are breathlessness, chest pain, wheeze, cough and associated sputum production. Non-respiratory conditions may also produce such symptoms. A systematic approach to history-taking should include all primary symptoms, their time-course, characteristics, severity and trajectory. A review of non-respiratory associations, pharmaceutical and historical aspects of respiratory symptoms should precede a thorough review of clinical signs. Further questioning or examination will lead to assimilation of information, synthesis with clinicopathophysiological knowledge of respiratory diseases, and formulation of a differential diagnosis. On examination, based on the model of inspection, palpation, percussion and auscultation, there are a few classical patterns of the most important focal abnormalities, although there may be an absence of clinical signs. This article reviews the key features of respiratory symptoms and signs, outlines tips on how best to elicit these, and discusses patterns of clinical features suggesting certain diagnoses.

Keywords Bayes' theorem; breathlessness; chest pain; cough; dyspnoea; haemoptysis; signs; stethoscope; symptoms; wheeze

Key points

- The hypotheticodeductive method of diagnosis (Bayes' theorem) requires a detailed history and examination skills to elicit symptoms and signs
- History-taking should include all primary symptoms, with their time-course, characteristics, severity and trajectory
- A review of non-respiratory associations, pharmaceutical and historical aspects of respiratory symptoms precedes a thorough review of clinical signs
- The 200th anniversary of Laënnec's invention of the stethoscope is marked in 2016
- Publication of auscultatory sounds related to clinicopathological diagnosis in *De l'Auscultation médiate* followed in 1819

Introduction

The aim of the history and examination is an accurate clinical diagnosis. Of a number of methods in use, such as pattern recognition, the hypotheticodeductive method based on Bayes' theorem is favoured.[1] Diagnostic hypotheses are considered. Clinical data based upon history, examination and tests are evaluated. Either these strengthen the hypothesis, at the expense of more implausible ones, or new hypotheses are entertained. A sound theoretical knowledge of disease states is implicit. The process of sequential revision leads to a likelihood of diagnosis either strong enough to lead to action, or low enough to abandon consideration of the disease, hence the importance of effective eliciting of symptoms and signs.

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Symptoms

The main symptoms of respiratory disorders are breathlessness (dyspnoea), chest pain, wheeze and cough, which may be productive of sputum. Non-respiratory conditions may produce such symptoms (e.g. gastrointestinal or cardiac causes of cough, breathlessness caused by anaemia, hypothyroidism, metabolic acidosis, myopathies). The lungs can also produce non-respiratory symptoms such as paraneoplastic symptoms of lung malignancy. To determine the origin and significance of the symptoms, an understanding of their anatomical and mechanistic basis is important.

A further history with *pulmonary risk factors* and potential associations of the primary symptoms are crucial to synthesize into a differential diagnosis. Thus, the following lines of questioning should be considered: childhood illness (e.g. congenital or neonatal, whooping cough, wheeze or asthma, allergies) and immunizations (or omissions, i.e. BCG); occupational (i.e. organic or inorganic exposures that may cause hypersensitivity, pneumoconiosis); environmental (i.e. airborne or waterborne pollution) and work-based exacerbations; travel (e.g. tuberculosis, tropical diseases, tick-borne parasites, hospitalization while overseas); medication (i.e. potentially pneumotoxic agents including recreational drugs, chemotherapy, newer monoclonal antibody-based biological agents, radiation therapy); smoking (e.g. tobacco, cannabis, passive smoking); nasal symptoms (i.e. postnasal drip, rhinorrhoea, blockage) and previous surgery; rheumatological or connective tissue disorders; dermatological conditions (e.g. eczema, erythemas, dermatomyositis); sleep-disordered breathing (e.g. daytime hypersomnolence, snoring, choking, witnessed apnoeas, symptoms of hypercapnia); HIV and risk factors; family history (i.e. asthma, atopy, chronic obstructive pulmonary disease (COPD), malignancy, pneumonias); and psychosocial history.

Breathlessness

Degree of breathlessness (dyspnoea) is used to characterize the subjective experience of breathing discomfort. It manifests as qualitatively distinct sensations. The experience derives from

interactions between multiple physiological, psychological, social and environmental factors, and may induce secondary physiological and behavioural responses.[2]

The main causes of unexplained dyspnoea are asthma, COPD, interstitial lung disease, myocardial dysfunction and deconditioning/obesity/anaemia.

Patients describe difficult, painful, laboured or inadequate breathing. The terms 'air hunger', chest tightness, choking and suffocation apply.

Certain patterns of verbal descriptors may favour particular physiological processes. Thus, acute hypercapnia or restricted thoracic movement produces a sensation of 'air hunger' or 'inability to catch a full breath'. Acute bronchoconstriction has descriptors such as 'chest tightness', 'increased effort of breathing' and 'air hunger'.

Patients with COPD often complain of an 'inability to take a deep breath', 'increased effort' or 'unsatisfying breathing'. Heart failure sufferers will describe 'air hunger' or 'suffocation'. The breathlessness of cardiac deconditioning is 'heavy breathing'. These descriptors may help to distinguish which of more than one cardiopulmonary disorder is contributing to the breathlessness. Furthermore, dyspnoea may seem out of proportion to the underlying lung disease. For instance, chest tightness or inability to get a full breath on exertion may suggest suboptimal control of airflow obstruction in COPD, rather than concomitant left ventricular dysfunction. Conversely, exertional breathlessness with fatigue may favour the latter as the primary symptom driver. Breathlessness can be a frightening and psychologically demanding experience.[3]

The pattern of breathlessness is important. Symptoms may vary with time, position and exertion. Exercise tolerance should be documented in terms of everyday achievable tasks, distance or number of stairs managed, its change over time and its trajectory (gradual or step change).

Mechanisms of breathlessness: respiratory system breathlessness is governed by dysfunction of the *respiratory central controller* (respiratory centre nuclei in the brainstem and medulla), the *ventilatory pump* (diaphragm and respiratory muscles, phrenic and other efferent and afferent nerves, pleura, thoracic cage, airways) or the *gas exchanger* (i.e. the alveolar–interstitial–capillary unit by which hypoxaemia and hypercapnia will influence the sensation of breathlessness).

The development of respiratory dyspnoea is a complex phenomenon, which arises through stimulation of mechano- and chemoreceptors in the upper and lower airways, lung parenchyma, pleura, chest wall and thoracic blood vessels, as a result of an applied mechanical load or central overdrive. An imbalance between the effect and the received central response (afferent) is perceived as breathlessness.

The key elements are:

- A central drive to breath (i.e. urge to breath). This drive to breath involves sensory input from chemosensors (e.g. medulla, carotid and aortic bodies), mechanoreceptors (e.g. chest wall, lung, neuromuscular receptors) and higher cerebral cortical modulation (e.g. anxiety, personality).
- The 'sense of respiratory effort' (i.e. work of breathing) associated with ventilation. A servo feedback loop with higher central nervous system (CNS) inputs exists. Efferent motor signals to the respiratory muscles are accompanied by concomitant signals to the cortex, allowing conscious modulation.

A discrepancy between what is expected after a given efferent message (the urge) and the response to the message (the sense of respiratory effort) is perceived as breathlessness. The mismatch is sometimes termed 'efferent–reafferent dissociation' or 'neuromechanical dissociation'.

These mismatches may be caused by obstruction, restriction or respiratory muscle weakness. In addition, breathlessness may also occur when the urge/central drive for ventilation is greater than the actual need for adequate gas exchange for a given mechanical load, as in hyperventilation, acidosis or hyperpyrexia, or after extreme exercise.

Characteristics of breathlessness

Speed of onset – sudden dyspnoea without an obvious cause suggests pulmonary embolism (PE) or pneumothorax (Table 1). Acute asthma may have associated wheeze. Progressive breathlessness with fever, cough and purulent sputum is more in keeping with pneumonia. A new-onset arrhythmia may present as breathlessness, even without palpitations.

Duration – this suggests the rate of disease progression, for which exercise tolerance is a good descriptor.

Timing – paroxysmal nocturnal dyspnoea implies waking from sleep and may identify left ventricular failure (LVF) and also severe COPD. Early morning waking that is recurrent and associated with wheeze or cough is typical of asthma.

Causes of breathlessness classified by speed of onset

Instantaneous
- Pulmonary embolism
- Pneumothorax

Acute (minutes to hours)
- Airways disease (e.g. asthma)
- Pulmonary embolism
- Parenchymal disease (e.g. pneumonia)
- Heart disease (e.g. LVF, MI)
- Hyperventilation syndrome
- Metabolic acidosis

Gradual (days)

Many of the above and:
- Lobar collapse (e.g. lung cancer)
- Pleural effusion
- SVC obstruction

Chronic (months to years)

Some of the above and:
- COPD
- Diffuse parenchymal disease (e.g. UIP)
- Bronchiectasis
- PVD (e.g. chronic thromboembolism, PHT)
- Hypoventilation (e.g. chest wall deformity)
- General (e.g. anaemia, thyrotoxicosis)

COPD, chronic obstructive pulmonary disease; LVF, left ventricular failure; MI, myocardial infarction; PHT, pulmonary hypertension; PVD, pulmonary vascular disease; SVC, superior vena cava; UIP, usual interstitial pneumonitis.

Table 1

Position – orthopnoea is the onset of breathlessness when the patient is supine and can indicate severe chronic lung disease or LVF. When sudden, it is characteristic of rare bilateral diaphragmatic paralysis (or immersion in water above the diaphragm). Platypnoea is breathlessness when upright that is relieved when supine. Platypnoea–orthodeoxia is the associated supine-related improvement in haemoglobin oxygen concentration. It may be noted when abdominal breathing is impaired (i.e. after major abdominal surgery), in severe COPD or in hepatopulmonary syndrome and other conditions associated with intracardiac or intrapulmonary right-to-left shunting.

Severity – breathlessness is effort dependent. Dyspnoea can be quantified, using validated breathlessness scales (e.g. Borg scale, Medical Research Council scale), questionnaires (e.g. St George's Respiratory Questionnaire) and exercise tests (e.g. Six-Minute Walk Test (6MWT), shuttle walk).

Chest pain

Chest pain may arise from a number of structures in the thorax. It can lead to hypoventilation, atelectasis and retention of secretions, and is often associated with breathlessness. Pleuritic pain varies with the respiratory cycle and can be made worse by deep inspiration, coughing and movement. A number of causes should be considered, including non-respiratory thoracic causes (i.e. originating from the myocardium, pericardium or oesophagogastrium), particularly as acute chest pain is synonymous with myocardial injury (Table 2).

Mechanisms of chest pain: pain arises from the musculoskeletal system, parietal (not visceral) pleura, major airways, diaphragm and mediastinum, but not parenchyma. Upper parietal pleural pathology presents as localized pain, whereas the lower parietal pleura and outer diaphragm cause referred pain in the abdomen, through lower intercostal nerve innervation. The phrenic nerve (C3/4/5) innervates the central diaphragm, with referred pain to the ipsilateral shoulder.

Localized constant chest pain (with or without tenderness) occurs with rib fractures, pleural infection (e.g. empyema), chest wall malignancy, costochondritis, benign asbestos pleural disease and connective tissue diseases (e.g. systemic lupus erythematosus (SLE)). 'Boring' constant pain is characteristic of malignant infiltration (e.g. mesothelioma, lung cancer).

Chest wall pain may be musculoskeletal, rheumatic, non-rheumatic, skin or sensory nerve related. Musculoskeletal causes include intercostal radiculitis (e.g. spinal osteoarthritis), previous surgical scars, costochondritis (i.e. Tietze's syndrome) or posterior chest syndrome as a result of vertebral costochondral joint dysfunction. Rheumatic involvement of the thoracic joints includes rheumatoid arthritis, ankylosing spondylitis and psoriatic arthritis. Non-rheumatic causes of chest wall pain are sickle cell crisis, stress fractures and infection (i.e. osteomyelitis). Chest wall pain is locally tender and made worse by movement. Neuropathic pains, including herpes zoster reactivation (shingles) and brachial plexus pain, are dermatomal, often excruciating and difficult to control. Visceral pain occurs in many cardiovascular, gastrointestinal (e.g. oesophagitis, cholecystitis) and psychological (e.g. hyperventilation syndrome) disorders. Tracheobronchitis is described as a raw substernal discomfort, exacerbated with the breathing cycle. Pericardial pain is often relieved on sitting forward, while myocardial pain is classically 'crushing' central pain, with associated symptoms.

Causes of pleuritic chest pain

- Pulmonary embolism/infarction
- Viral pleurisy
- Pneumothorax
- Pericarditis
- Collagen vascular disease (i.e. systemic lupus erythematosus, mixed connective tissue disease)
- Rheumatic diseases
- Inflammatory bowel disease
- Familial Mediterranean fever
- Radiation pneumonitis

Table 2

Cough

Cough is perhaps the most common outpatient symptom. It can be classified by its duration. It can be acute (<3 weeks), subacute (3–8 weeks) or chronic (>8 weeks). More women present with chronic cough, with some evidence of a heightened cough reflex sensitivity compared with men.

Mechanisms of cough: every cough occurs via a reflex arc, which is initiated by chemical and mechanical cough receptors in the epithelium of the respiratory tract, pericardium, oesophagus, diaphragm and stomach. Chemoreceptors are sensitive to acid, cold, heat, capsaicin-like compounds and other chemical irritants, while mechanoreceptors can be stimulated by touch and movement. The larynx and tracheobronchial tree have both receptor types. Vagal afferents to the medulla are modulated by higher CNS inputs, with efferents to the laryngopharyngeal and respiratory muscles, producing the cough. Expiratory intrapleural pressures (+40 kPa) and airflow rates (500 miles/hour) are generated. It can occur even without glottic closure. Diminished cough caused by central depression, pain, muscle weakness or laryngeal or respiratory disease can result in retained secretions, infection and their consequences (i.e. bronchiectasis if recurrent).

Characteristics of cough: acute cough is usually caused by an acute respiratory tract infection, although acute exacerbations of asthma and chronic pulmonary disease, pneumonia, PE and gastro-oesophageal reflux, especially during pregnancy, are other causes.

Subacute cough is frequently post-infectious, often persisting after resolution of other infective symptoms. More than 50% of cases resolve spontaneously.

The common causes of chronic cough, in up to 90% of cases, are upper airway cough syndrome (due to a nasal source, such as postnasal drip, rhinitis or rhinosinusitis), asthma-like syndromes (including bronchial hyperreactivity) and laryngopharyngeal reflux (non-acid reflux or gastro-oesophageal reflux). Questioning should be directed at these if a clear explanation is not forthcoming from the initial history. The causes of postnasal drip – a sensation of liquid sliding down the naso-oropharynx – include allergic and perennial non-allergic rhinitis, vasomotor rhinitis, acute nasopharyngitis and sinusitis. Upper airway secretions perpetuate cough.

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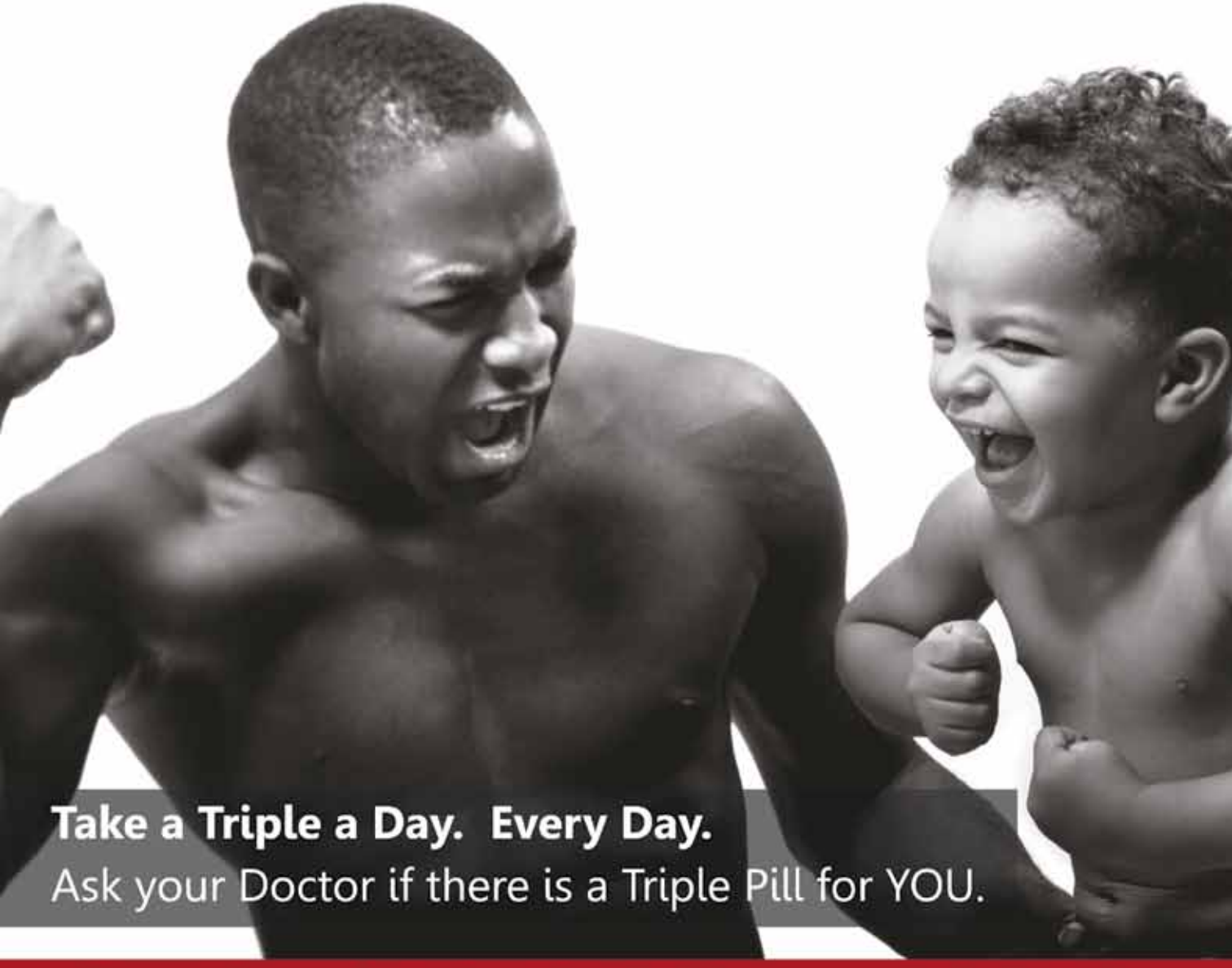

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Other important aetiologies in persistent cough include drug-induced cough, (i.e. angiotensin-converting enzyme (ACE) inhibitors, or β-adrenoreceptor blockers in airways disease).[4]

Less common causes of chronic cough include disorders of the airways, i.e. non-asthmatic eosinophilic bronchitis (diagnosed by cough, sputum eosinophilia without bronchial hyperreactivity), atypical infections (e.g. adult *Bordetella pertussis*), chronic bronchitis, bronchiectasis, neoplasm, foreign body (enquire for any provoking event), and parenchymal (interstitial lung disease, lung abscess) and pleural disease. Chronic idiopathic cough is associated with an exaggerated cough reflex sensitivity (Table 3).

Other features of cough: classic cough descriptors define certain aetiologies. Thus, a 'barking' cough refers to laryngeal disease, a 'brassy' cough to tracheobronchitis and a 'bovine' cough to vocal cord paralysis caused by laryngeal nerve palsy (e.g. bronchogenic carcinoma).

Paroxysms of cough with 'whoops' are characteristic of *B. pertussis* (whose adult prevalence in the USA and Europe has increased over the previous 5—10 years and is characterized by a catarrhal, a paroxysmal and an often prolonged recovery phase), while mealtime and reclining cough are indicative of laryngopharyngeal reflux (which can be non-acid-reflux).

Associated weight loss suggests tuberculosis, malignancy or chronic infection. Fever suggests infective or inflammatory causes. Recurrence of cough and infection raise the possibility of foreign body aspiration, bronchiectasis or an obstructing tumour.

Sputum

Over 100 ml/day of sputum is produced by the respiratory tract, to be absorbed or swallowed after removal by the mucociliary escalator. An excess is caused by inflammatory (e.g. infection) or allergic causes. Volume estimation in part defines chronic bronchitis and postnasal drip.

A change in character is instructive. Purulence — the presence of degenerate white cells in secretions — may be a sign of infection or inflammation (e.g. eosinophils in acute asthma). Blood-stained, 'rusty' sputum classically occurs in pneumococcal pneumonia, and 'red-currant jelly' sputum is characteristic of *Klebsiella pneumoniae*. Sputum plugs and bronchial casts may occur in asthma and allergic bronchopulmonary aspergillosis. Foul-smelling sputum indicates anaerobic infection in bronchiectasis, lung abscess or necrotizing pneumonia. Consider bronchiectasis if there is excessive purulent, sporadically blood-streaked sputum, whereas copious clear sputum is sometimes a feature of adenocarcinoma of the bronchioloalveolar type. Pink-tinged frothy sputum, caused by blood staining, occurs in pulmonary oedema.

Causes of cough

Cause of cough	Typical examples
Respiratory	
Acute infection	Viral, bronchopneumonia Tracheobronchitis
Chronic infection	Bronchiectasis, cystic fibrosis
Nasal, sinus disease	Postnasal drip, sinusitis
Airways disease	Asthma, COPD, LN compression
Parenchymal disease	Interstitial fibrosis, lung cancer
Irritant	Foreign body, allergy, smoke
Pleural disease	Pneumothorax, pleural effusion
Cardiovascular	LVF, mitral stenosis
Gastrointestinal	GORD, tracheobronchial fistula
Central nervous system	Recurrent aspiration e.g. MS, stroke
Drug induced	ACE inhibitors, inhaled drugs

ACE, angiotensin-converting enzyme; COPD, chronic obstructive pulmonary disease; GORD, gastro-oesophageal reflux disease; LN, lymph node; LVF, left ventricular failure; MS, multiple sclerosis.

Table 3

Wheeze and stridor

Wheeze: refers to the noisy musical sound produced by turbulent flow through narrow small airways. It occurs mainly during expiration and is characteristic of asthma, COPD, bronchiolitis and occasionally LVF. Associations are breathlessness or chest tightness caused by increased work of breathing and airway resistance, respectively. Bronchial smooth muscle contraction, oedema and excessive mucus production are pathophysiological correlates. Certain characteristics of the wheeze can help to identify its cause. Intermittent wheeze caused by allergens (e.g. pollen, house dust, moulds), exercise, drugs (e.g. aspirin, non-steroidal anti-inflammatories, β-adrenoreceptor blockers) or chemicals (e.g. di-isocyanates in paints), and associated with 'early morning waking', is suggestive of asthma. Reduced or absent wheeze or a 'silent' chest during a severe asthmatic attack indicates very poor airflow and is a medical emergency. Persistent wheeze, characteristic of COPD, is often associated with a smoker's 'productive' morning cough. Nocturnal wheeze or wheeze caused by drugs (e.g. β-adrenoreceptor blockers) may also be due to LVF (so-called 'cardiac asthma'). A fixed monophonic wheeze suggests local obstruction, which if new should raise concern of possible malignant airway involvement.

***Stridor*:** describes a coarse inspiratory wheeze that is caused by upper airway obstruction (laryngeal, tracheobronchial). Airways infection (e.g. epiglottitis, diphtheria), anaphylaxis, tumours of the upper airways (e.g. larynx), tracheal stenosis or extrinsic tracheal compression (e.g. goitre), aspirated foreign bodies (e.g. food), blood clots and sputum plugs can all obstruct the upper airways and cause stridor. Paroxysmal vocal cord dysfunction may have a psychosocial cause underlying the stridor.

Haemoptysis

Haemoptysis is perhaps the most alarming respiratory symptom that patients report. Most cases are infective (e.g. pneumonia, tuberculosis, bronchiectasis) or non-malignant (e.g. PE). A minority (10—20%) are caused by malignancy, and up to one-third are idiopathic.

Epistaxis and haematemesis are important non-respiratory causes to exclude. Rarer causes include lung abscess, mycetomas, arteriovenous malformations (i.e. hereditary haemorrhagic telangiectasia), pulmonary hypertension, vasculitides, aortic aneurysmal fistulae, pulmonary endometriosis and fungal (e.g. *Aspergillus*) or parasitic infections. Non-pulmonary causes include coagulopathies, anticoagulation, LVF and iatrogenic causes (e.g. lung biopsy).

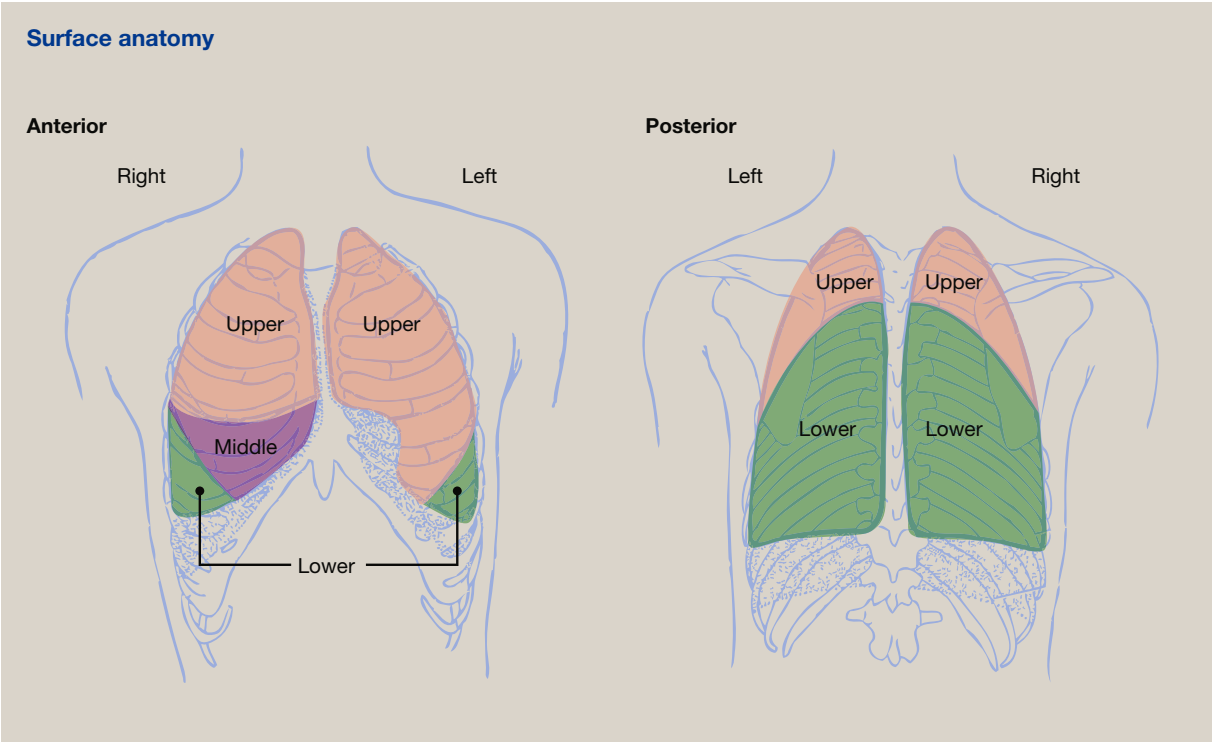

Figure 1

Most episodes of haemoptysis are less than massive. Massive haemoptysis (>0.5 litre/day of blood or any life-threatening haemoptysis) accounts for less than 20% of episodes but requires immediate attention. Bronchial tumours, bronchiectasis (including exacerbations of cystic fibrosis) and tuberculosis are potential causes, as is fibrocavitatory lung disease of any cause. Massive haemoptysis may lead to asphyxia. Initial assessment should determine the amount and character of the blood (e.g. fresh, old), time-course (e.g. intermittent, constant), associated symptoms (e.g. fever, pleurisy) and past history, with the potential aetiology, and an emergency management plan in mind for massive haemoptysis.

Signs on examination

The art of eliciting clinical signs is essential to corroborate findings from the history, but subject to interobserver variation.[5]

General

An initial assessment of how unwell the patient is, incorporating the ABC (airway, breathing, circulation…) approach and conscious level (i.e. Glasgow Coma Scale, or AVPU — Alert, response to Voice, Pain or Unresponsive) allows recognition of the need for immediate supportive care. Indeed, certain urgent situations such as tension pneumothorax require a quick correct diagnosis based only on the clinical signs. Check the observation charts for temperature, heart rate, blood pressure, respiratory rate, oxygen saturation and their trends, which may predict outcome. Cues from the patient's bedside include the sputum pot and respiratory paraphernalia (inhalers, bronchodilator nebulizer, high-flow oxygen, peak flow meter, incentive spirometer, positive airway pressure device). Ability to undress also provides information.

Hands and limbs

Examine the hands for clubbing, nicotine staining, a bounding pulse and the coarse flap of hypercapnia. Skin lesions suggestive of eczema, erythema nodosum (tender indurated raised plaques on the shins in sarcoidosis or tuberculosis), erythema multiforme (target lesions) in mycoplasma pneumonia or the purple hue of Gottron's patches in dermatomyositis should be sought, as should signs of intravenous drug use, tremor caused by β-adrenoreceptor and muscarinic agonists or hypoxic pulmonary osteoarthropathy (HPOA).

Clubbing: typical features are loss of the nailfold angle and a spongy sensation on pressing the nailbed, especially if the finger appears bulbous.

HPOA: this is characterized by pain and swelling of the wrists and ankles caused by subperiosteal new bone formation at the distal ends of the long bones, in association with lung cancer, particularly with superior vena cava obstruction (SVCO).

Head and neck

Look at the conjunctivae for the pallor of anaemia, and the tongue and lips for central cyanosis. This is detectable when the concentration of deoxygenated haemoglobin (deoxyHb) is greater than 15 g/litre or more than 10% of total haemoglobin. However, it may not

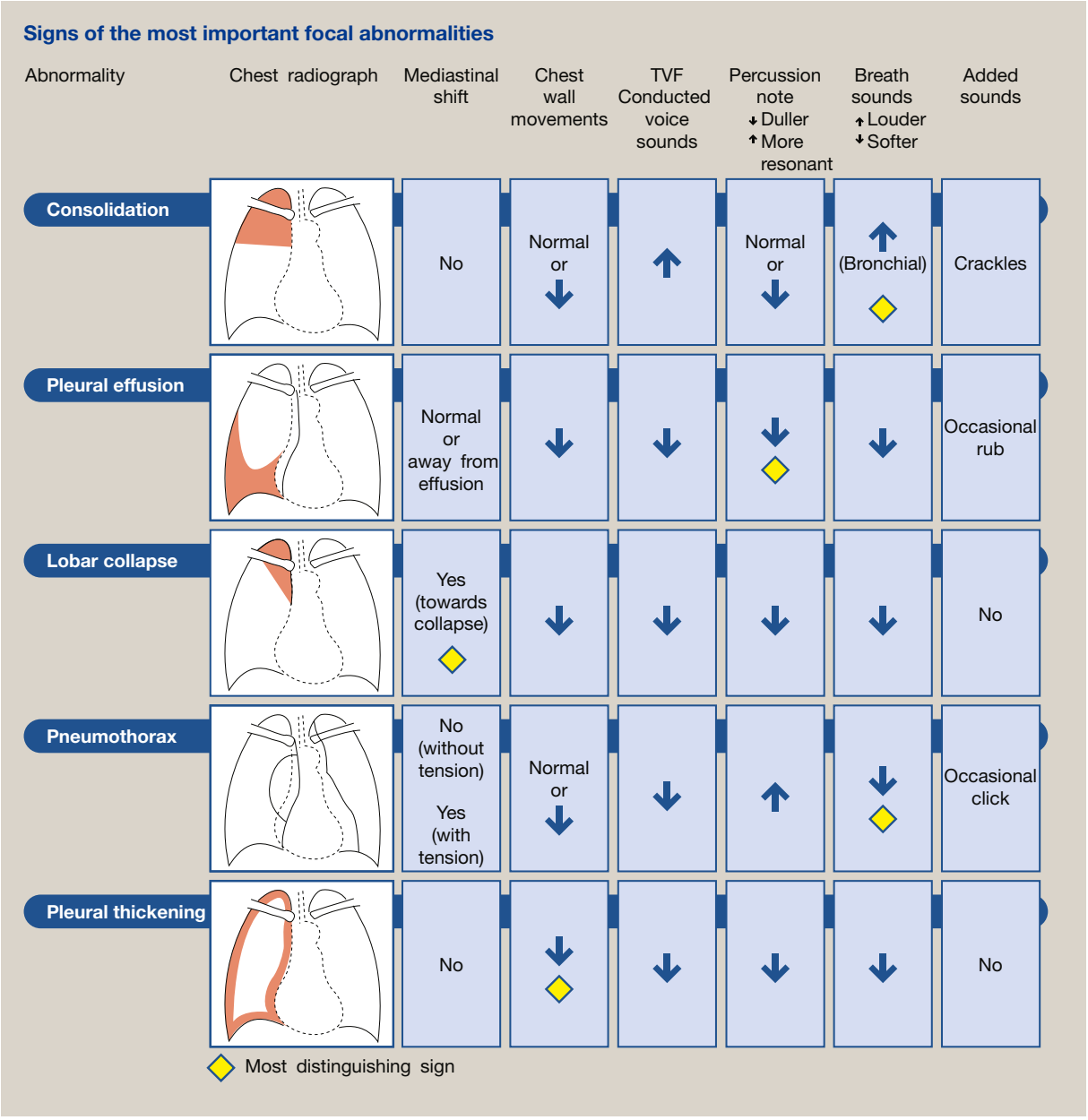

Figure 2

be a reliable sign of hypoxaemia, particularly in severe anaemia, where there is a low dexoxyHb concentration (<10 g/litre) or in polycythaemia (e.g. haemoglobin 200 g/litre), where cyanosis is present due to deoxyHb greater than 20 g/litre despite oxygen saturation being about 90%. Patients with Raynaud's syndrome will appear cyanosed in the cold, and 'apparent cyanosis' is the blue discoloration caused by pigment rather than deoxyHb — for instance, from methylene blue or methaemoglobinaemia (use of sulphonamides). Salivary gland enlargement (including the parotid and lacrimal glands) can be noted in sarcoidosis. Be aware of alternative systemic causes of lymphadenopathy, not least malignancy (e.g. lymphoma), and tuberculosis.

Chest

Examination of the chest should involve inspection, palpation, percussion and auscultation. Eliciting vocal resonance on auscultation is often more useful than tactile vocal fremitus (TVF) on palpation. For assimilation and synthesis of the symptoms and signs, decide which of the signs elicited are most reliable (Figure 2).

Ideally, position the patient at about 45°, comfortable and fully exposed. An awareness of surface anatomy is important for understanding the potential site of abnormal signs (Figure 1). Radiological thoracic anatomy (i.e. coronal and sagittal computed tomography) is helpful in contextualizing the surface anatomy. Thus, examination of the anterior right hemithorax will be mainly that of the upper lobe, with the middle lobe lower down the thorax, and the lower lobe laterally. The back of the thorax is occupied predominantly by the lower lobes. On percussion of the left anterior thorax, the absence of cardiac dullness suggests hyperexpansion of the lung (e.g. in emphysema) or displacement of the heart.

Inspection: inspection of the chest and vertebral column shape may reveal kyphoscoliosis, pectus excavatum or the 'barrel' chest of hyperinflation. Look carefully for scars of previous surgery (i.e. under skin-folds), radiotherapy markers, skin lesions, engorged chest wall veins (suggesting SVCO), hyperinflation, symmetry of chest wall movement and use of accessory muscles of respiration. Patients with severe airflow obstruction and hyperinflation will tend to fix their rib cage and arms, allowing the accessory muscles to lift the rib cage when diaphragmatic action is inefficient. These patients will also breathe with 'pursed lips', thus increasing intrinsic positive end-expiratory pressure, shifting the equal pressure point towards the mouth and delaying closure of the small airways during expiration. This reduces gas-trapping and subsequent dynamic hyperinflation. Abdominal breathing is best noted with the patient lying flat, if tolerated. In diaphragmatic weakness, there is indrawing of the abdomen on inspiration. An obstructed airway (e.g. sleep apnoea syndrome or reduced conscious state) also produces paradoxical abdominothoracic movements (Figure 2).

Palpation: adequate chest wall expansion (>3 cm) should be sought. Hyperinflation may limit this, as may pathologies causing pain or muscle weakness. Absence of symmetry should be noted (i.e. reduction on the side of the abnormal signs). Determine the position of the mediastinum by palpating the trachea and apex beat. Assessment for tracheal deviation can be performed with a single finger in the suprasternal notch, rolling it off the trachea on each side, or with fingers on either side of the trachea. A tracheal tug is upwards movement of the rib cage by the accessory muscles, shortening the palpable trachea in the suprasternal notch. It signifies increased respiratory effort, particularly in airflow obstruction.

Tactile vocal fremitus is the transmission of voice sounds from the central airways to the chest wall through patent conducting bronchi. It is increased by consolidated or locally fibrosed lung, but not by pleural effusion, pneumothorax or collapsed lung (except in the right upper lobe, due to collateral ventilation). TVF should be performed symmetrically over three or four regions anteriorly and posteriorly. It may be enhanced by the patient saying 'Ninety-nine'.

Percussion: this was introduced into clinical practice by the Austrian physician Leopold Auenbrugger in 1761, and promulgated by the Parisian physician Jean-Nicolas Corvisart after 1808. It should be performed by tapping the middle phalanx of the third finger placed in the intercostal space with the middle of the third finger on the tapping hand. Assess symmetry. Dullness suggests pleural fluid ('stony dull'), collapse or consolidation, while hyperresonance may indicate pneumothorax or severe emphysema.

Auscultation: René Théophile Hyacinthe Laënnec (1781–1826) is widely regarded as a forefather of the modern clinical assessment of respiratory diseases, through his invention of the stethoscope (1816), and publication of *De l'Auscultation médiate* (1819), in which he described auscultatory sounds and their pathoanatomical correlates.

Normal breath sounds are produced by turbulent airflow through the respiratory system. They should be assessed and characterized by their pitch (high, low), intensity (i.e. absent, enhanced), quality (harshness, loudness) and duration (inspiratory and expiratory phases). Typically, they are classified as tracheal, bronchial, vesicular or bronchovesicular. They are best heard depending on the area of thorax being auscultated. *Tracheal sounds* are high-pitched, loud and best heard over the neck. *Bronchial sounds* are high-pitched, 'blowing' and loud, with a gap between inspiration and expiration. *Vesicular sounds* are low-pitched, soft sounds with no gap, heard at the peripheries, as opposed to *bronchovesicular sounds*, which are high-pitched but soft (Table 4).

Added (adventitious) sounds are abnormal. They may be discontinuous (i.e. crackles, pleural rub) or continuous (i.e. wheezes, rhonchi–rales, stridor) Listen for their pitch, intensity, quality, duration and response to coughing (i.e. resolution of added sounds with mucus clearance) (Table 5).

Bronchial breathing heard at the periphery is abnormal. It occurs with consolidation and above pleural effusions (a term

Characteristics of normal breath sounds

Feature	Normal breath sounds			
	Tracheal	Bronchial	Bronchovesicular	Vesicular
Location	Trachea	Manubrium	Mainstem bronchi	Peripheral lung
Quality	Loud, harsh, hollow	Loud, less harsh, hollow	Soft	Softer
Pitch	Highest	Higher	High	Low
Duration	/\	/\	/\	/\

/ = inspiratory phase; \ = expiratory phase.

Table 4

Abnormal (adventitious) breath sounds

Discontinuous (non-musical)

- Crackles (generally high-pitched, discontinuous sounds)
  - Coarse: loud, low-pitched sounds
  - Fine: soft, high-pitched sounds
- Pleural friction rubs (grating sound)

Continuous (musical)

- Wheezes (high-pitched sounds that are musical in quality)
- Rhonchi (sounds with a 'snoring' or 'gurgling' quality)
- Stridors (sounds heard over a trachea)

Table 5

known as aegophony). Reduced or absent breath sounds indicate effusions, collapse, pneumothorax or an elevated diaphragm. *Crackles* (*crepitations*) represent the snapping opening of airways. They may be early and coarser in COPD, or later and fine in fibrosis or pulmonary oedema. Coarse, variable crackles that partially clear on coughing are caused by excessive bronchial secretions (e.g. bronchiectasis, pneumonia). *Wheeze* has a continuous, high-pitched musical quality. It usually occurs on expiration and may be generalized and polyphonic (e.g. asthma, COPD) or localized, fixed and monophonic (e.g. tumours, lymph glands, foreign body). *Stridor* is an example of a monophonic wheeze in inspiration from large airway narrowing. *Rhonchi* have a continuous, low-pitched snoring or gurgling quality, often due to secretions in the larger airways.

Vocal resonance is the elicitation of transmitted bronchial sounds by auscultation. It provides similar information to TVF and is present in bronchial breathing. *Whispering pectoriloquy* (when whispered sounds are transmitted clearly — 'speech of the chest') confirms bronchial breathing or cavitatory disease. *Aegophony* (from the greek for 'bleating of a goat) refers to the nasal sound heard over an area of consolidation or at the air—fluid interface of compressed lung above a pleural effusion. When asked to say 'E', the auscultated sound changes from an 'E' to an 'A'.

Pleural friction rubs (described as crunching snow underfoot or originally as the creaking of new leather '*bruit de cuir*') are heard in pneumonia, pleurisy and PE. The pericardial 'crunch' in time with the heartbeat is heard with a pneumomediastinum or small, left-sided pneumothorax. In obliterative bronchiolitis, crackles, wheezes and coarser creaking sounds may be heard. In extrinsic allergic alveolitis, late inspiratory squeaks or squawks (short, abrupt, high-pitched sounds), are often present, perhaps related to airway narrowing proximal to the alveoli.

Synthesis and analysis

Findings should be recorded coherently after the examination has been completed. The implications of any signs should be considered during the examination, and any uncertainties should be clarified by re-examination of a particular sign. Subsequent synthesis with the history should enable a differential diagnosis, from which relevant confirmatory tests may be requested. ◆

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