

FAST FACTS

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*Indispensable
Guides to
Clinical
Practice*

Respiratory Tract Infection

Second edition

by Robert C Read and Donald E Craven

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HEALTH PRESS

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Respiratory Tract Infection

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Glossary of abbreviations

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|--|--|
| ABPA: allergic bronchopulmonary aspergillosis | HAART: highly active antiretroviral therapy |
| AFB: acid-fast bacilli | HIV: human immunodeficiency virus |
| APACHE: acute physiology and chronic health evaluation | HRCT: high-resolution computed tomography |
| ARDS: adult respiratory distress syndrome | HSV: herpes simplex virus |
| BAL: bronchoalveolar lavage | ICU: intensive care unit |
| CAP: community-acquired pneumonia | IFA: immunofluorescence assay |
| CFTR: cystic fibrosis transmembrane regulator (protein) | IFN: interferon |
| CMV: cytomegalovirus | KS: Kaposi's sarcoma |
| COPD: chronic obstructive pulmonary disease | MDR: multi-drug-resistant |
| CPIS: clinical pulmonary infection score | MIC: minimum inhibitory concentration |
| CSF: cerebrospinal fluid | MRSA: methicillin-resistant <i>S. aureus</i> |
| CT: computed tomography | NPA: nasopharyngeal aspirate |
| DIC: , diffuse intravascular coagulation | PCP: <i>Pneumocystis carinii</i> pneumonia |
| DLCO: diffusing capacity for carbon monoxide | PCR: polymerase chain reaction |
| DOTS: directly observed therapy, short course | PEF: peak expiratory flow |
| FEV₁: forced expiratory volume in 1 second | PSB: protected specimen brush |
| FNA: fine-needle aspirate | PSI: pulmonary severity of illness |
| FVC: forced vital capacity | QEA: quantitative endotracheal aspirates |
| GNB: Gram-negative bacteria | SIRS: systemic inflammatory response syndrome |
| HAP: hospital-acquired pneumonia | TB: tuberculosis |
| | TNF: tumor necrosis factor |
| | VAP: ventilator-associated pneumonia |
| | VRE: vancomycin-resistant enterococci |

Pneumonia is a general term used to describe disease that leads to consolidation of the lung parenchyma. It is characterized by acute inflammation within the gas-exchanging areas of the lung with an intense infiltrate of neutrophils in and around the alveoli and the respiratory and terminal bronchioles. The affected bronchopulmonary segment or the entire lobe may be consolidated by the resulting inflammation and edema.

Epidemiology

In the UK, the rate of hospital admissions for pneumonia is approximately 1/1000/year. In the USA, the prevalence is estimated to be 12/1000/year (i.e. approximately 3.3 million cases/year), and bacterial pneumonias account for 500 000 hospital admissions/year of patients aged 15 years or older.

Pneumonia is substantially more common in the winter and affects males more often than females (ratio 2–3:1). It most commonly affects the elderly: in the USA, the incidence of pneumonia requiring hospitalization in those over 75 years of age is 11.6/1000/year, compared with 0.54/1000/year in those aged 35–44 years. Rates of pneumonia in the elderly are expected to double in the next 25 years.

Mortality and morbidity

Mortality due to community-acquired pneumonia (CAP) has decreased markedly since the introduction of antibiotics; the outcome for patients admitted to hospital with pneumonia can be greatly improved by prompt antibiotic therapy. Mortality from ambulatory pneumonia is now about 1% (ambulatory pneumonia is that affecting outpatients/people in the community). In hospitalized patients, however, mortality is approximately 13–15% and, in patients requiring intensive care, it ranges from 22% to 54%.

Patients who survive pneumonia generally recover completely, though there are occasionally long-term sequelae. Patients with sepsis

syndrome, usually in association with *Streptococcus pneumoniae*, *Legionella pneumophila* or *Klebsiella pneumoniae* infections, often suffer significant morbidity.

Pathogenesis

Most infections result from initial colonization of the upper respiratory tract by a pathogen, with subsequent translocation by aspiration into the lower airways. The most common cause of community-acquired pneumonia is *S. pneumoniae*, but a wide range of other pathogens may also be implicated (Table 1.1).

Assessment and treatment

Clinical features. In most patients, pneumonia usually develops over several days with cough and sputum production, dyspnea, pleuritic chest pain, weakness, malaise and often myalgia. Occasionally, the presentation may be hyperacute with a dramatic rigor as the first symptom; this is more common in healthy young adults. In older patients, the presentation may be more insidious, with minimal cough and absence of fever; confusion and hypothermia are often presenting features in this group.

Physical examination usually reveals fever, particularly in young individuals. Patients are usually uncomfortable and may often be breathless at rest. The trachea is usually central, but expansion on the affected side is reduced. Percussion is dull over the diseased lobe or lobes, and auscultation may reveal rales or bronchial breathing, depending on the degree of consolidation. Occasionally, there is evidence of an effusion with stony dullness on the affected side. Typical chest radiographs are reproduced in Figures 1.1 and 1.2.

In pneumococcal pneumonia, the sputum is classically rust colored, but can be mucoid, scanty or absent. In *Mycoplasma*, *Chlamydia*, *Legionella* and viral infections, sputum is usually absent. Characteristic epidemiological and clinical features and laboratory abnormalities that may point to a specific microbiological diagnosis are listed in Table 1.2.

Investigations. Chest radiography is critical for establishing the diagnosis and for distinguishing pneumonia from acute bronchitis.

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Infective exacerbation of chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is characterized by airflow obstruction due to chronic bronchitis or emphysema; this is defined as a ratio of forced expiratory volume in 1 second to forced vital capacity ($FEV_1:FVC$) of less than 70%. The decline in FEV_1 with advancing age is much greater in patients with COPD than in normal individuals, and is particularly rapid in those patients who continue to smoke (Figure 3.1). Infective exacerbations are also associated with an acute decline in FEV_1 . Although infection is the most common cause of death in patients with COPD, it is unclear whether infective exacerbations lead to an accelerated loss of lung function during the natural history of chronic bronchitis.

Epidemiology

In contrast to statistics for heart disease and cerebrovascular disease, the incidence and prevalence of COPD are increasing. In the USA,

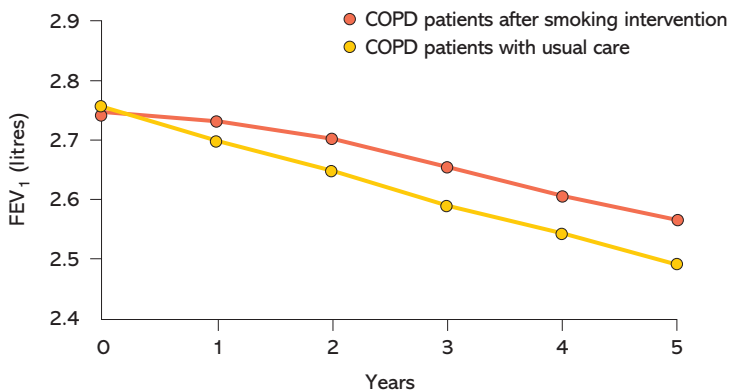


Figure 3.1 The accelerated decline in FEV_1 in individuals with COPD is greatest in those who continue to smoke. Data from Anthonisen et al. 1994.

COPD is the fourth leading cause of death, affecting 14% of adult men and 8% of adult women; UK figures are similar. Infective exacerbations of COPD are common during the winter months and occur, on average, three times per year.

COPD has a significant economic impact. In the UK, chronic bronchitis is associated with a loss of 28 million working days/year. In addition to this, COPD accounts for a large proportion of the £42 million (\$61 million) spent on antibiotic prescriptions for lower respiratory tract infections every year in the UK.

Pathology

The underlying pathology of stable COPD is airway remodeling as a consequence of chronic narrowing of the airways. In emphysema, the chronic airflow limitation is due to destruction of the alveolar structures, while in chronic bronchitis, it is due to chronic inflammation secondary to mucous-gland hypertrophy and hyperplasia, and peribronchial fibrosis.

In infective exacerbations of COPD, purulent sputum most commonly yields:

- *H. influenzae*
- *S. pneumoniae*
- *Moraxella catarrhalis*.

Indeed, these organisms are present in many patients between exacerbations, and their number simply increases during the infective episode. In most cases, the initiating factor of an infective exacerbation is unknown, but viral and *Mycoplasma* infections are responsible for up to one third of the acute exacerbations of COPD; the viruses most commonly identified include influenza A, para-influenza virus, coronavirus and rhinovirus.

H. influenzae has an affinity for airway epithelium and attaches to the damaged airway mucosa of patients with COPD (Figure 3.2). It adheres to mucus via pili, and to galactoside sequences on the epithelial cells. Soluble products of *H. influenzae* have been shown to reduce ciliary beat frequency and mucociliary clearance. In patients with more severe disease, Gram-negative bacilli (including *P. aeruginosa*) and *S. aureus* can be recovered from purulent sputum.