Respiratory symptoms and signs

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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