ESSENTIALS

ESSENTIAL RESPIRATORY MEDICINE

SHANTHI PARAMOTHAYAN

WILEY Blackwell

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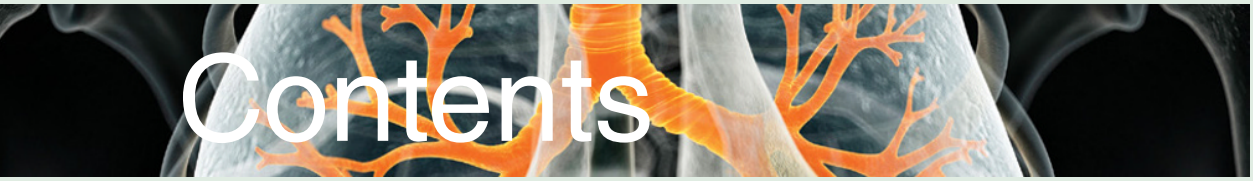
This textbook is dedicated to the memory of my aunt and teacher
Miss Sushila Balamani Navaratnasingam



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About the author

This textbook is written by Dr. Shanthi Paramothayan, a Consultant Respiratory Physician with 17 years of clinical experience in the NHS. As an Honorary Senior Lecturer for 15 years, the author has significant experience in teaching, assessing and examining undergraduates, foundation doctors, core medical trainees and respiratory registrars. She is a Fellow of the Royal College of Physicians, Fellow of the American College of Chest Physicians, and a Fellow of the Higher Education Academy. She has been a member of the Education and Training Committee of the British Thoracic Society, a member of the Question Writing Committee for the specialist respiratory examinations, a member of the MRCP 1 Board and a PACES examiner for the Royal College of Physicians. She has been a Foundation Training Programme Director, Director of Medical Education, Associate Medical Director for Education and Associate Foundation Quality Dean, Health Education South London.

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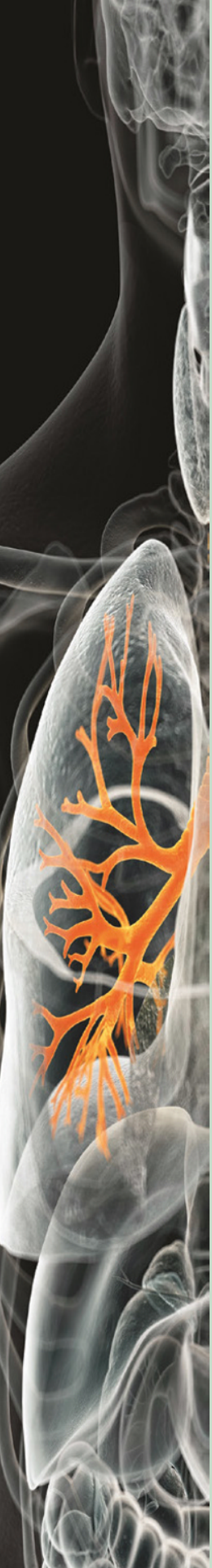
About the companion website

This book is accompanied by a companion website:

www.wiley.com/go/paramothayan/essential_respiratory_medicine

The website includes:
- Image bank
- Videos of patient examination
- Example respiratory sounds
- Multiple-choice questions

Scan this QR code to visit the companion website:



CHAPTER 1

Introduction to respiratory medicine

Essential Respiratory Medicine, First Edition. Shanthi Paramothayan.

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The respiratory system is essential for gas exchange in a multicellular organism. The lungs are also important as a defence against infectious microorganisms. Worldwide, diseases of the respiratory system cause significant morbidity and mortality; this includes infectious diseases, malignancies, allergic diseases, autoimmune disorders, and occupational diseases. Diseases of other parts of the body, for example, rheumatological and renal conditions, often affect the lungs.

Respiratory diseases can present acutely with severe, life-threatening breathlessness, for example, when someone develops a pulmonary embolus or a pneumothorax, or more insidiously with a steady decline in lung function over time, as occurs in chronic obstructive pulmonary disease or parenchymal lung diseases. In the United Kingdom (UK), respiratory diseases account for one-third of acute admissions to hospitals and for more than a quarter of all deaths in hospitals. Respiratory tract infections are the commonest conditions seen in General Practice.

In the last half a century there has been a decline in the prevalence of certain diseases, such as pneumoconioses, and other occupational lung diseases because of the recognition of the harm caused by exposure to certain agents at work. The introduction of masks, better ventilation, and other safety measures at work, together with appropriate legislation, has been the key to this success.

In the next few decades it is likely that asbestos-associated diseases (asbestosis and mesothelioma) will reduce in incidence and prevalence in the UK because of the prohibition of the use of asbestos. Asbestos, however, is still used in several developing countries. The recognition that air pollution is responsible for respiratory diseases will, hopefully, lead to cleaner air, especially in urban areas.

However, there has been an increase in the prevalence of allergic asthma, and there are various hypotheses to explain this increase. *Mycobacterium tuberculosis* has still not been eradicated, resulting in millions of deaths across the globe. Tuberculosis, also called 'phthism', 'consumption', or the 'white plague', was found in the spines of Egyptian mummies dating back to 3200–2400 BCE and is associated with poverty and deprivation.

Respiratory diseases are managed jointly by respiratory physicians, specialist nurses, physiotherapists, and occupational therapists in a multi-disciplinary way. Other specialists, including radiologists, pathologists, oncologists, thoracic surgeons, palliative care physicians, intensivists, and physiologists (for example, lung function technicians) are also essential in the management of patients with respiratory diseases. Patients who are acutely ill are managed in hospital, often on specialist respiratory wards, sometimes in single rooms if infectious, and in the Intensive Care Unit if respiratory support is required.

There has been increasing understanding of the physiology of the respiratory system and the pathophysiology of respiratory diseases in the last few centuries. Table 1.1 summarises some of the key developments in respiratory medicine.

About the book

Respiratory diseases are common, and this textbook offers a practical guide to those who care for patients with respiratory diseases. This textbook is aimed at medical students studying for their MBBS examination and postgraduate doctors of all grades, especially those studying for postgraduate examinations, including the MRCP examination. This book will also be useful for non-respiratory doctors, specialist nurses, physiotherapists, occupational therapists, pharmacists, respiratory physiologists, and physicians associates.

This text covers the entire respiratory curriculum and contains information that is useful and relevant to everyday clinical practice, with a focus on clinical presentation and management. Essential basic anatomy, physiology, pharmacology, and pathology are introduced to help understand the clinical presentation. A structured approach is taken to explain how to construct a sensible differential diagnosis of common respiratory conditions. There is a clear explanation of the common diagnostic tests required to make a diagnosis, including the interpretation of lung function tests. The mechanism of action of drugs commonly prescribed to treat respiratory diseases is discussed, with a description of their common side effects and interaction with other medications. The evidence-based management of common conditions is discussed with reference to the current British Thoracic Society (BTS) and National Institute for Health and Care Excellence (NICE) guidelines. Common pitfalls in diagnosis and management are highlighted.

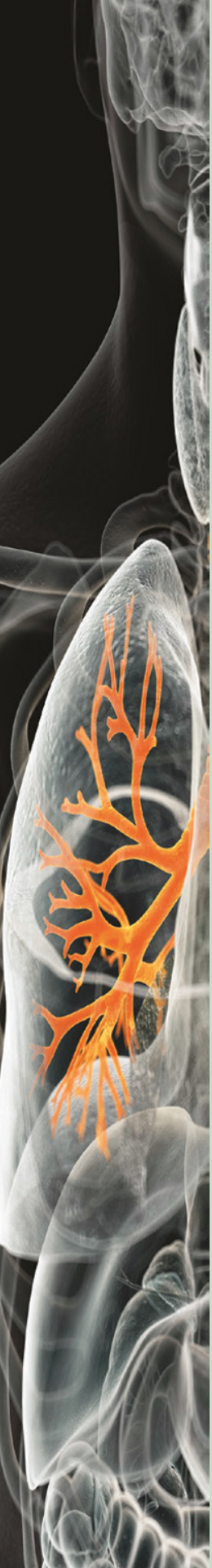
Table 1.1 Brief history of respiratory medicine.

Year	Development	Scientist
Greece, 460–370 BCE	Beginning of modern medicine	Hippocrates
Greece, 304–250 BCE	Some understanding of the physiology of the lung	Erisistratus
Greece, 129–165 BCE	Anatomy of trachea, larynx, and lungs understood Believed air had substance vital for life	Galen
Egypt, 1210–1288	Some understanding of pulmonary circulation	Ibne Nafis
Italy, 1500	Understood anatomy and physiology of lungs Determined sub-atmospheric pressures inflated lungs	Leonardo da Vinci
Belgium, 1543	Tracheostomy used for ventilation	Andreas Vesalius
UK, 1700	Constructed first air pump for physiological research	Robert Hooke
France, 1778	Discovered role of oxygen	Antoine Lavoisier
France, 1816	Invention of stethoscope	René Laennec
Scotland, 1832	Invention of negative pressure tank-type ventilator	John Dalziel
Germany, 1882	Tuberculosis bacterium discovered	Robert Koch
Germany, 1895	First chest X-ray	Wilhelm Rötgen
UK, 1928	First non-invasive ventilation	Drinker-Shaw
USA, 1963	First human lung transplant	James Hardy
UK, 1972	First computed tomography scan	Godfrey Hounsfield

The book contains several boxes, tables, and algorithms set out in a clear, and concise way. It also contains several good quality colour photographs, and radiological and histological images to support the information in the text.

There are multiple choice questions which can be used by the reader to check their understanding, with a clear explanation of the correct answer. There is also a list of references for suggested further reading.

Supplementary material includes videos demonstrating how to take a history and conduct a clinical examination (http://www.wiley.com/go/Paramothayan/Essential_Respiratory_Medicine). There are also videos showing how to carry out common tests, such as peak flow, spirometry, the skin prick test, the Mantoux test, the shuttle test, and how to fit a patient for a sleep study.



CHAPTER 2

Embryology, anatomy, and physiology of the lung

Learning objectives

- To gain a basic understanding of the development of the lung
- To be aware of the common developmental lung abnormalities
- To understand the anatomy of the respiratory system which is relevant to clinical practice
- To be aware of the structure and function of the diaphragm
- To understand the muscles of respiration
- To understand how mechanical ventilation occurs
- To gain knowledge of the structure of the bronchial tree and the alveoli
- To gain knowledge of the blood supply, nerve supply, and lymphatics of the respiratory system
- To understand the physiology of the respiratory system which is relevant to clinical practice
- To gain some understanding of the control of breathing
- To gain knowledge of the receptors in the lungs
- To appreciate the function of the central and peripheral chemoreceptors
- To understand how oxygen is transported in the blood from the lungs to tissues
- To understand how carbon dioxide is transported in the blood from tissues to the lungs
- To understand the importance of carbon dioxide in the acid-base balance of the body
- To understand the causes of physiological shunts
- To understand the causes of ventilation-perfusion mismatch
- To have some understanding of the defence mechanisms of the lungs

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Abbreviations

ASD	atrial septal defect
CA	carbonic anhydrase
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
CSF	cerebrospinal fluid
FRC	functional residual capacity
H^+	hydrogen ion
H_2CO_3	carbonic acid
HB	haemoglobin
HCO_3^-	bicarbonate ion
MCE	mucociliary escalator
NANC	non-noradrenergic, non-cholinergic
NO	nitric oxide
O_2	oxygen
O_3	ozone
PCD	primary ciliary dyskinesia
PCO_2	partial pressure of carbon dioxide
PO_2	partial pressure of oxygen
R	respiratory quotient
SO_2	sulphur dioxide
VSD	ventricular septal defect

Introduction

The respiratory system's main role is to provide oxygen (O_2) that is required for glycolysis, and the removal of the waste product of respiration, carbon dioxide (CO_2). This involves two separate processes: (1) mechanical ventilation whereby air is moved into and out of the lungs, and (2) gas exchange across the alveolar-capillary membrane.

The respiratory system also has an important role in acid-base balance, the defence against airborne pathogens, and in phonation, which is essential for audible speech. The conversion of angiotensin 1 to angiotensin 11 occurs in the lungs as does the deactivation of bradykinin, serotonin, and various drugs, including propranolol.

The lungs act as a reservoir of 500 ml blood and therefore participate in heat exchange. The lungs filter and lyse microemboli from the veins, preventing them from reaching the systemic circulation.

Development of the respiratory system

The lungs are not required for respiration *in utero*, but start working as soon as the baby is born and is independent from its mother. The development of the lungs starts in week three of the embryonic period (3–16 weeks), continues through the foetal period (16–38 weeks), beyond birth, and into childhood. During intrauterine life, the lungs are an important source of amniotic fluid, producing around 15 ml kg^{-1} of body weight, which flows out via the trachea or is swallowed.

Development of the lungs

During the embryonic period, the structures of the respiratory system are formed: the trachea, bronchial tree, blood vessels, nerves, lymphatics, and the structures of the thoracic cage (Figure 2.1). In the latter part of the second trimester and during the third trimester, there is functional development, with lung maturation and the production of surfactant. Five phases of structural lung development are recognised. In the embryonic phase (3–16 weeks), at approximately 28 days after conception, lung development begins with the formation of the sulcus laryngotrachealis in the lower part of the pharynx. At 30 days, a bud, called the true lung primordium, forms from the lower part of the foregut, but remains in communication with it. The oesophagotracheal ridges then fuse to form the oesophagotracheal septum, which divides the oesophagus from the trachea. Failure of the formation of this septum occurs in 1 : 3000 births and results in the formation of a trachea-oesophageal fistula.

The diaphragm develops in the third week after fertilisation, with transverse and longitudinal folding. The septum transversum is the primitive central tendon and forms in the cervical region and migrates downwards, therefore the innervation is from the phrenic nerve that originates from the cervical spinal cord.

Failure of one of the pleuroperitoneal membranes to close results in a congenital diaphragmatic hernia which occurs in 1 : 2000 births. It occurs more commonly on the left side and results in the intestinal contents moving up into the left hemithorax, compromising lung development resulting in lung hypoplasia. Surgical repair carries a high mortality.

Normal lung development depends on the interaction between the epithelium and the mesenchymal tissue which lies beneath it. During the pseudoglandular period of the embryonic phase (5–16 weeks), there is an asymmetrical subdivision

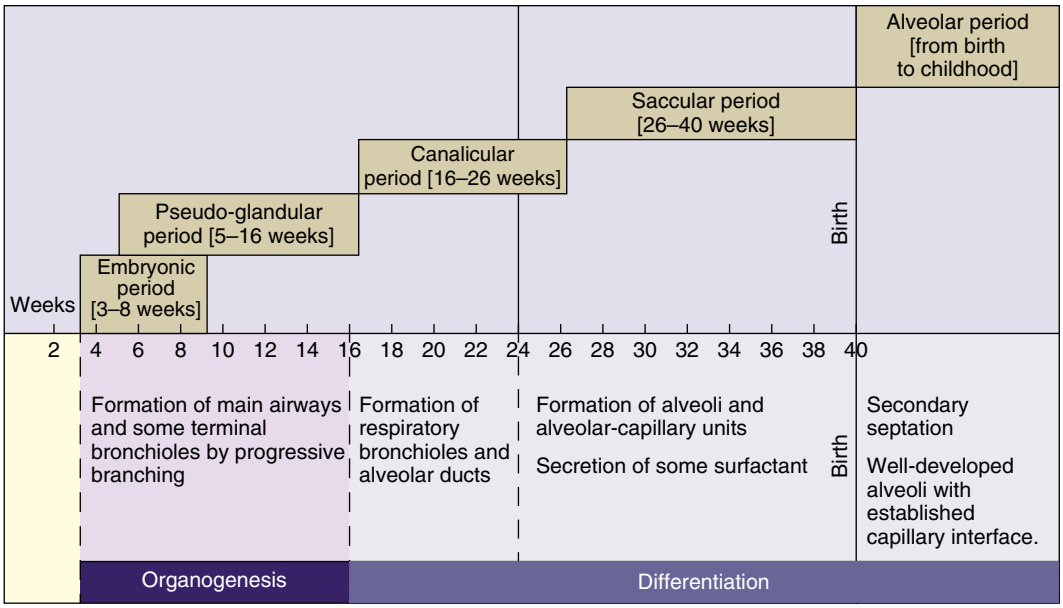

Figure 2.1 Stages of lung development.

of the lung primordium into the two buds which will form the main bronchi. The smaller left main bronchus is directed more acutely away from the trachea while the larger right main bronchus leads more directly from the trachea. The two main bronchi subdivide unequally, giving rise to three lobes on the right and two lobes on the left.

Progressive branching during the embryonic phase results in the formation of the first 16 generations of the conducting airways, composed of the trachea, bronchi, bronchioles, and terminal bronchioles. Differentiation of the epithelium derived from the endoderm, with formation of cilia in the proximal airways, occurs at 13 weeks and is controlled by the mesenchyme beneath it. This ciliated epithelium lines the entire conducting airway system and is important in host defence. In primary ciliary dyskinesia (PCD), the ciliary structure is abnormal, and the consequences are significant, as discussed in Chapter 12. The innervation of the lungs is derived from the ectoderm while the vascular structures, smooth muscle, cartilage, and connective tissue are derived from the mesoderm.

During the canalicular period (16–26 weeks), there is further branching of the bronchial tree, with the terminal bronchioles dividing into the respiratory bronchioles (generations 20–22), which further subdivide into the alveolar ducts (generations 20–22) and finally the alveolar sacs (generation 23). Generations 17–23 are called the respiratory zones and will be responsible for gas exchange. Once the alveolar sacs have been formed, further growth occurs by elongation and widening of the airways.

Type 1 pneumocytes, the main cells of the alveolus, are formed with very thin membranes. There is vascularization, with establishment of the capillary network very close to the type 1 pneumocytes in preparation for the gas exchange. Type 2 pneumocytes, which contain lamellar (or inclusion) bodies, also develop and will eventually synthesise and store surfactant.

At the end of the embryonic period (16 weeks), the pulmonary vessels have developed. The pulmonary circulatory system is smaller than the systemic circulatory system and is formed out of the sixth pharyngeal arch artery and a vessel plexus which originates from the aortic sac. The true sixth aortic arch is only then formed after vessels from the dorsal arch grow into this plexus and there is a connection between the truncus pulmonalis and the dorsal aorta.

During the terminal sac period of foetal development (26–38 weeks), there is further differentiation of the type 1 and type 2 pneumocytes, with progressive thinning of the alveolar walls which will facilitate gas exchange.

At full gestation, there are approximately 20×10^6 alveoli, often called 'primitive saccules', which mature during the neonatal period and connect to other alveoli through the pores of Kuhn. The pulmonary arterial network gradually develops a muscle layer during childhood and the capillary network extends and becomes entwined between two alveoli. The lungs continue to develop after birth until the age of 8, with the formation of a total of 300×10^6 mature alveoli.

As the alveoli in the foetus contain fluid and not air, the oxygen tension is low, resulting in pulmonary vasoconstriction and diversion of blood across the ductus arteriosus into the systemic circulation. After the first breath is taken, oxygen enters the alveoli, resulting in an increase in oxygen tension and increased blood flow to the alveoli. Nitric oxide (NO), a potent vasodilator, is secreted by the respiratory epithelium which results in significant vasodilation of the pulmonary blood vessels.

Surfactant is composed of a hydrophilic macromolecular complex of phosphatidylcholine (lecithin), phosphatidylglycerol and hydrophobic surface proteins B and C which project into the alveolar gas and float on the surface of the lining fluid. Surfactant decreases surface tension within the alveoli, preventing the collapse of the alveoli during exhalation. In the absence of surfactant, the alveolus would be unstable and would collapse at the end of each breath. During the latter part of gestation, surfactant production and secretion gradually increase. At 36 weeks of gestation there is sufficient surfactant so that spontaneous breathing can occur and the foetus is viable.

Prematurity carries a high mortality and a significant risk of neonatal respiratory distress syndrome. Corticotrophin stimulates the synthesis of the fibroblast pneumocyte factor from the foetal lung fibroblasts which stimulates surfactant production in type 2 cells. Corticosteroids given antenatally to premature babies will promote lung maturity. Exogenous surfactant can also improve the survival of the premature baby.

Amniotic fluid, originating in the foetal lungs and kidneys, is required for normal lung development. During foetal breathing movements, when the upper airways' resistance is decreased, diaphragmatic movements help maintain lung liquid volume. Oligohydramnios, called Potter's syndrome, occurs when there is a decreased volume of amniotic fluid, resulting in lung hypoplasia and renal agenesis. Other causes of lung hypoplasia include congenital diaphragmatic hernia, musculoskeletal abnormalities of the thorax which restrict the full expansion of the thoracic cage, and space-occupying lesions of the thorax.

The respiratory tract

The **upper respiratory tract** comprises of the nose, the paranasal sinuses, the epiglottis, pharynx, and larynx (Figure 2.2). The larynx is important in speech. During swallowing, the epiglottis closes the larynx which leads to the trachea, preventing food from entering the respiratory tract. Failure of this process will lead to aspiration of food contents into the lungs.

The **lower respiratory tract** begins at the trachea, which corresponds to the lower edge of the cricoid cartilage, at the level of the sixth cervical vertebra. The lower respiratory tract is enclosed within the thoracic cavity which is composed of the sternum anteriorly, the vertebral column posteriorly, the mediastinum, the diaphragm, which divides the thorax from the abdomen, and the ribs with their intercostal spaces (Figure 2.3, Figure 2.4). The bony sternum is divided into the manubrium, the body, and the xiphisternum, which is cartilaginous until late adulthood. The manubrium is joined to the cartilages of the first and second ribs at the level of T3 and T4, and to the body by the manubriosternal joint which lies at T4 and is called the angle of Louis or the sternal angle. This is an important landmark in surface anatomy. The body of the sternum joins the second to seventh ribs at the level of T5–T8.

The vertebrosternal, or true ribs, are the first to seventh ribs, and are connected to the sternum by their costal cartilages. Inflammation of the costochondral junction (costochondritis) results in 'pleuritic' chest pain which is worse on breathing, movement, and palpation. The eighth, ninth, and tenth ribs are called the vertebrochondral, or false ribs, and are joined to the cartilages of the ribs above. The eleventh and twelfth ribs are called floating or vertebral ribs.

Each rib is composed of a head and a shaft. The head is attached to the body and transverse process of the adjacent vertebra, the intervertebral disc, and the vertebra above (Figure 2.5). The shaft curves forward to join the sternum. The joints between the ribs and vertebra act like a hinge, causing the ribs to move during inspiration.

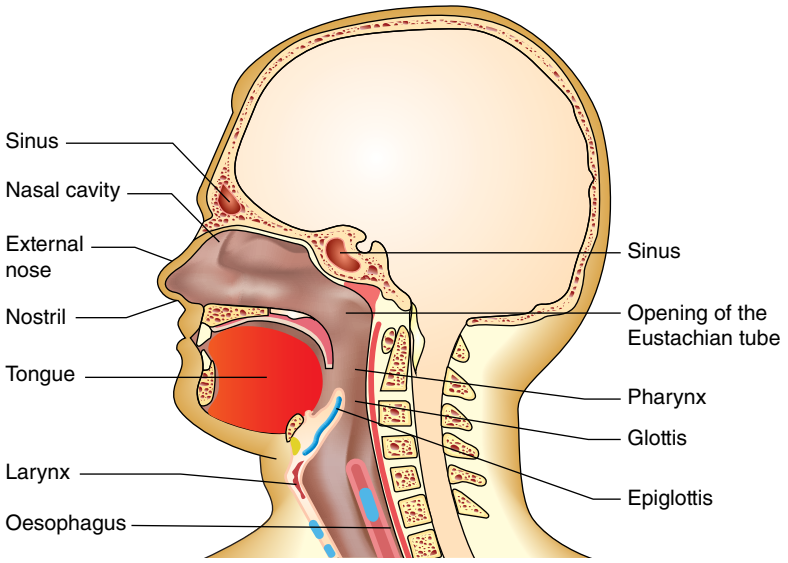

Figure 2.2 The upper respiratory tract.

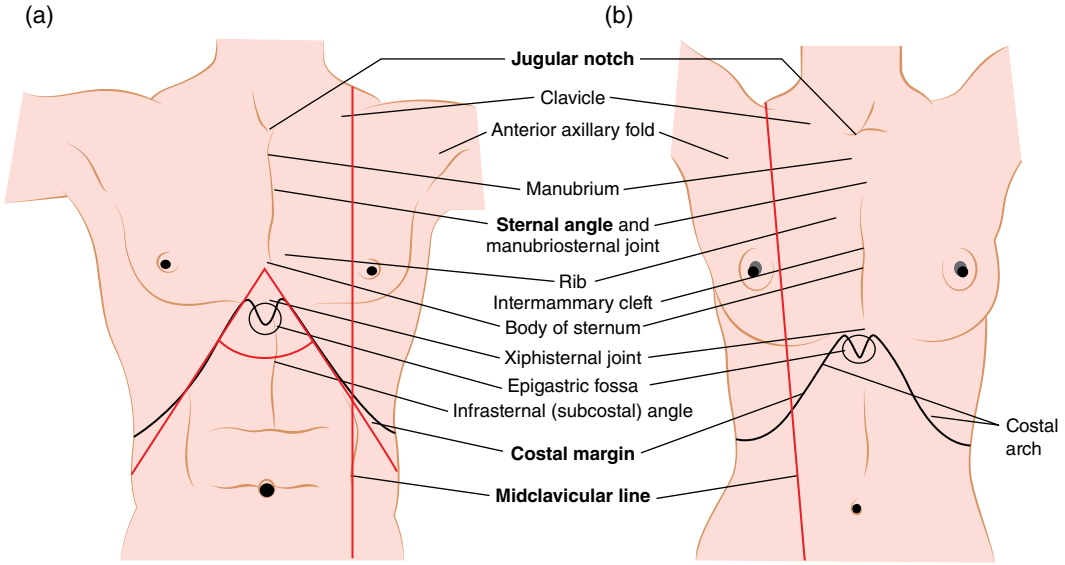

Figure 2.3 Surface anatomy of the thorax.

The rib cage protects the heart, lungs, and great vessels from damage. Trauma to the chest wall can result in fracture of the shaft of the ribs at the angle of the rib. Multiple rib fractures can result in a 'flail' segment which can cause significant difficulty with inspiration. The clavicles protect the first and second ribs which are less likely to fracture than the other ribs.

One in 200 people have a cervical rib which is attached to the transverse process of C7. A cervical rib can press on the brachial plexus and cause neurological symptoms, including paraesthesia of the arms and hands. Pressure on the subclavian artery can cause vascular symptoms.

The intercostal spaces between the ribs contain external and internal intercostal muscles (Figure 2.6).

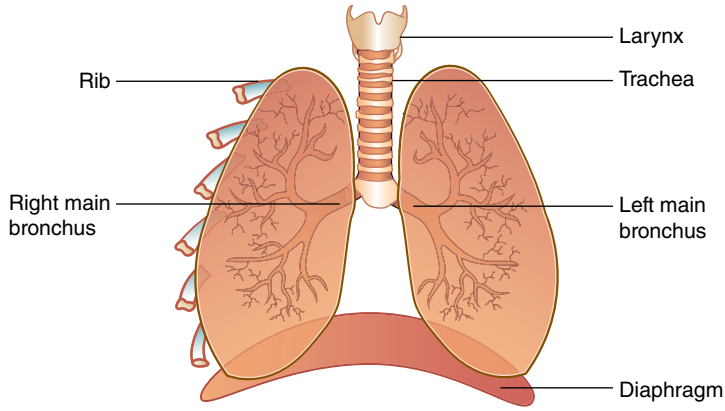

Figure 2.4 The lower respiratory tract.

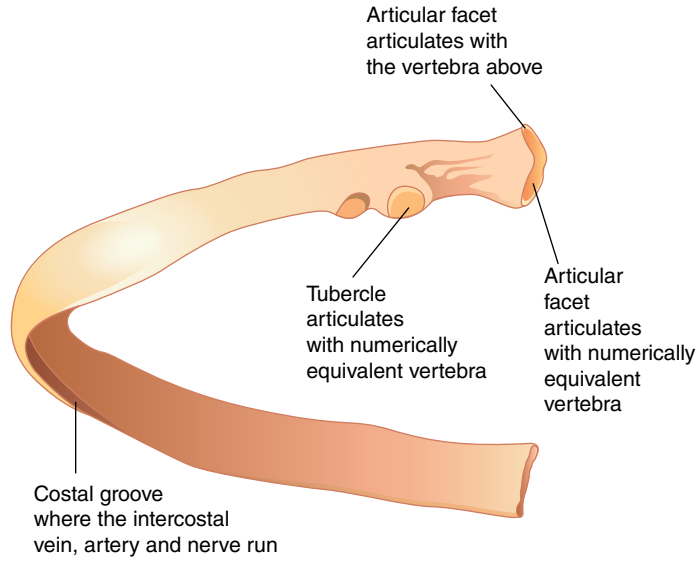

Figure 2.5 Structure of the rib.

The fibres of the external intercostal muscles pass downwards and forwards between the ribs, while the fibres of the internal intercostal muscles pass downwards and backwards. There is also an incomplete innermost intercostal layer. The intercostal muscles are innervated by the intercostal nerves, which are the anterior primary rami of thoracic nerves. The intercostal veins, arteries and nerves lie in grooves on the under-surface of the corresponding ribs, with the vein above, the artery in the middle and the nerve below. It is important, therefore, to avoid the underside of the rib when carrying out pleural procedures, but to insert the needle or drain just above the rib into the pleural space.

The **diaphragm**, which means 'partition' in Greek, has a central tendon which is attached to the pericardium, and thick skeletal muscle on either side, which separates the thoracic and abdominal cavities. It is the most important muscle of inspiration. Several key structures traverse the diaphragm between the abdomen and thorax. The sternal part of the diaphragm consists of two strips of muscle that arises from the posterior surface of the xiphisternum. The costal part comprises of six

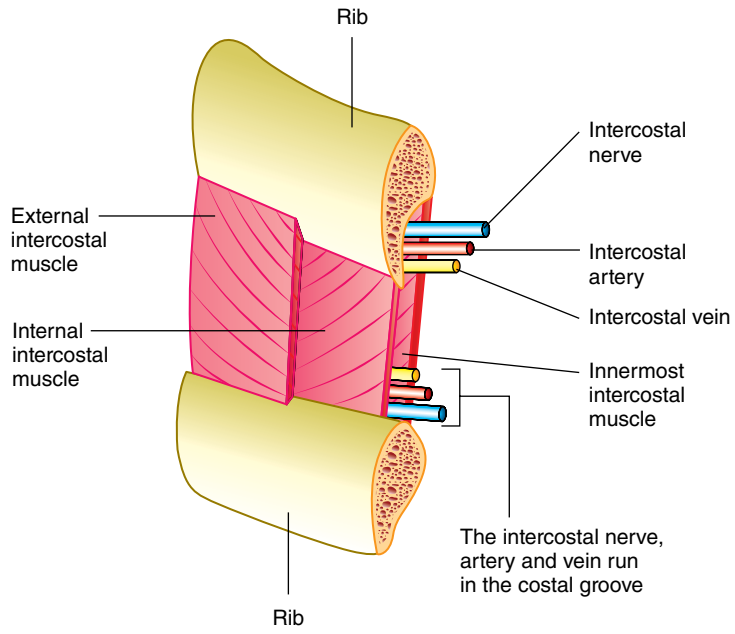

Figure 2.6 The ribs and intercostal space.

muscular strips that originate from the seventh–twelfth ribs and their costal cartilages. The vertebral part of the diaphragm originates from the crura and the arcuate ligaments on both sides. The muscular right crus arises from the bodies and intervertebral discs of the three lumbar vertebrae, and the left crus arises from the bodies and intervertebral discs of the upper two lumbar vertebrae. The medial and lateral arcuate ligaments are thickenings of the fascia overlying the psoas major and the quadratus lumborum respectively.

The inferior vena cava and right phrenic nerve pass through the diaphragm at T8, the oesophagus, branches of the left gastric artery, the gastric vein, and both vagi pass through at T10, and the aorta, thoracic duct, and zygos vein pass behind the diaphragm between the left and right crus at T12 (Figure 2.7). The sympathetic trunk passes through the diaphragm under the medial lumbocostal arch, and branches of the internal thoracic artery and lymphatics pass through the foramina of Morgagni.

The phrenic nerves (C3, C4, and C5) supply motor and sensory innervation to the diaphragm. Pain from irritation of the diaphragm is referred to the corresponding dermatome for C4 at the shoulder. Irritation to the phrenic nerve can cause intractable hiccoughs. The lower intercostal (T5–T11) and subcostal (T12) nerves supply sensory fibres to the peripheral diaphragm. Damage to the phrenic nerve, for example, by a tumour, will result in a unilateral diaphragmatic palsy, as discussed in Chapter 9.

The blood supply to the diaphragm is from the pericardiophrenic, musculophrenic, lower internal intercostal and inferior phrenic arteries. The superior and inferior phrenic veins drain blood from the diaphragm into the brachiocephalic vein, the azygos vein, the inferior vena cava, and the left suprarenal vein.

Muscles of respiration and mechanical ventilation

The inspiratory muscles are the diaphragm, and the intercostal and the scalene muscles. When they contract to expand the thoracic cavity, there is a decrease in intrapleural and alveolar pressure which creates a pressure gradient between the alveoli and the mouth, resulting in air entering the lungs. Elastic recoil of the lungs and the chest wall results in expiration, which is a passive process, not requiring any muscular activity. Forced expiration, for example, coughing, will require contraction of the abdominal muscles which push the diaphragm upwards.

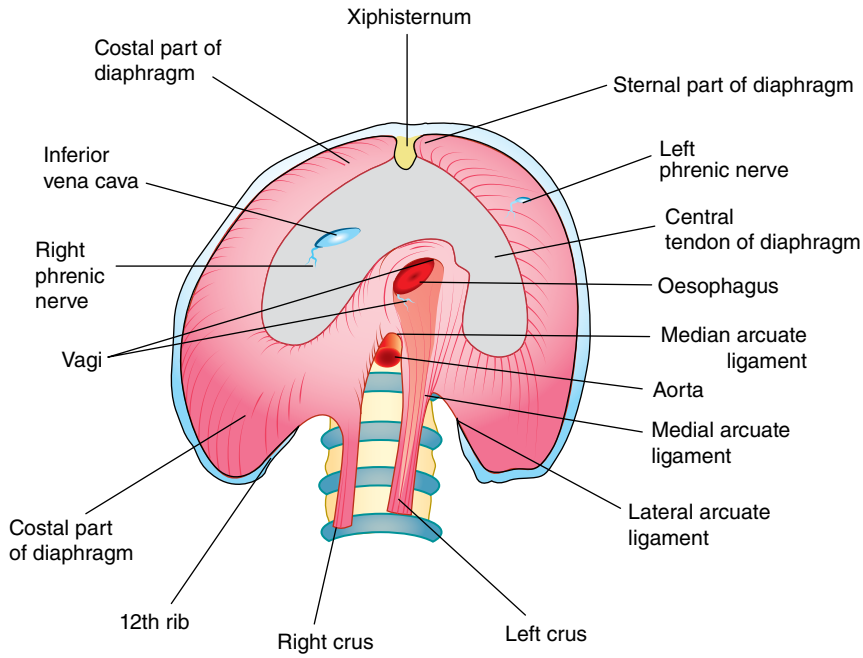

Figure 2.7 Diaphragm and the structures that traverse it.

Inspiration is an active process. The domed diaphragm is the main muscle of inspiration and is positioned high in the thorax at the end of expiration. During quiet breathing, the diaphragm contracts and moves down by 1.5 cm, pushing the abdominal contents down. This increases the intra-abdominal pressure and pushes the abdominal wall and the lower ribs outwards and downwards. During deep breathing, the diaphragm contracts harder and can move by as much as 6–7 cm.

During quiet breathing, the first rib remains almost motionless and the intercostal muscles elevate and evert the other ribs. The intercostal muscles support the intercostal spaces preventing them from being sucked in during inspiration. The scalene muscles, which insert into the first two ribs, are also active in normal inspiration. Movement of the upper ribs upwards pushes the sternum forward (the pump action), increasing the anterior–posterior diameter of the chest, and as the sloping lower ribs rise, they move out (the bucket handle action), and the transverse diameter of the chest wall increases. At the beginning of inspiration, the inspiratory muscles contract to overcome the impedance offered by the lungs and chest wall. The volume of the thoracic cavity can increase from 1.5 l up to 8 l with deep inspiration.

Diaphragmatic paralysis results in paradoxical movement: as the intercostal muscles contract and the ribs move, the diaphragm is sucked into the chest due to a fall in intrathoracic pressure. In a high cervical cord transection, all the respiratory muscles are paralysed, but when the damage is below the phrenic nerve roots, breathing continues via the diaphragm alone. In infants, the movement of the horizontal ribs cannot increase the volume of the chest, and breathing is reliant on diaphragmatic contraction alone; this is called abdominal breathing. As the infant grows, the ribs become more oblique and contribute to thoracic inspiration.

When the rate of ventilation or the resistance to breathing increases, the scalene muscles, sternocleidomastoids, and serratus anterior, which are called the accessory inspiratory muscles, are recruited to help inspiration. Splinting of the arms, for example, by grasping the edge of the table, will result in contraction of the pectoralis major muscle which will expand the chest further. When ventilation exceeds 40 lmin^{-1}, there is activation of the expiratory muscles, especially the abdominal muscles, the rectus abdominis, the external and internal

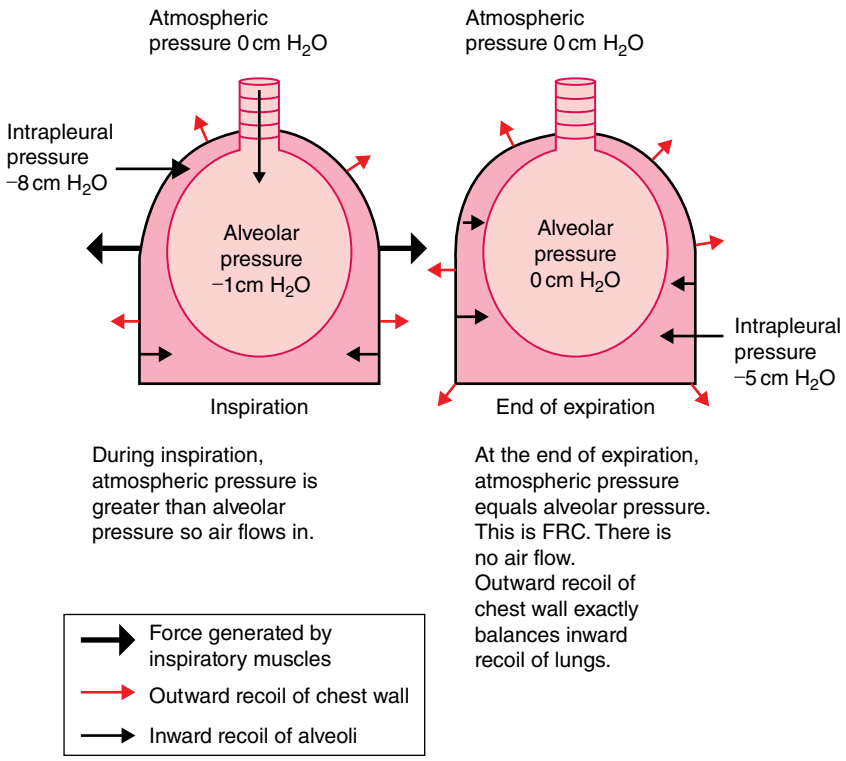

Figure 2.8 Relationship between elastic recoil and functional residual capacity.

oblique, which speed up recoil of the diaphragm by raising intra-abdominal pressure.

At functional residual capacity (FRC), the respiratory muscles are relaxed, and the outward recoil of the chest wall exactly balances the inward recoil of the lungs which creates a negative pressure in the space between them (Figure 2.8).

In lung fibrosis, the lungs are stiff (decreased lung compliance) and have increased elastic recoil, so the FRC is smaller. In emphysema, the FRC increases due to loss of alveolar tissue, loss of elastic recoil, increase in lung compliance, and air trapping. This leads to the development of a barrel chest. Mouth breathing, as adopted by patients with chronic obstructive pulmonary disease (COPD), decreases the FRC, enabling these patients to inspire.

Dynamic and static lung volumes and their measurements are discussed in detail in Chapter 4. The normal breath is called the tidal volume and is about 500 ml at rest, which is 10% of the vital capacity. At a normal respiratory rate of 15 breaths min^{-1}, the minute ventilation, which is the volume of air entering the lungs each minute is 7500 ml min^{-1} (500 × 15). Alveolar ventilation is the actual volume taking part in gas exchange every minute. As the dead space is 150 ml, alveolar ventilation is 5250 ml min^{-1} (7500-2250 ml/min).

The main resistance to airflow occurs in the upper respiratory tract, especially the nose, pharynx, and the large airways. The intrapleural pressure can be indirectly assessed from oesophageal pressure using a small pressure transducer. During inspiration, the chest wall expands and the intrapleural pressure falls. This increases the pressure gradient between the intrapleural space and the alveoli, stretching the lungs. The alveoli expand, and alveolar pressure falls, creating a pressure gradient between the mouth and the alveoli, causing air to flow into the lungs. During expiration, both intrapleural pressure and alveolar pressure rise. In quiet breathing, the intrapleural pressure remains negative for the whole respiratory cycle, whereas alveolar pressure is negative during inspiration and positive during expiration. Alveolar pressure is always higher than intrapleural pressure because of

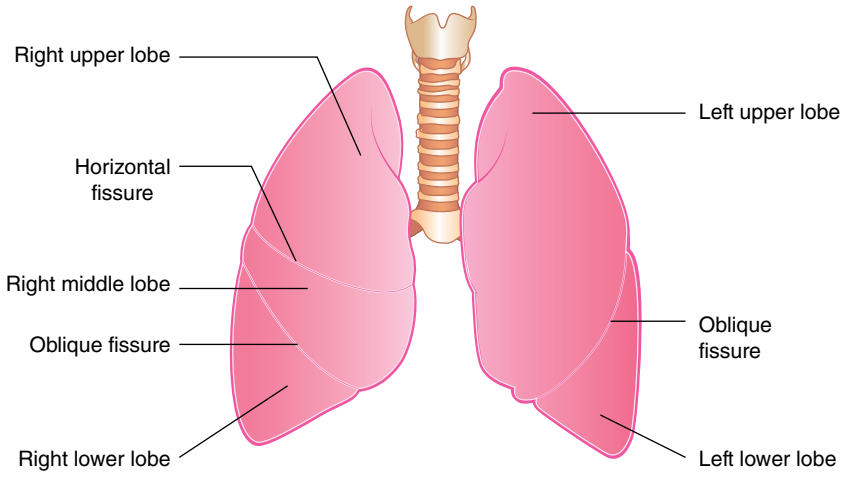

Figure 2.9 Lobes and fissures of the lungs.

the recoil of the lungs. It is zero at the end of both inspiration and expiration, and airflow ceases momentarily. When ventilation is increased, the changes in intrapleural pressure and alveolar pressure are greater, and in expiration intrapleural pressure may rise above atmospheric pressure. In forced expiration, such as coughing or sneezing, intrapleural pressure may rise to +8 kPa or more.

Structure of the lungs

The right lung has three lobes and the left lung has two lobes (Figure 2.9). The heart lies close to the left lung which has a cardiac notch. The conducting airways comprise of the trachea which bifurcates at the carina (T4/T5) into the two main bronchi which divide into smaller bronchi, eventually leading to the terminal bronchioles. The bifurcation of the trachea corresponds on the surface anatomy (see Figure 2.3) to the sternal angle or angle of Louis.

The trachea is a semi-rigid structure which leads from the oropharynx into the thoracic cavity. The trachea and main bronchi have U-shaped cartilage linked posteriorly by smooth muscle. The anterior and lateral walls of the trachea are supported by rings of cartilage, but the posterior wall does not have any cartilage and is therefore collapsible. Diseases of the cartilage, such as tracheobronchomalacia, can affect the entire tracheobronchial tree.

The right main bronchus is wider, shorter, and more vertical than the left main bronchus, so inhaled material is more likely to enter the right main bronchus. The left main bronchus is longer and leaves the carina at a more abrupt angle. The right lung is divided by the horizontal and oblique fissures into the upper, middle, and lower lobes. The left lung is divided into the upper and lower lobes by the oblique fissure. The vessels, nerves, and lymphatics enter the lungs on their medial surfaces at the hilum. Each lobe is divided into several wedge-shaped bronchopulmonary segments with their apices at the hilum and bases at the lung surface. Each bronchopulmonary segment has a bronchus, artery, and vein (Figure 2.10).

Each lung is lined by visceral pleura which is continuous with the parietal pleura, lining the chest wall, diaphragm, pericardium, and mediastinum. In health, the space between the parietal and visceral layer is very small with a few millilitres of pleural fluid. The right and left pleural cavities are separate and each extends as the costodiaphragmatic recess below the lungs. The parietal pleura is segmentally innervated by intercostal nerves and by the phrenic nerve (C3, C4, and C5), so pain from pleural inflammation is often referred to the chest wall or shoulder tip. The visceral pleura lacks sensory innervation.

The main bronchi divide into the three main lobar bronchi on the right (upper, middle, and lower) and into two lobar bronchi on the left (upper and lower). These lobar bronchi divide further into segmental bronchi (generations 3 and 4) which continue to divide further into 22