THIRD EDITION

英文影印版

第3版

Textbook of Anaesthesia

麻醉学

Alan R.Aitkenhead
Greham Smith

科学出版社
Harcourt Asia
CHURCHILL LIVINGSTONE

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北京市版权局著作权登记号:01—1999—1542

图书在版编目(CIP)数据

麻醉学:第 3 版:英文/(英)艾肯海德(Ailkenhead, A. R.)等编著.
-3 版 .-北京:科学版社,1999. 9
ISBN 7-03-007725-3
Reprint ISBN 981-4033-04-09
Ⅰ. 麻… Ⅱ. 艾… Ⅲ. 麻醉学-英文 Ⅳ. R614

中国版本图书馆 CIP 数据核字(1999)第 27621 号

科学出版社 出版
Harcourt Asia 出版
CHURCHILL LIVINGSTONE
北京市东黄城根北街 16 号
邮政编码:100717
中国科学院印刷厂 印刷
新华书店北京发行所发行 各地新华书店经销
*
1999 年 9 月第 一 版 开本:787×1092 1/16
1999 年 9 月第一次印刷 印张:50
印数 1—3 000 字数:1 168 000
定价:98. 00
(如有印装质量问题,我社负责调换(环伟))

SCIENCE PRESS
16 Donghuangchenggen North Street,
Beijing 100717
China

HARCOURT ASIA PTE. LTD
A Harcourt Publishers International Company
583 Orchard Road # 09-01 Forum
Singapore 238884

Distribute in the Mainland China by Science Press,
16 Donghuangchenggen North Street, Beijing 100717, China.

Printed in China by HARCOURT ASIA PTE LTD and SCIENCE PRESS under special arrangement with Churchill Livingstone, A HARCOURT HEALTH SCIENCES COMPANY. This Edition is the only authorized complete and unabridged reproduction of the latest American Edition, published and priced for sale in China only, not including HongKong SAR and Taiwan.

This Edition First Printed in China in 1999.
ISBN 7-03-007725-3/R • 479
Reprint Edition ISBN 981-4033-04-9

Printed in China

Textbook of Anaesthesia

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Preface

The first edition of this book was intended to satisfy the needs of the new recruit into anaesthesia during the first 1–2 years in the specialty. In addition, it was hoped that it might provide suitable reading for anaesthetists studying for the (then) new Part 1 FFARCS examinations (later Part 1 FRCA), the European Diploma of Anaesthesiology, or equivalent examinations in other parts of the world. The response to the first edition was very encouraging, and it clearly proved useful not only to the intended audience but also to a wider readership including medical practitioners in rural areas or underdeveloped countries, and non-medical staff involved full-time in anaesthesia.

The success of the first edition stimulated us to produce a second edition, in which we undertook major revisions of several chapters, and introduced new chapters dealing with basic sciences and some additional clinical fields encountered frequently by trainee anaesthetists. The second edition also proved to be very popular, and this has prompted us to compile a third edition.

In this edition, we have invited new authors to contribute approximately one-third of the chapters in the book. This is not a reflection of the quality of the contributions of the previous authors, to whom we are very grateful; our intentions are simply to ensure that fresh minds are applied to the subject matter, and to avoid the risks which can be associated with asking authors merely to update their contributions. In future editions, it is our aim to pursue a similar policy with the remaining chapters. However, we are grateful to the authors of these chapters for the quality of the revisions which they have made in this edition. We are also indebted to the many reviewers and readers of the book who have provided helpful comments, which we have tried to address.

The publication of this edition coincides with the introduction of the new Primary FRCA examination. This book is not intended to provide comprehensive coverage of the syllabus of that examination, although, like the syllabus, it does embrace principles of physiology and pharmacology as well as clinical anaesthesia.

The astute observer will note that we have omitted the chapter on anatomy which opened the first two editions. Whilst useful, this chapter was not sufficiently comprehensive and we felt that its inclusion might undervalue the importance of this subject, especially to trainees.

As with previous editions, we are grateful to all of our contributors for allowing us to undertake widespread revision of manuscripts in an attempt to obtain uniformity of style. We are indebted again to our publishers, Churchill Livingstone, who have allowed extensive revisions of the proofs in order to accommodate very recent advances, particularly in relation to newly introduced drugs. Our gratitude must be recorded to Mrs Alice Whyte in Nottingham and Mrs Karen Marden in Leicester for substantial secretarial work.

We hope that this text will prove as popular as the first two editions, and will be used by trainees as a practical guide in the operating theatre and as the foundation of their theoretical training. It may be valuable also as an 'aide memoire' for teachers in anaesthesia, and may be appropriate reading for undergraduates who undertake an elective period of training in anaesthesia, for anaesthetic and recovery rooms nurses, for operating department practitioners and for nurse anaesthetists.

Nottingham and
Leicester, 1996

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Contents

1. Respiratory physiology

The principal purpose of the respiratory system is the exchange of oxygen and carbon dioxide between the blood and the respired gas. It has secondary roles in the control of acid base balance, the metabolism of hormones and the removal of compounds and particulate matter, taking advantage of its position as the only organ which receives the entire cardiac output.

Breathing is the most obvious attribute of the respiratory system and the sequence of events in a normal breath in the upright posture will be outlined as a basis for further consideration.

A BREATH

Initiation of a breath starts in the inspiratory neurones of the respiratory centre in the floor of the fourth ventricle. As expiration ends, increasing neuronal traffic develops in the descending motor neurones of the lateral and ventral columns which synapse with the anterior horn cells of the nerves supplying the respiratory muscles. As the muscles start to contract, muscle spindles sense the load and adjust anterior horn cell activity to achieve the required force.

The diaphragm is the main muscle of respiration and contraction of the inverted J shaped fibres causes it to descend with a consequent decrease in intrapleural pressure. Simultaneously, the dilator muscles of the upper airway (alae nasi, tensor palatini, palatoglossus, myoglossus, posterior cricoarytenoid) constrict, opening the airway and resisting the developing subatmospheric collapsing force. The strap muscles and the intercostal muscles also contract, stabilizing the upper chest, preventing it from being indrawn and aiding the expansion of the lower rib cage by the 'bucket handle' movement of the ribs.

The increasing subatmospheric intrapleural pressure expands the lung and dilates the intrathoracic airways. Air is drawn through the nose, where it is warmed and humidified, through the pharynx, larynx, trachea and bronchi until it reaches the terminal bronchioles. The increase in total cross-sectional area of the airways is so great at this point that little further mass movement of gas occurs and transfer of gas to and from the alveoli is by diffusion. The distance is less than 5 mm and takes less than 1 s to reach equilibrium.

The inspired air is not distributed evenly around the lung but is directed preferentially to those areas which are best perfused, the dependent areas of the lung. Final matching of blood flow and gas exchange is achieved by the mechanism of hypoxic pulmonary vasoconstriction (HPV).

Oxygen diffuses from the terminal bronchioles, through the respiratory bronchioles and alveolar sacs into the alveoli. It then diffuses across the alveolar epithelium, basement membranes, capillary endothelium, plasma and red cell membrane before combining with haemoglobin. Carbon dioxide diffuses in the reverse direction.

As inspiration proceeds, increasing afferent neuronal traffic from stretch receptors in the lungs, rib cage and muscles, coupled with increasing feedback from the inspiratory neurones themselves, ultimately inhibits the inspiratory neurones so that inspiration ceases.

Expiration then generally proceeds passively with the stored elastic energy in the lung and chest wall providing the force to overcome the resistance to airflow through the bronchial tree and upper

airway. As lung volume decreases to the functional residual capacity (FRC), activity in the expiratory neurones decreases and increases in the inspiratory neurones, heralding the start of the next breath.

CONTROL OF RESPIRATION

Respiration is regulated by the respiratory neurones (often known as the respiratory centre) to maintain homoeostasis. Arterial carbon dioxide tension (Pa_{CO_2}) is regulated at about 5.3 kPa (40 mmHg) and thus under normal circumstances the main determinant of the minute ventilation ($\dot{V}$) is the production of carbon dioxide ($\dot{V}_{CO_2}$) which in turn is determined by the metabolic activity of the body and the energy source. Ventilation is greater on a carbohydrate based diet (respiratory quotient (RQ) = 1.0) than a fat based diet (RQ = 0.7) as the energy produced per unit of CO_2 evolved is greater with the latter.

Respiration is modified by many other factors, particularly from higher centres in the brain including the cortex. The pattern of respiration is modulated by speech and ingestion of food and drink. The anticipation of exercise as well as the activity itself increases respiration. The respiratory centre also balances the depth of respiration (tidal volume (V_t)) against the rate so that the least energy is spent on breathing ($\dot{V}_{O_2\,resp}$). Increases in the elastic work of breathing (e.g. pulmonary oedema or fibrosis) tend to increase the respiratory rate whereas increases in the resistive work (e.g. asthma) tend to increase V_t.

Respiration is influenced also by the Pa_{CO_2}, the arterial pH and the Pa_{O_2}, the former two via the central chemoreceptors and the latter via the peripheral chemoreceptors.

Central control

The central chemoreceptors lie in the floor of the fourth ventricle and are either the neurones responsible for generation of the respiratory rhythm or are closely related to them. The cells are responsive to changes in the interstitial fluid pH and their sensitivity to changes in Pa_{CO_2} is in part due to the poor buffering of cerebrospinal fluid (CSF) compared with blood. The response is very rapid and injection of blood with an increased Pa_{CO_2} into the carotid artery of an experimental animal during inspiration results in an augmentation of that breath. The change in $\dot{V}$ with Pa_{CO_2} is approximately linear up to a Pa_{CO_2} of about 12 kPa (90 mmHg) and averages about 15 litre.min^{-1}.kPa^{-1} (Fig. 1.1).

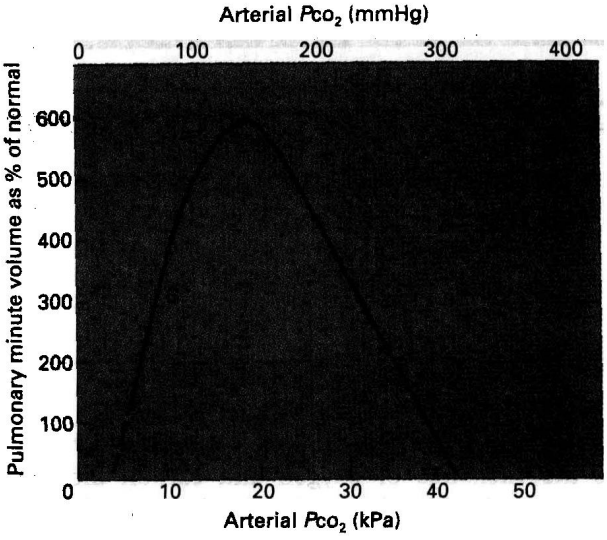

Fig. 1.1 The complete ventilatory response to carbon dioxide. Only the straight portion of the ascending limb has been determined in man and the slope, s, is used to quantify the response.

Peripheral chemoreceptors

The peripheral chemoreceptors are located in the carotid and aortic bodies. They are best regarded as sensors of oxygen delivery as they respond to both a decrease in Pa_{O_2} and in blood flow rate. The carotid bodies effectively monitor the oxygen supply to the brain, the organ most easily damaged by hypoxaemia. The mechanism is probably similar to the central chemoreceptors in that the sensor cells respond to changes in pH. The ventilatory response to hypoxaemia is shown in Figure 1.2 and, if the Pa_{CO_2} is kept constant, changes exponentially with Pa_{O_2}. The response is linear when oxygenation is expressed as oxyhaemoglobin saturation (Sa_{O_2}). The response is much greater if Pa_{CO_2} increases at the same time.

Respiratory reflexes

Cough

A cough is one means of removing unwanted

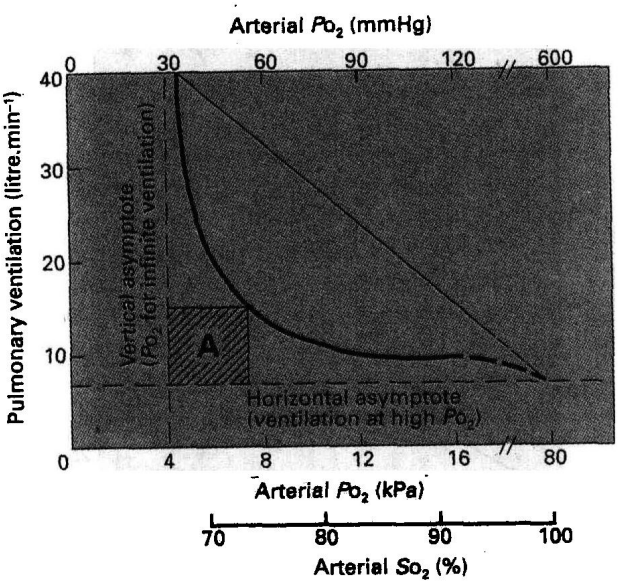

Fig. 1.2 The ventilatory response to hypoxaemia, expressed as Pa_{O_2} (heavy line) and as Sa_{O_2} (light line).

material from the respiratory tract. It complements the mucociliary escalator which clears small particulate matter. A cough may be induced voluntarily but is normally spontaneous from stimulation of receptors in the airways. It comprises a maximal inspiration followed by a forced expiration against a closed glottis, when intrathoracic pressures may reach 80 cmH_2O. The larynx then opens allowing expiration to occur at maximum velocity. The increased intrathoracic pressure causes dynamic compression of the bronchi, thus further increasing the velocity of expired air, often approaching the speed of sound and creating shear forces which detach the mucus from the mucosa. A wave of dynamic compression sweeps from the smaller to the larger bronchi as the cough progresses.

An effective cough thus requires three elements: an adequate inspired volume, adequate expiratory power and a functioning glottis. Absence of any of these elements leads to impaired coughing and retention of secretions.

Laryngospasm

Laryngospasm is, phylogenetically, a very primitive reflex and is intended to protect the lungs from inhalation of noxious substances. It is induced by stimulation of both chemical and touch receptors above and below the glottis. The reflex is less vigorous in the elderly.

Arousal

The ability to arouse from sedation or sleep in response to apnoea, airway obstruction or the need to cough is an important respiratory response. It is obtunded during normal sleep and by sedative and analgesic drugs such as morphine and may be a major contributory factor in postoperative respiratory complications.

MECHANICS OF RESPIRATION

The respiratory system may be regarded as a collapsible elastic sac (the lungs) surrounded by a semirigid cage (the thorax) with a piston at one end (the diaphragm) supplied through a branching set of semirigid tubes (the airway and bronchial tree). The volume of the system at rest is a balance between the tendency of the lungs to collapse, the thorax to expand and the position of the diaphragm.

Lung volumes

The total volume of the respiratory system (total lung capacity (TLC)) when fully expanded by voluntary effort is about 3–6 litres in the average adult and is related more to height than weight. It can be divided into the parts that participate in gas exchange (alveolar volume) and those that do not (dead space). The alveolar volume may be divided also into that which can be measured at the lips (vital capacity (VC)) and that which remains in the lung after a maximal expiration (residual volume (RV)) (Fig. 1.3). These volumes change little with body position, unlike the volume left in the lungs after a normal expiration (functional residual capacity (FRC)). The FRC is influenced by body position, being greatest in the upright position and least when lying head downwards, the changes being mostly due to movement of the diaphragm. The closing capacity (CC) is that volume of the lung where small airways in the dependent parts of the lung begin to collapse during expiration. Normally CC is less than FRC

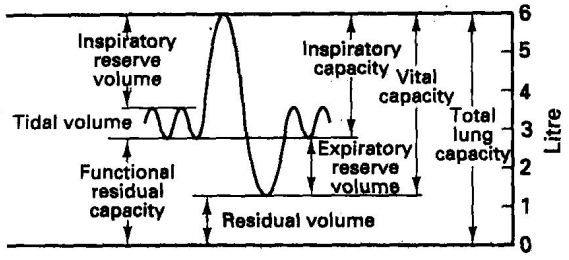

Fig. 1.3 The static lung volumes in a normal 70 kg adult male.

Table 1.1 Factors influencing the functional residual capacity (FRC).

Factors decreasing FRC
Increasing age
Posture – supine
Anaesthesia – intraoperative
Abdominal and thoracic surgery – postoperative
Pulmonary fibrosis
Pulmonary oedema
Obesity
Abdominal swelling – pregnancy, tumour, ascites
Thoracic cage distortion
Reduced muscle tone
Factors increasing FRC
Increased intrathoracic pressure – PEEP, CPAP
Emphysema
Asthma

but greater than RV. This can be demonstrated by expiration to RV which is inevitably followed by a sigh to re-expand collapsed lung. CC increases with age and FRC is decreased by a number of factors (Table 1.1) and if CC is greater than FRC, dependent parts of the lung collapse during normal tidal breathing resulting in hypoxaemia.

The volumes described above are obtained by slow breathing so that airway resistance is not important. For clinical evaluation of patients, dynamic lung volumes are more useful such as the forced vital capacity (FVC) and the forced expiratory volume in the first second (FEV_1). The limiting factor in a forced expiration is the dynamic compression of the intrathoracic airways by the raised intrathoracic pressure.

Compliance

Both the lungs and the chest wall require a distending force, usually expressed as volume change

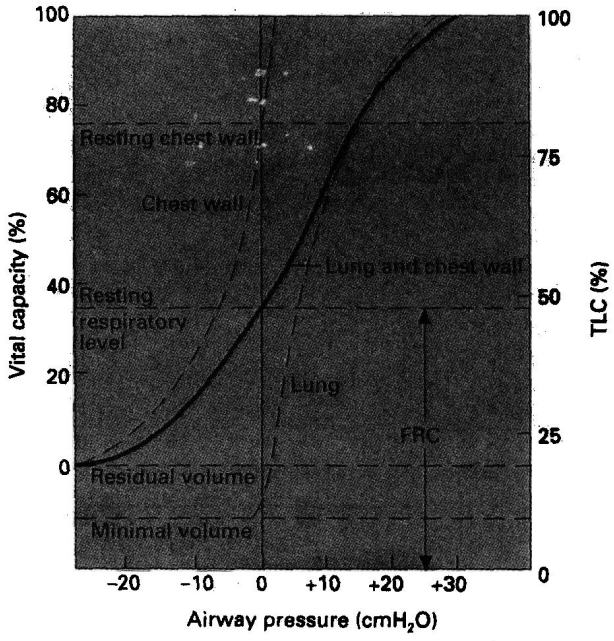

Fig. 1.4 The compliance curves of the lungs, chest wall and total respiratory system. The latter is obtained by adding the individual curves. The FRC is a balance between the lung and chest wall.

per unit of distending pressure (compliance) ($ml.cmH_2O^{-1}$) and are both approximately 200 $ml.cmH_2O^{-1}$. The compliance of the whole respiratory system (100 $ml.cmH_2O^{-1}$) is clearly less than the individual components and is derived by adding the reciprocals (1/200 + 1/200). The compliance curve of the lung is shown in Figure 1.4. The compliance is approximately linear over most of the range but is less when the lung is small and nearly fully inflated. The former is due to the added force needed to expand collapsed areas of the lung and overcome surface tension effects and the latter is due to the elastic fibres in the lung reaching their limit.

The lung exhibits hysteresis, i.e. the compliance differs during inflation and deflation. This is due to the effect of alveolar surfactant and is absent if the lung is inflated with a fluid.

Surfactant

If the alveoli are regarded as a series of interconnected bubbles then the normal surface tension effects would ensure that the smaller bubbles

emptied into the larger. However, the presence of a surface active material, dipalmitoyl lecithin, secreted by the type II alveolar cells, ensures that this does not occur. As the alveolus decreases in size, the concentration of surfactant in the surface layer of fluid increases, thus effectively reducing the surface tension. Collapsed lung and small airways have no air–liquid interface and thus additional force is required to open those areas.

Resistance

The flow of air into and out of the lungs is opposed by the frictional resistance of the airways and to a lesser extent by the inertia of the gas. The type of flow is important, with laminar flow offering less resistance than transitional or turbulent flow. Laminar flow occurs at low flow rates and in the smaller bronchi. In the larger airways and at branches in the bronchial tree, transitional and turbulent flow may occur.

Laminar flow rate ($\dot{V}$) is related to driving pressure (δP) by Poiseuille's equation:

$$\dot{V} = \frac{\delta P \pi r^4}{8 \eta L}$$

where r is the radius of the tube, L its length and η the viscosity of the gas. Note that the radius of the tube is critical, a halving of the tube diameter reducing the flow by a factor of 16 for the same δP, an important factor in paediatric practice.

Airway resistance is related to lung volume, decreasing as the lung expands. It is also related to bronchomotor tone and the thickness of the mucosal layer.

MATCHING OF VENTILATION AND PERFUSION

As noted earlier, the purpose of the lungs is to exchange gases by bringing the inspired gas into contact with pulmonary capillary blood. Under normal circumstances, the distributions of ventilation and blood flow are nearly perfectly matched. The main determinant of the distribution of perfusion is gravity which is not under bodily control and thus the changes necessary to ensure matching with changes in posture predominantly occur in the distribution of ventilation.

Distribution of perfusion

The distribution of blood flow within the lungs is largely influenced by gravity with the dependent portions of the lung being best perfused. In the erect posture three distinct zones may be described (Fig. 1.5). In the upper zone, alveolar pressure exceeds both pulmonary arterial and venous pressures and there is no flow. This zone does not occur under normal conditions but may occur in the presence of hypovolaemia and with increased alveolar pressure. In the middle zone alveolar pressure is exceeded by pulmonary artery pressure but is greater than pulmonary venous pressure. In the lower zone, both pulmonary arterial and venous pressures exceed alveolar pressure. In both these latter zones flow increases in the more dependent areas.

Within the substance of the lung, the pulmonary arteries divide and subdivide following the lobar pattern of the bronchi. The pulmonary capillaries form a dense network around the alveoli. The nuclei of the endothelial cells and supportive collagen fibres are arranged so that they are on the opposite side to the alveoli and gas diffusion occurs through the thinned service wall which comprises just the flattened epithelial and endothelial cells and their fused basement membranes (Fig. 1.6). A red cell traverses two or three alveoli in its passage through the lung.

Diffusion of CO_2 and oxygen is very rapid and under normal circumstances saturation of the

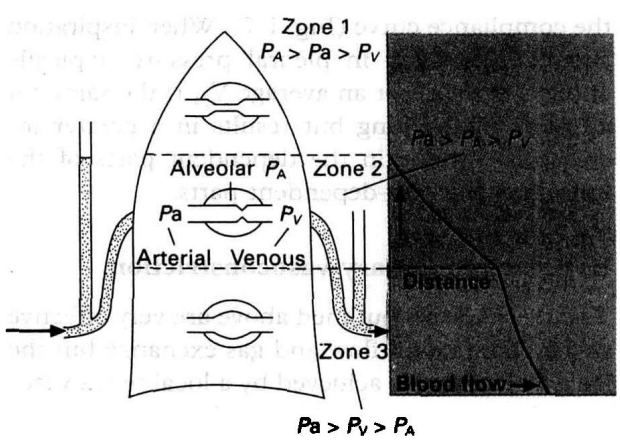

Fig. 1.5 Pressure flow relationships in different parts of the lung in the erect posture. The three zones are described in the text. (Redrawn from West et al 1964.)

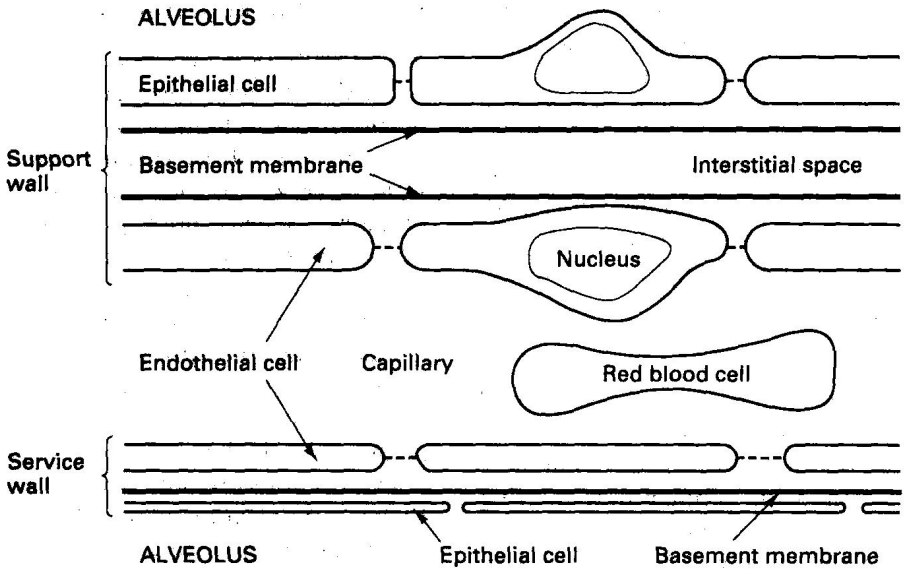

Fig. 1.6 Cross-section of the alveolar wall.

haemoglobin with oxygen is complete before the red cell is halfway through its journey.

Distribution of ventilation

Several factors ensure that the inspired gas is directed towards the dependent parts of the lungs. The major factor is the compliance of the different parts of the lung. A pressure gradient exists from the top to the bottom of the pleural space due to the weight of the lung such that it is less negative at the base compared with the apex. The different parts of the lung are thus on different parts of the compliance curve (Fig. 1.7). When inspiration occurs the *change* in pleural pressure, typically about 5 cmH_2O for an average V_t, is the same for all parts of the lung but results in a greater increase in volume in the dependent parts of the lung than the non-dependent parts.

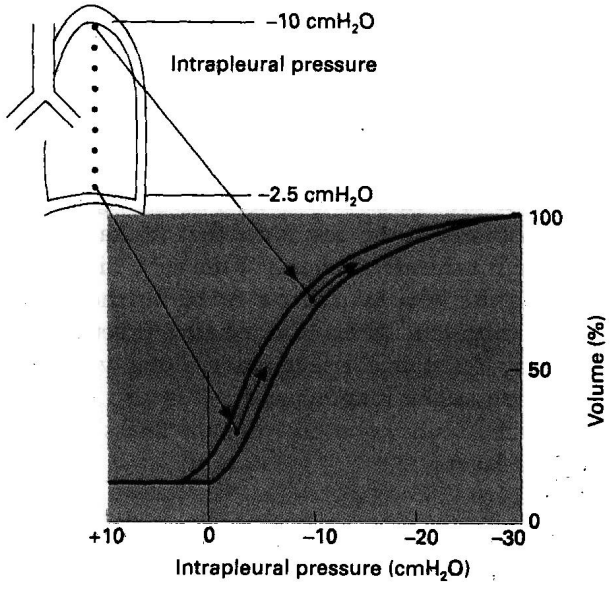

Fig. 1.7 Regional differences in the pressure volume curve of the lung. The same change in pressure causes a greater change in volume at the base of the lung compared with the apex.

Hypoxic pulmonary vasoconstriction

The mechanisms outlined above are very effective at matching blood flow and gas exchange but the local fine tuning is achieved by a local reflex vasoconstriction in the supplying pulmonary artery in response to alveolar hypoxaemia. This mechanism is of minor importance in the normal lung but very important in the presence of disease such as pneumonia or during one-lung anaesthesia for thoracic surgery.

Dead space, $\dot{V}/\dot{Q}$ matching and shunt

In ideally ventilated and perfused lung the ratio