Respiratory tract infections are common in the community and in hospital, and today's physician has much to contend with:

- newly described respiratory pathogens
- emerging antibiotic resistance among previously known pathogens
- difficulty in reaching a rapid diagnosis to guide treatment
- high morbidity and mortality associated with improper selection of therapy.

Written by internationally recognized experts, *Fast Facts – Respiratory Tract Infection* is an up-to-date, logically organized and sharply focused practical guide to the management of these diseases. Several chapters have been completely rewritten for this revised edition, and a new chapter on bioterrorism has been included. Physicians, nurses and medical students will all find it invaluable.

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Respiratory Tract Infection

Second edition

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Glossary of abbreviations

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

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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.
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 (FEV₁:FVC) of less than 70%. The decline in FEV₁ 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 FEV₁. 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,
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.