Lung ventilation and the physiology of breathing

Anna Barrow

Jaideep J Pandit

Abstract

This article summarizes the anatomical features of the lungs, airway and thorax pertinent to the physiology of breathing and discusses chemoreceptor detection mechanisms, brainstem centres and relays involved in the control of breathing. We will discuss lung mechanics including spirometry, lung compliance, airway resistance and the role of surfactant and consider how these can be affected by disease states. It is recommended to revise principles of arterial blood gas analysis in addition to this article.

Keywords Airway resistance; breathing; control of breathing; compliance; dead space; flow volume loops; perfusion; spirometry; surfactant; VA/VQ mismatching; ventilation

Anatomy of the lungs, airways and thoracic cage

Airways

The upper respiratory tract begins at the nose and runs down to the nasopharynx, oropharynx and the larynx. The function the conducting airways is to filter, warm and humidify inspired gases.

The lower respiratory tract begins with the trachea at the level of C6 and extends to T4 where it bifurcates into the right and left main bronchi, lobar bronchi, segmental bronchi and terminal and respiratory bronchioles leading to the alveolar ducts and sacs. The respiratory bronchioles, alveolar ducts and sacs are the sites of gas exchange.

Lungs and pleura

The right lung is divided into upper, middle and lower lobes by the oblique and horizontal fissures. The left lung is divided into upper and lower lobes by the oblique fissure. The lungs are separated from the thoracic cage by two thin layers: the visceral pleura that invests the lung and, in continuity with it, parietal pleura, which abuts the chest wall, diaphragm, mediastinum and pericardium. The pleura are lubricated with pleural fluid and the space between them is normally very small. The intercostal nerves innervate the parietal pleura but the visceral pleura has no sensory supply. The arterial supply of the main bronchi as far as the terminal bronchioles is the bronchial arteries. The respiratory bronchioles alveolar ducts and sacs are supplied by the pulmonary veins.

Anna Barrow *MBBS MA MRCP FRCA is an ST4 in Anaesthesia at Oxford University Hospitals, Oxford, UK. Conflict of interest: none.*

Jaideep J Pandit *MA BMBCH DPhil FRCA FFPMRCA is a Consultant Anaesthetist at Oxford University Hospitals, Oxford, UK and Professor and Fellow, St John's College, Oxford, UK. Conflict of interest: none.*

Ciliated columnar epithelium lines the airways as far as the respiratory bronchioles. Goblet cells secrete mucus, which provides a physical defence mechanism through the mucociliary escalator. Squamous epithelium lines the alveolar ducts and sacs. Type 1 pneumocytes are thin walled and form the site of gas exchange. The type 2 pneumocytes are important for two reasons: surfactant secretion, which reduces surface tension, and secondly they are stem cells thus can divide to repair areas of damaged lung tissue.

Thoracic cage

The thoracic cage is formed by the sternum, ribs, thoracic vertebrae and the diaphragm inferiorly. Of the twelve pairs of ribs, the first seven pairs articulate via costal cartilages with the sternum. Rib pairs eight, nine and ten articulate with the cartilage superiorly and the last two pairs ribs are 'floating' ribs. Intercostal spaces lie between the ribs and contain muscles, arteries, veins and nerves. The intercostal muscles are arranged such that the external intercostals run downwards and anteriorly, the internal intercostals downwards and posteriorly and the innermost intercostal layer is incomplete. The intercostal arteries, veins and nerves run on the underside of the rib, protected in a groove.

The diaphragm

The diaphragm divides the thorax and the abdomen. It comprises peripheral muscular areas: the left and right crus that arise from the upper two and three lumbar vertebrae, respectively. The costal part of the diaphragm attaches to ribs seven to twelve and their costal cartilages. The sternal part arises from the xiphisternum. The central part is tendinous and forms the medial and lateral arcuate ligaments which are condensations of the fascia investing quadratus lumborum and psoas major. The phrenic nerve (C3, 4, 5) supplies motor function of the diaphragm. The sensory supply is from lower intercostal nerves peripherally and from the phrenic nerve centrally. Openings in the diaphragm exist for the inferior vena cava and right phrenic nerve at T8; the oesophagus, left gastric artery and vein and the vagus nerves at T10; and the aorta, thoracic duct and azygous vein at T12.

Respiratory muscles

In order to generate a negative pressure gradient for inspiration, inspiratory muscles act to increase the volume of the thoracic cage. The diaphragm is the main inspiratory muscle during quiet breathing. When it contracts, it descends by approximately 2 cm; however, during exercise it can descend by 7 cm. This increases the vertical dimensions of the thoracic cavity. Contraction of the inspiratory muscles pulls the ribs upwards and outwards, increasing the anterior–posterior diameter of the thorax. During inspiration the action of the innermost intercostals prevents the in-drawing of the intercostal space. As the diaphragm contracts during inspiration, it increases the intra-abdominal pressure and abdomen and chest wall both move outwards during normal inspiration. The anteroposterior diameter of the chest is also increased by the action of the scalene muscles that raise the upper two ribs and move the sternum anteriorly. The sloping lower ribs rise in a bucket handle fashion which increases the transverse diameter of the thorax.

Please cite this article in press as: Barrow A, Pandit JJ, Lung ventilation and the physiology of breathing, Surgery (2017), http://dx.doi.org/10.1016/j.mpsur.2017.02.004

When work of breathing is increased, accessory muscles become important: the sternocleidomastoids, serratus anterior and pectoralis major (when the arms are fixed) assist chest expansion.

Expiration occurs largely by passive recoil of the chest wall and lungs during quiet breathing. During exercise, expiration is augmented by the action of rectus abdominis and the internal and external obliques, which assist the recoil of the diaphragm by raising intra-abdominal pressure.

Diaphragmatic paralysis leads to paradoxical movement (see-sawing) of the chest and abdominal walls, as the diaphragm is drawn up into the chest with negative intrathoracic pressure during inspiration.

Dead space

The conducting airways, above the terminal bronchioles, do not take part in gas exchange. They form the anatomical dead space and the volume of this (Vd) is approximately 150 ml.

Alveolar dead space is formed by alveoli that do not take part in gas exchange: for example due to reduced or absent perfusion in pulmonary embolism, or shunting of blood from areas of pneumonia.

The sum of anatomical and alveolar dead space is the physiological dead space (i.e. the volume of gas that has not taken part in gas exchange). Measurement of these proportions of the tidal volume can be achieved using the Bohr method, details of which are beyond the scope of this article.

The respiratory cycle

Intrapleural pressure

During inspiration, expansion of the chest wall increases the pressure gradient between the intrapleural space and the alveoli as the lungs are stretched. This creates a negative pressure gradient from the mouth and nose to the alveoli which draws in air. During quiet breathing, alveolar pressure rises towards the end of inspiration, whereas intrapleural pressure remains negative for the duration of the respiratory cycle. During forced inspiration, in obstructed breathing, the intrapleural pressure may reach -80 cmH_2O (normal <-10 cmH_2O). During forced expiration, the both intrapleural pressure and alveolar pressure rise. Intrapleural pressure may reach $+80$ cmH_2O in healthy individuals. Due to the lung's elastic recoil, the pressure within the alveolus exceeds that within the pleura. When airflow stops, at end inspiration, and end expiration, the alveolar pressure is 0.

Lung volumes

The volume inhaled and exhaled during a 'normal breath' is known as the tidal volume (TV) and is approximately 500 ml. After a normal tidal breath, the volume further inspired with additional effort, would be the inspiratory reserve volume (IRV). Similarly, the volume which could be further exhaled at the end of a normal tidal breath is the expiratory reserve volume (ERV). Maximal inspiration and expiration to the expiratory and inspiratory reserve volumes would give the vital capacity (VC). The volume remaining in the lungs after maximal expiration is the residual volume (RV). Functional residual capacity (FRC) is the sum of the inspiratory reserve volume and the residual volume. FRC is the volume left in the lungs at the end of a normal breath and it is determined by the elastic properties of the lungs and the chest wall. At FRC, outward recoil of the chest wall and inward recoil of the lungs are exactly balanced. Thus, in lung fibrosis, where the lungs are stiff with increased recoil, FRC will occur at a smaller volume. Conversely in emphysema where there is destruction of lung parenchyma and loss of elastic recoil, FRC occurs at a larger volume. FRC can also be increased due to air trapping and application of positive end-expiratory pressure (PEEP). Total lung capacity (TLC) is equal to the residual volume plus the vital capacity (Figure 1).

Lung mechanics

During inspiration, work must be done to overcome impedance from the lungs and chest wall, mainly due to frictional forces:

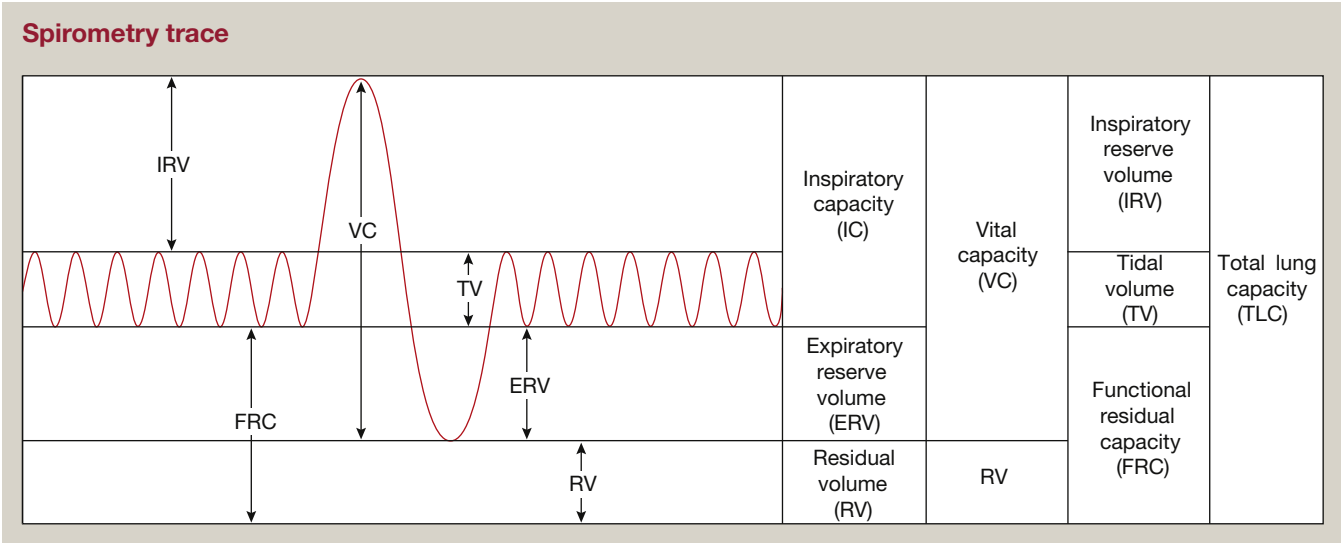

Figure 1 Lung volumes during tidal breathing, forced inspiration and forced expiration. Capacities are the sum of two or more lung volumes. Residual volume and functional residual capacity cannot be directly measured by spirometry and are calculated by helium dilution or body plethysmography.

Please cite this article in press as: Barrow A, Pandit JJ, Lung ventilation and the physiology of breathing, Surgery (2017), http://dx.doi.org/10.1016/j.mpsur.2017.02.004