Respiratory system

Points

- Ventilation
- Diffusion
- Perfusion

Function

- The main role of the respiratory system is to work closely with the heart and blood to extract oxygen from the external environment and dispose of waste gases, principally carbon dioxide.
- This requires the lungs to function as an efficient bellows, expelling used air, bringing fresh air in and mixing it efficiently with the air remaining in the lungs.
- The lungs have to provide a large surface area for gas exchange and the alveoli walls have to present minimal resistance to gas diffusion. This means the lungs have to present a large area to the environment and this can be damaged by dusts, gases and infective agents.
- Host defence is therefore a key priority for the lung and is achieved by a combination of structural and immunological defences.

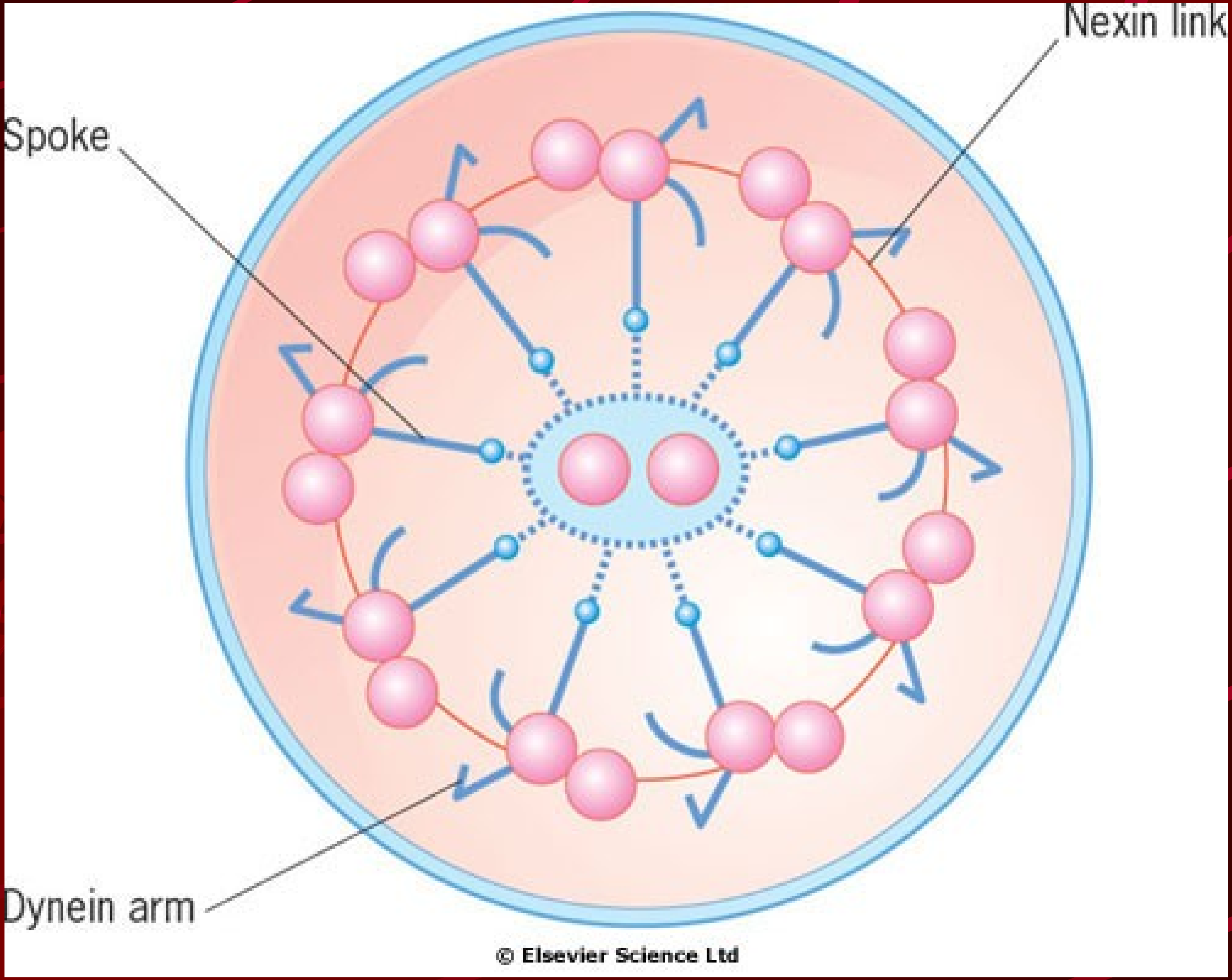
Of the first seven divisions, the bronchi have:

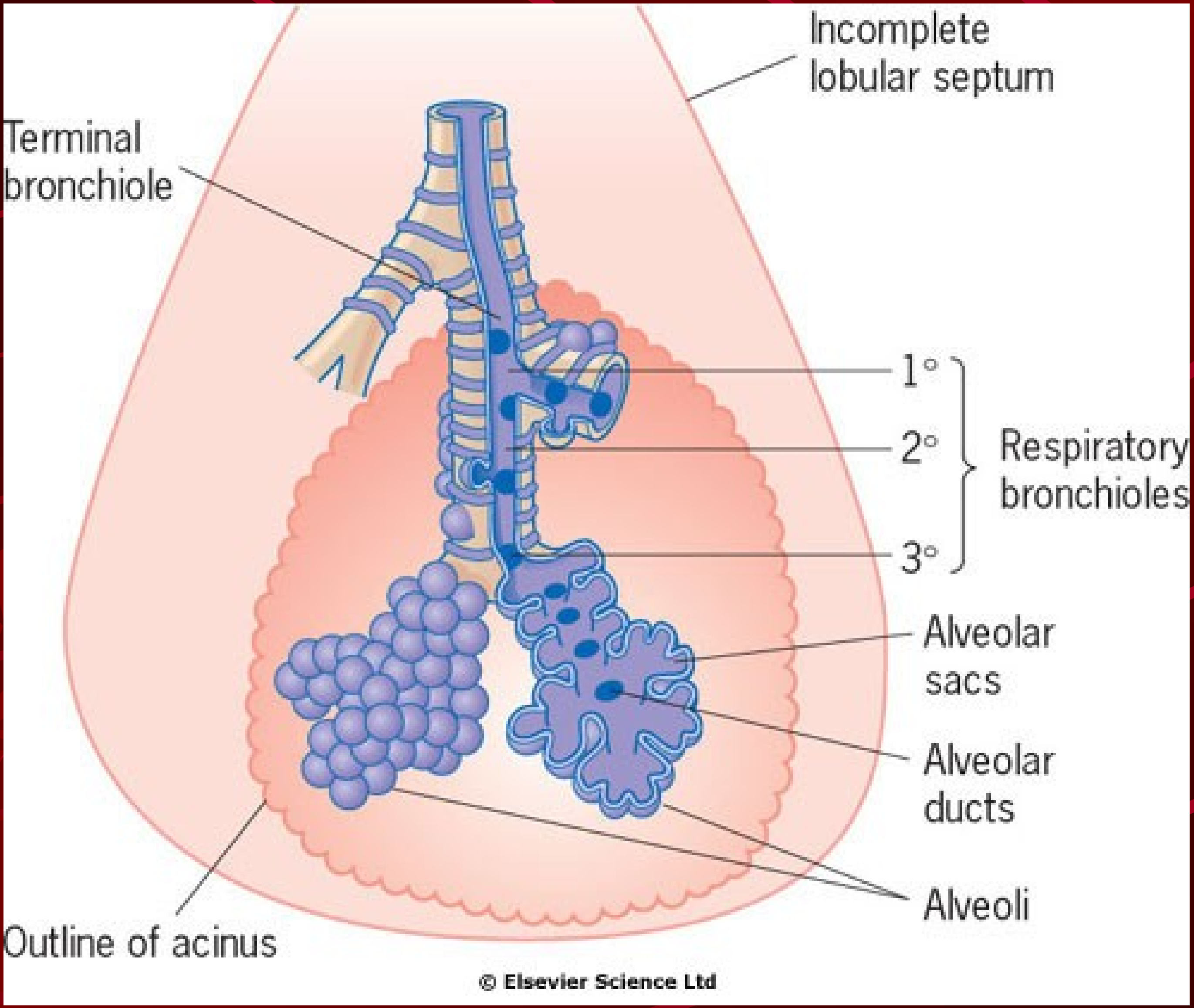
- walls consisting of cartilage and smooth muscle
- epithelial lining with cilia and goblet cells
- submucosal mucus-secreting glands
- endocrine cells - Kulchitsky or APUD (amine precursor and uptake decarboxylation) containing 5-hydroxytryptamine

In the next 16-18 divisions the bronchioles have:

- no cartilage and a muscular layer that progressively becomes thinner
- a single layer of ciliated cells but very few goblet cells
- granulated Clara cells that produce a surfactant-like substance.

The ciliated epithelium




Incomplete
lobular septum
Terminal
bronchiole
1°
2°
3°
Respiratory
bronchioles
Alveolar
sacs
Alveolar
ducts
Alveoli
Outline of acinus
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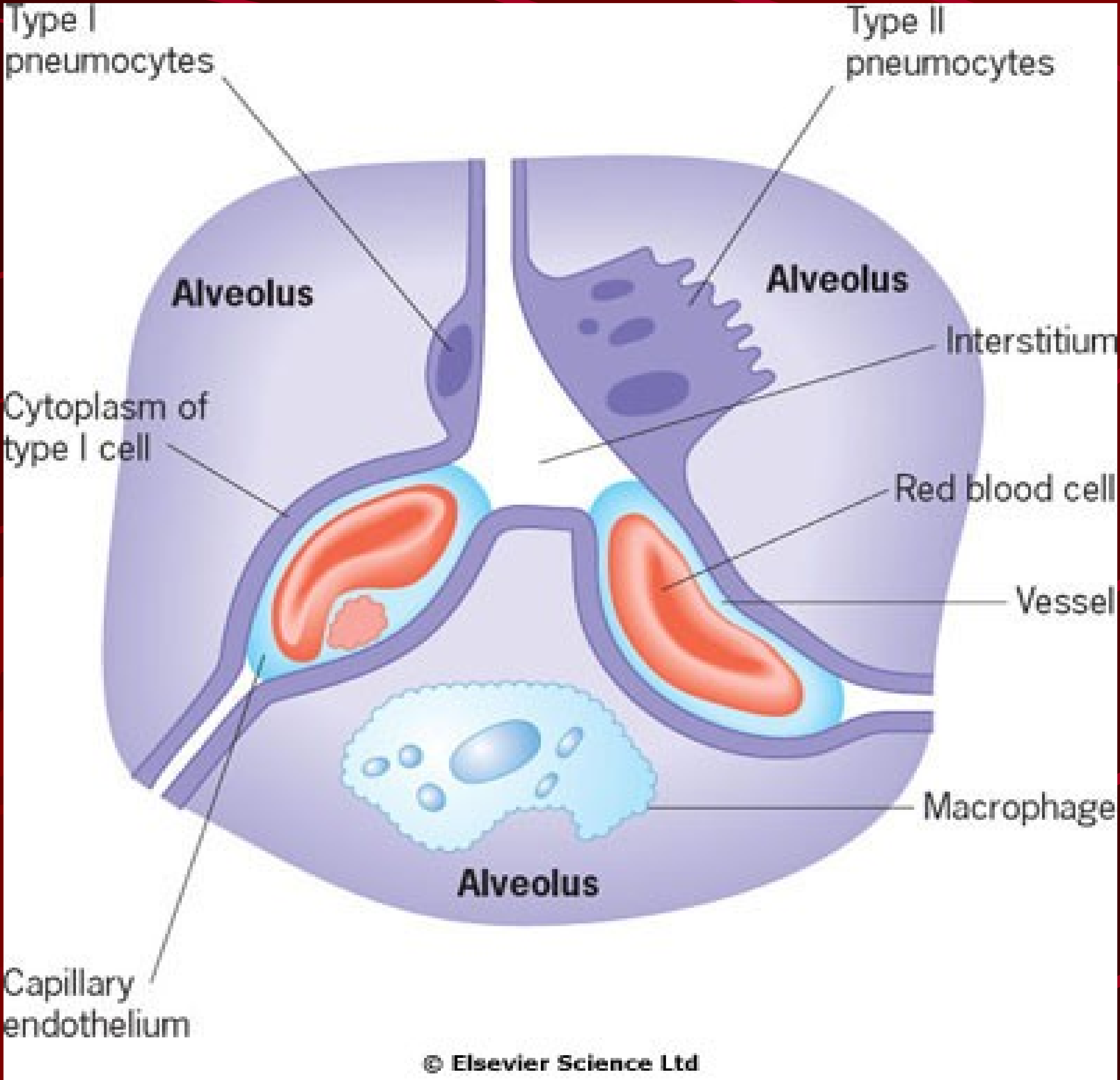
Type I
pneumocytes
Type II
pneumocytes
Alveolus
Alveolus
Interstitium
Cytoplasm of
type I cell
Red blood cell
Vessel
Macrophage
Alveolus
Capillary
endothelium
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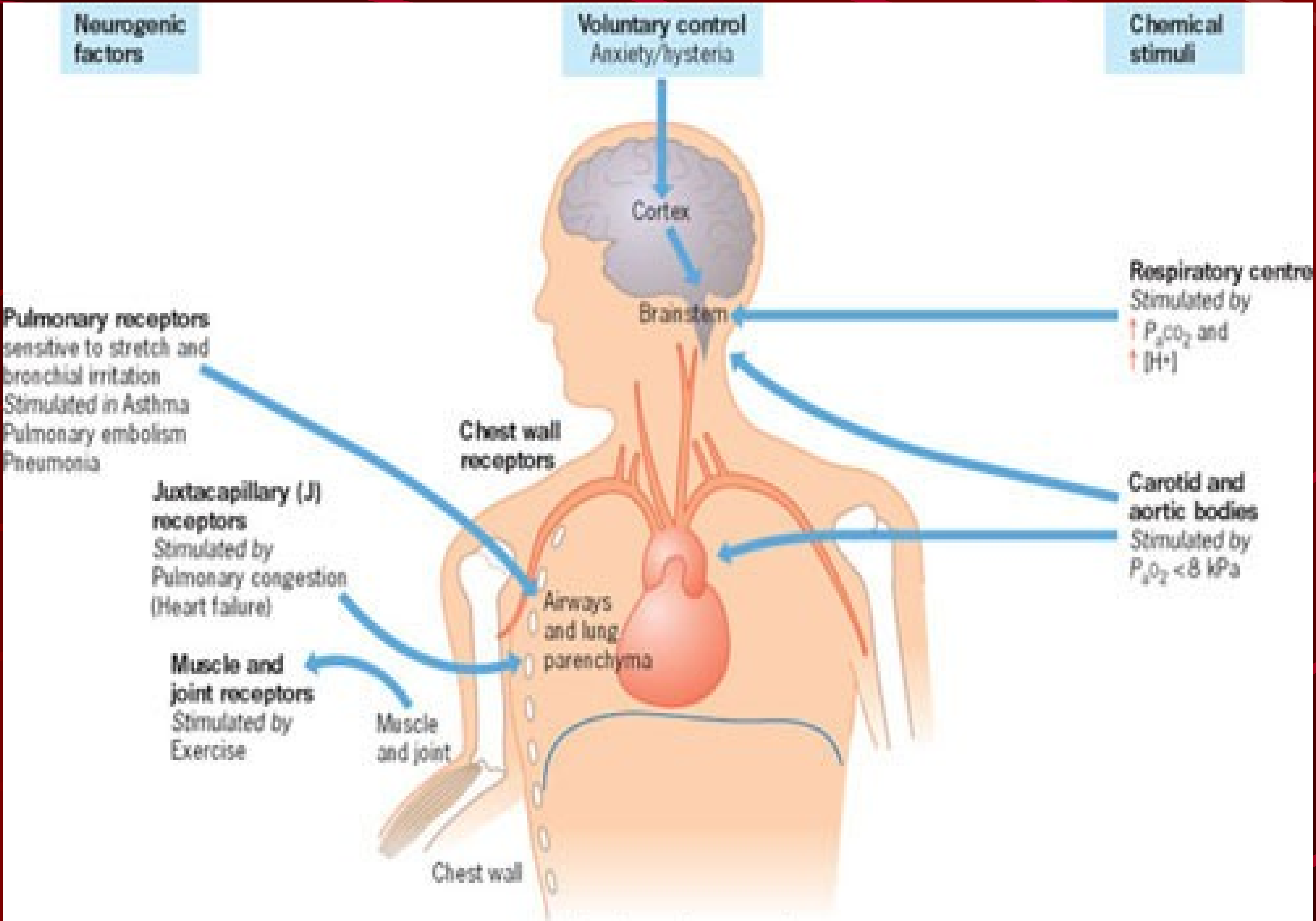
Breathing

- Lung ventilation can be considered in two parts:
- the mechanical process of inspiration and expiration
- the control of respiration to a level appropriate for the metabolic needs.



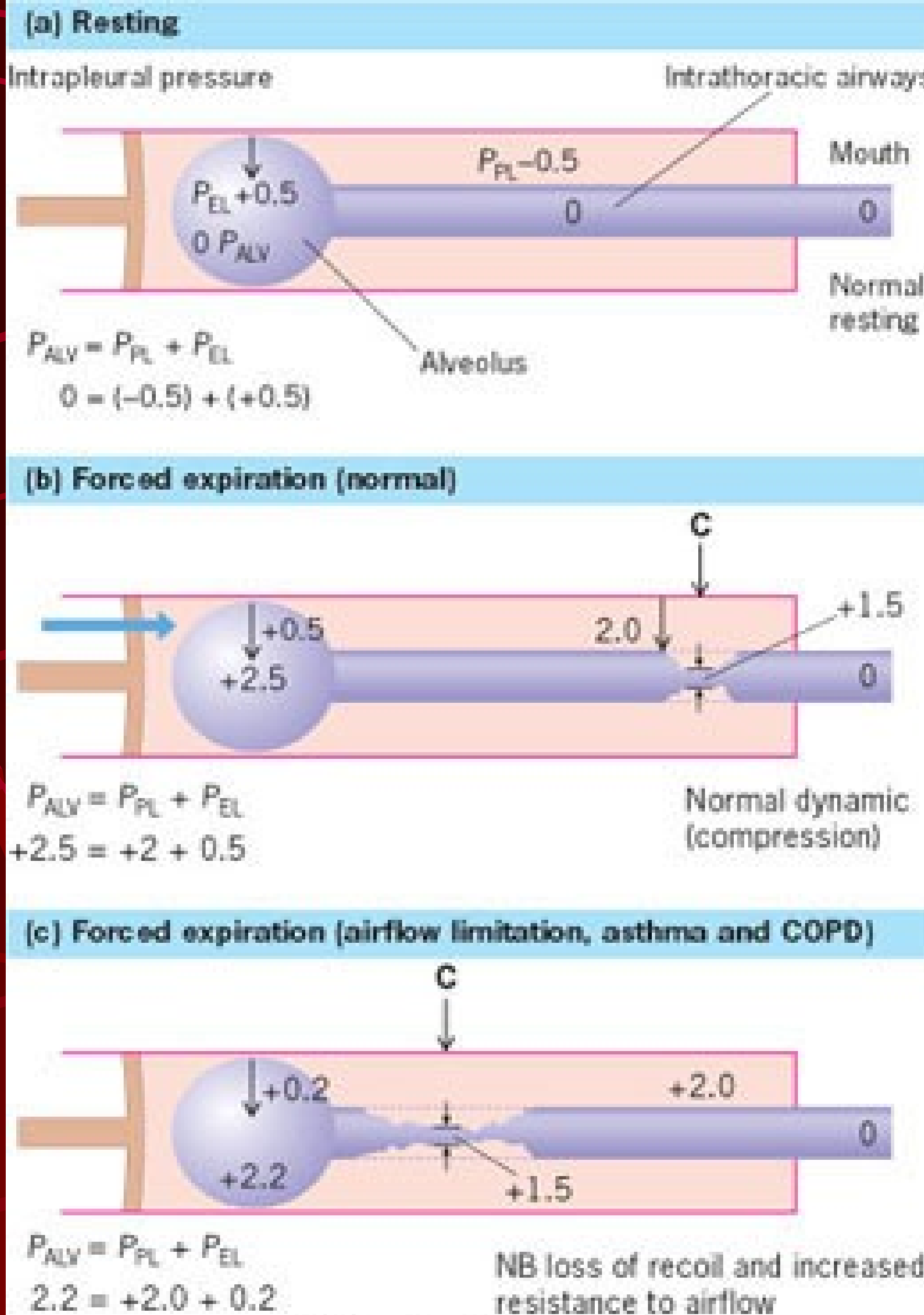
Neurogenic factors
Voluntary control
Anxiety/hysteria
Chemical stimuli
Cortex
Brainstem
Respiratory centre
Stimulated by
↑ P_aco_2 and
↑ [H+]
Pulmonary receptors
sensitive to stretch and bronchial irritation
Stimulated in Asthma
Pulmonary embolism
Pneumonia
Chest wall receptors
Juxtacapillary (J) receptors
Stimulated by
Pulmonary congestion
(Heart failure)
Carotid and aortic bodies
Stimulated by
P_ao_2 < 8 kPa
Airways and lung parenchyma
Muscle and joint receptors
Stimulated by
Exercise
Muscle and joint
Chest wall

Airway resistance

- From the trachea to the periphery, the airways become smaller in size (although greater in number). The cross-sectional area available for airflow increases as the total number of airways increases. The flow of air is greatest in the trachea and slows progressively towards the periphery (as the velocity of airflow depends on the ratio of flow to cross-sectional area). In the terminal airways, gas flow occurs solely by diffusion.
- The resistance to airflow is very low (0.1-0.2 kPa/L in a normal tracheobronchial tree), steadily increasing from the small to the large airways. Airways expand as lung volume is increased, and at full inspiration (*total lung capacity*, TLC) they are 30-40% larger in calibre than at full expiration (*residual volume*, RV). In chronic obstructive pulmonary disease (COPD) the small airways are narrowed and this can be partially compensated by breathing at a larger lung volume.

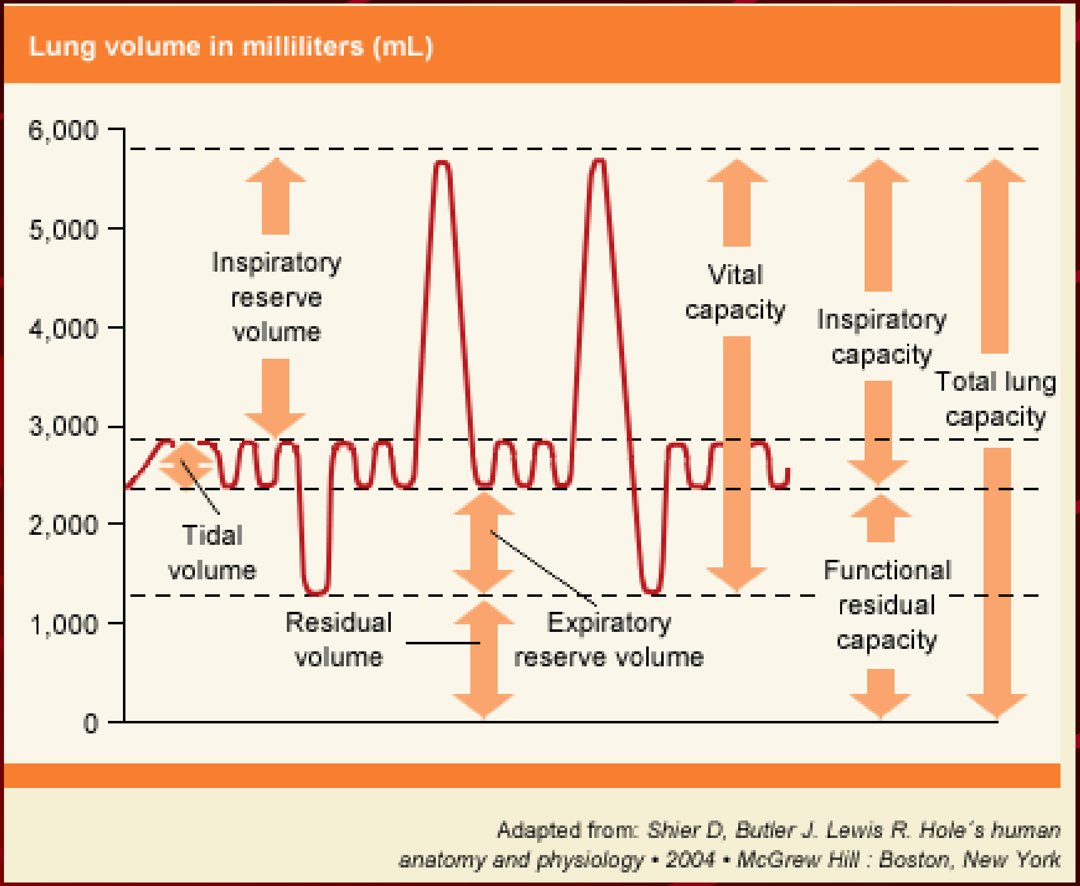
Control of airway tone

- This is under the control of the autonomic nervous system. Bronchomotor tone is maintained by vagal efferent nerves. Many adrenoceptors on the surface of bronchial muscles respond to circulating catecholamines; sympathetic nerves do not directly innervate them.
- Airway tone shows a *circadian rhythm*, which is greatest at 04.00 and lowest in the mid-afternoon. Tone can be increased briefly by inhaled stimuli acting on epithelial nerve endings, which trigger reflex bronchoconstriction via the vagus. These stimuli include cigarette smoke, inert dust and cold air; airway responsiveness to these increases following respiratory tract infections even in healthy subjects. In asthma, the airways are very irritable and as the circadian rhythm remains the same, asthmatic symptoms are usually worst in the early morning.


(a) Resting
Intrapleural pressure
Intrathoracic airways
Mouth
P_{PL} −0.5
P_{EL} +0.5
0 P_{ALV}
0
0
Normal resting
Alveolus
$P_{ALV} = P_{PL} + P_{EL}$
0 = (−0.5) + (+0.5)
(b) Forced expiration (normal)
C
+0.5
+2.5
2.0
+1.5
0
$P_{ALV} = P_{PL} + P_{EL}$
+2.5 = +2 + 0.5
Normal dynamic (compression)
(c) Forced expiration (airflow limitation, asthma and COPD)
C
+0.2
+2.2
+2.0
0
+1.5
$P_{ALV} = P_{PL} + P_{EL}$
2.2 = +2.0 + 0.2
NB loss of recoil and increased resistance to airflow

Static spirometry

- parameters independent on time



Lung Volume Measurements

- Measured by dilutional techniques (helium dilution or nitrogen washout) or by displacement techniques in a body box
- The FRC is measured and then the other measurements are determined:
- TLC = inspiratory capacity + FRC
- RV= FRC – ERV (expiratory reserve volume)

Dynamic spirometry

- – Forced Vital Capacity – FVC
- – Forced Expiratory Volume in One Second - FEV1
- – **Peak respiratory flow** - PEF
- – Forced Expiratory Volume in One Second Expressed as a Percentage of the Forced Vital Capacity - FEV1/FVC %
- - **Mean Forced Expiratory Flow during the Middle Half of the Forced Vital Capacity**
  – FEF 25-75%

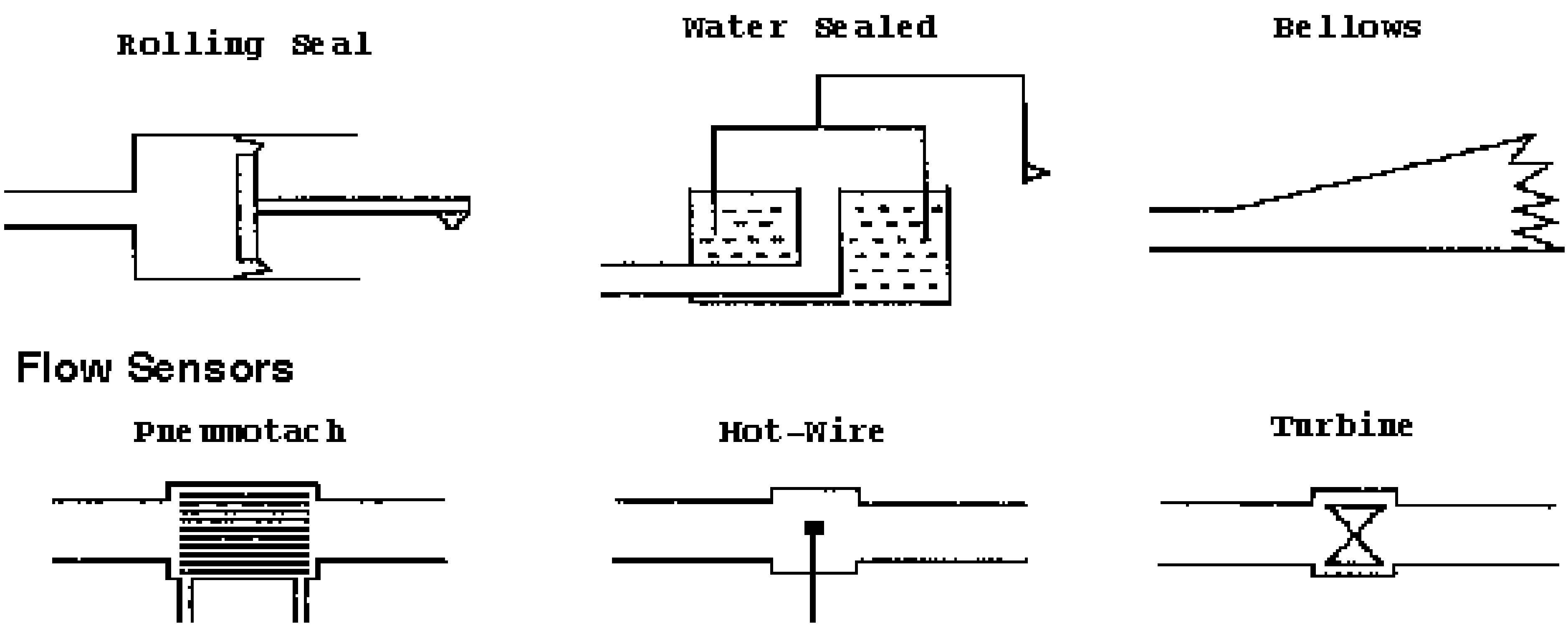
Ventilatory dysfunction

- **Obstructive**: airways collaps during exspiration, air-trapping phenomenon
- **Restrictive**: difficulties in inspiration, lung tissue scarring, infiltration or weak muscles, lung volumes decrease
- **Combined**

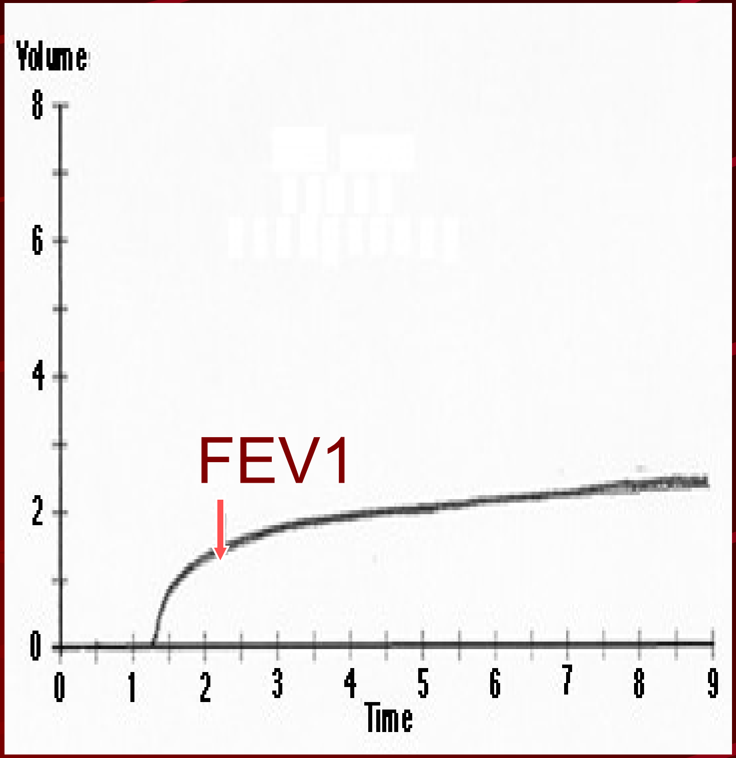
Lung Volume Patterns

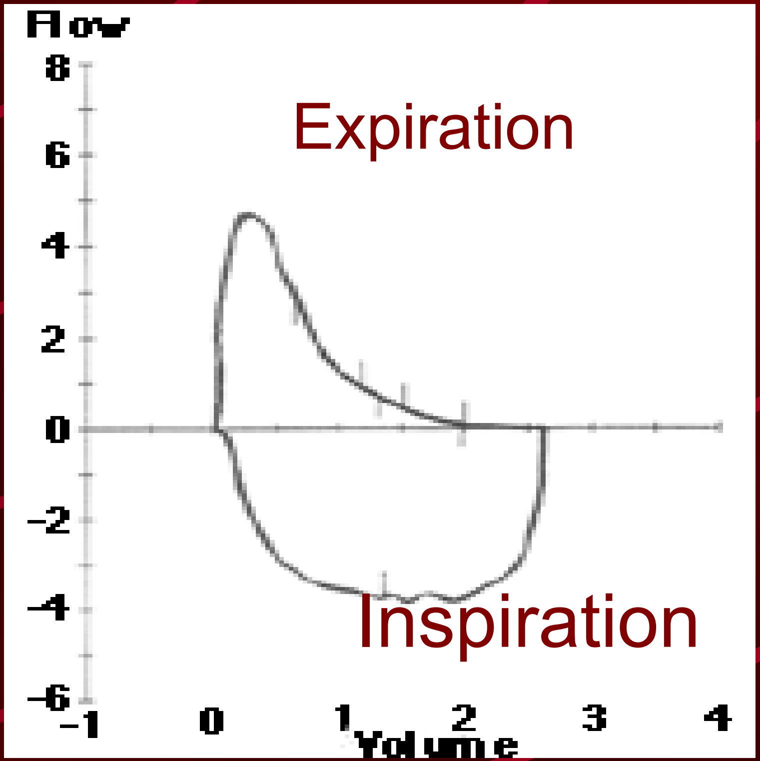
- Obstructive Disease: Characterized by hyperinflation and gas trapping (decreased PEF, FEV1/FVC, increased TLC and RV/TLC)
- Restrictive Disease: Characterized by generalized reduction in lung volume (decreased TLC, RV and FRC)

Types of spirometers



Graphs





Volume-time

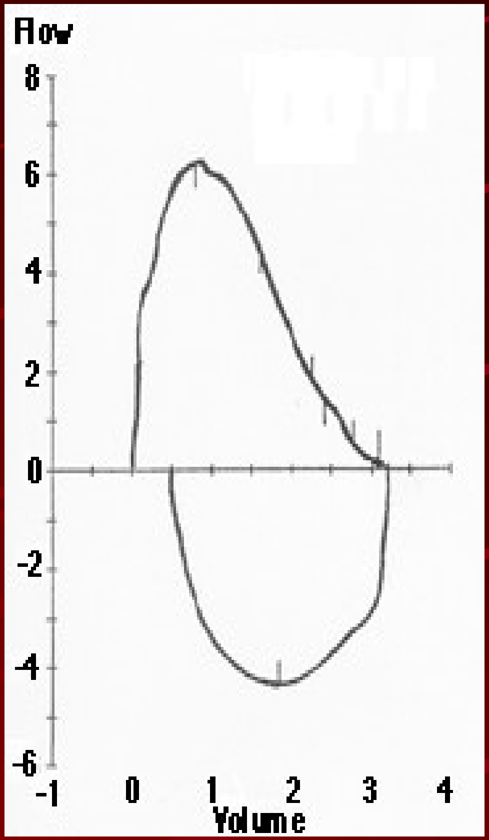
Flow-volume

Flow-volume loops

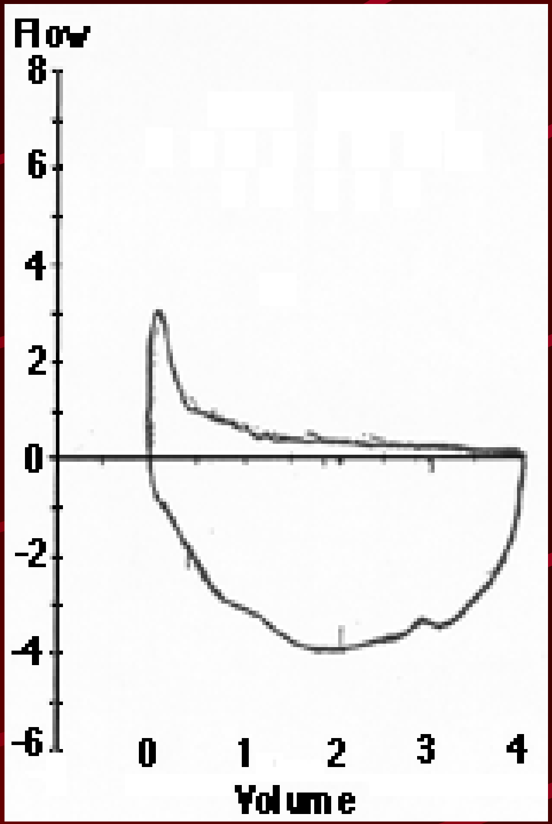
- The relationship between maximal flow rates on expiration and inspiration is demonstrated by the maximal flow-volume (MFV) loops.
- In subjects with healthy lungs the clinical importance of flow limitation will not be apparent, since maximal flow rates are rarely achieved even during vigorous exercise.
- In patients with severe COPD, limitation of expiratory flow occurs even during tidal breathing at rest.To increase ventilation these patients have to breathe at higher lung volumes and also allow more time for expiration by increasing flow rates during inspiration, where there is relatively less flow limitation. Thus patients with severe airflow limitation have a prolonged expiratory phase to their respiration.

Flow-volume graphs

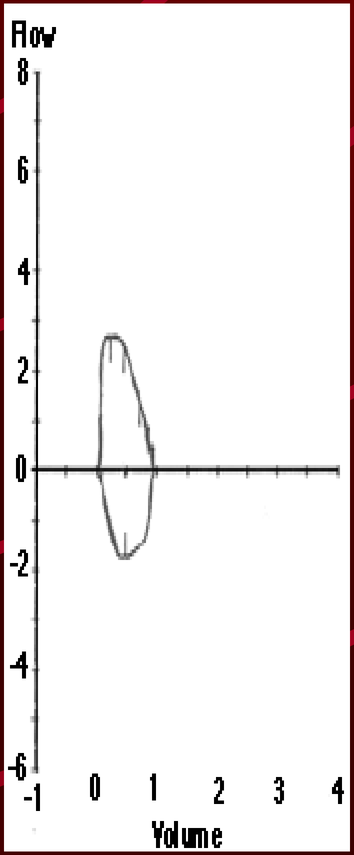
Normal

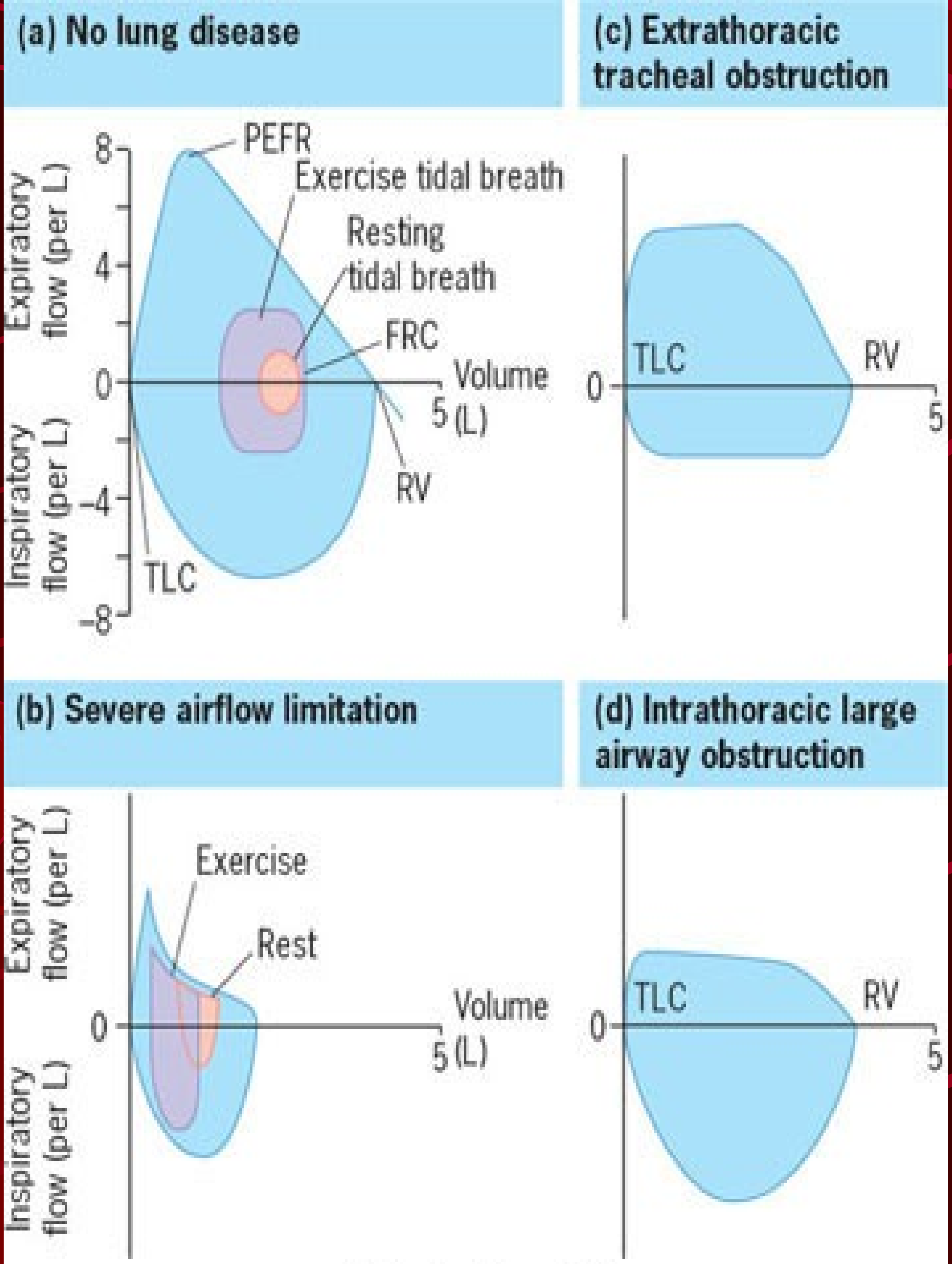


Obstructive

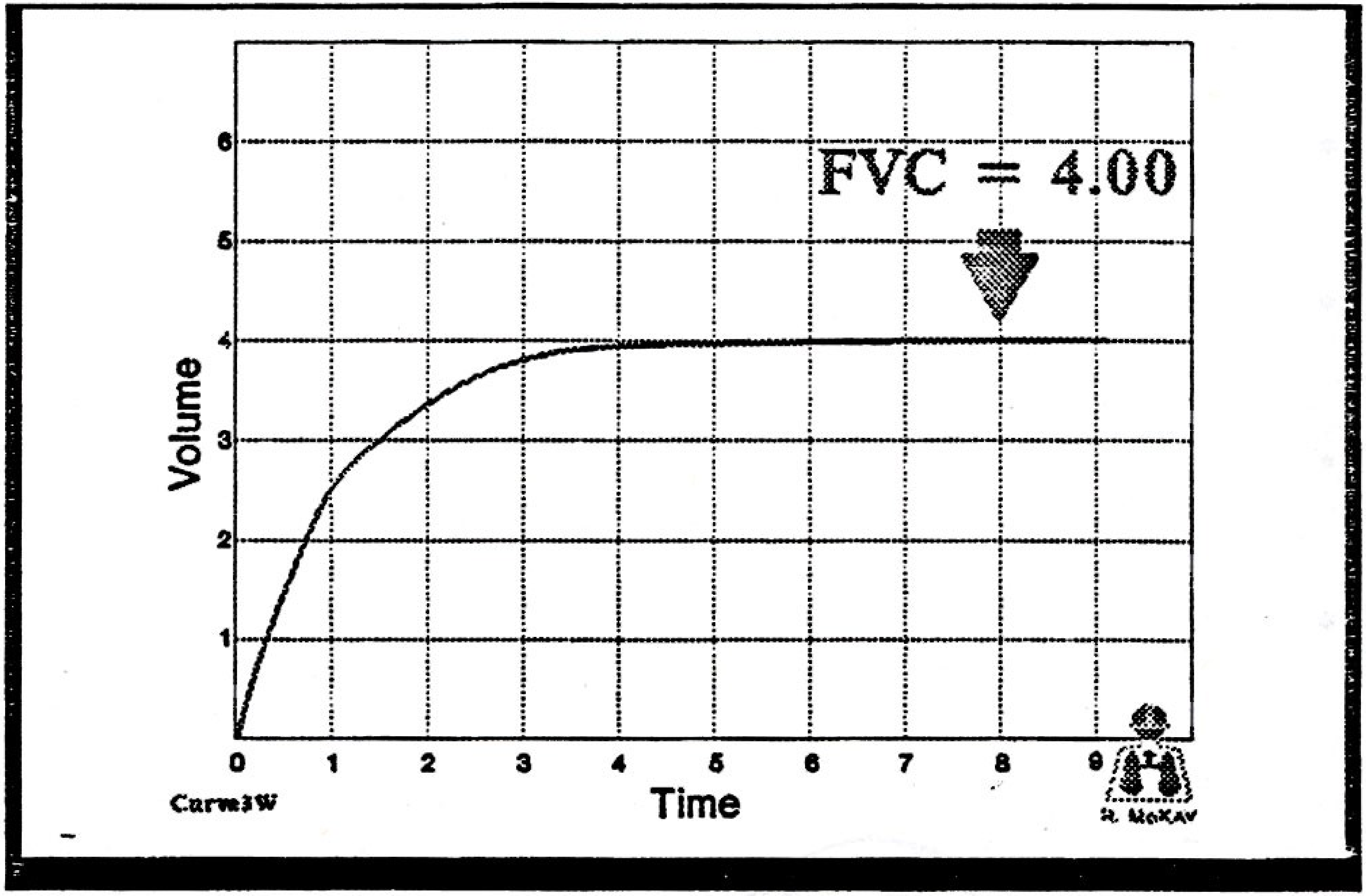


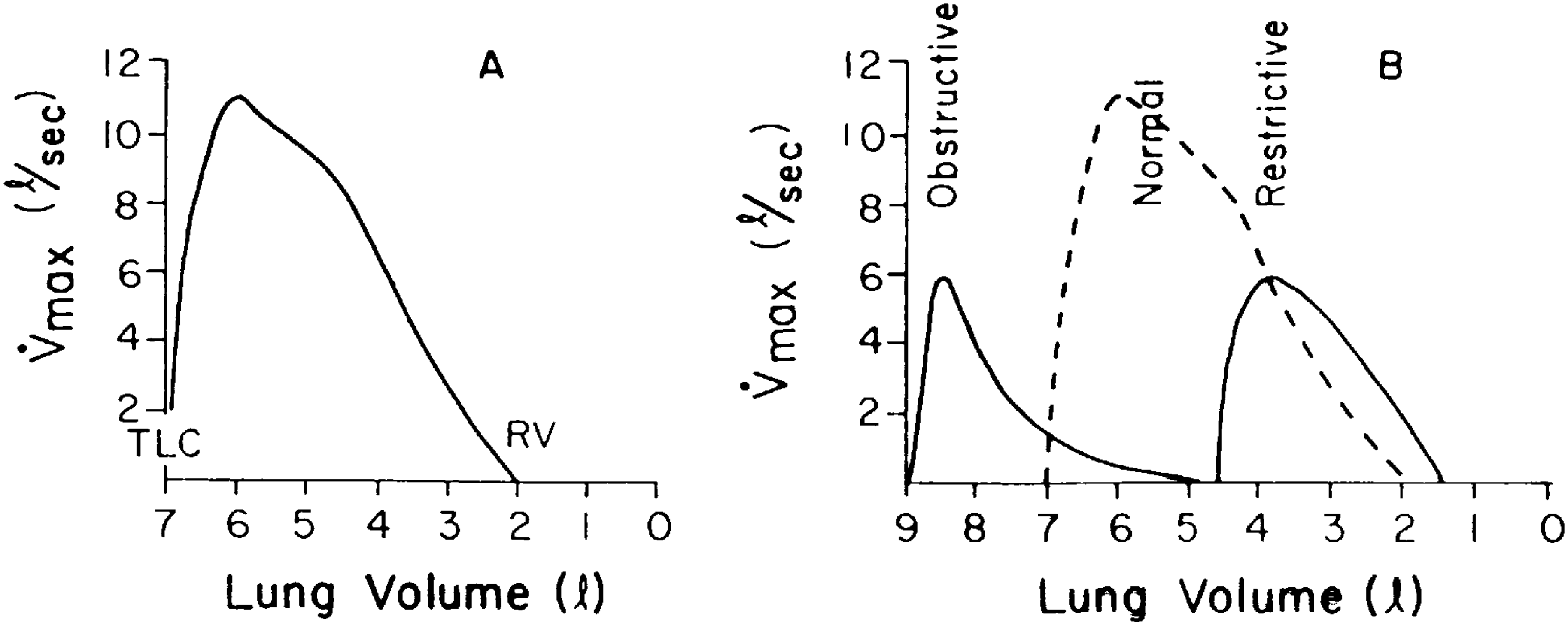
Restrictive




(a) No lung disease
PEFR
Exercise tidal breath
Resting tidal breath
FRC
Volume (L)
RV
TLC
Expiratory flow (per L)
Inspiratory flow (per L)
8
4
0
−4
−8
5
(b) Severe airflow limitation
Exercise
Rest
Expiratory flow (per L)
Inspiratory flow (per L)
0
Volume (L)
5
(c) Extrathoracic tracheal obstruction
TLC
RV
0
5
(d) Intrathoracic large airway obstruction
TLC
RV
0
5

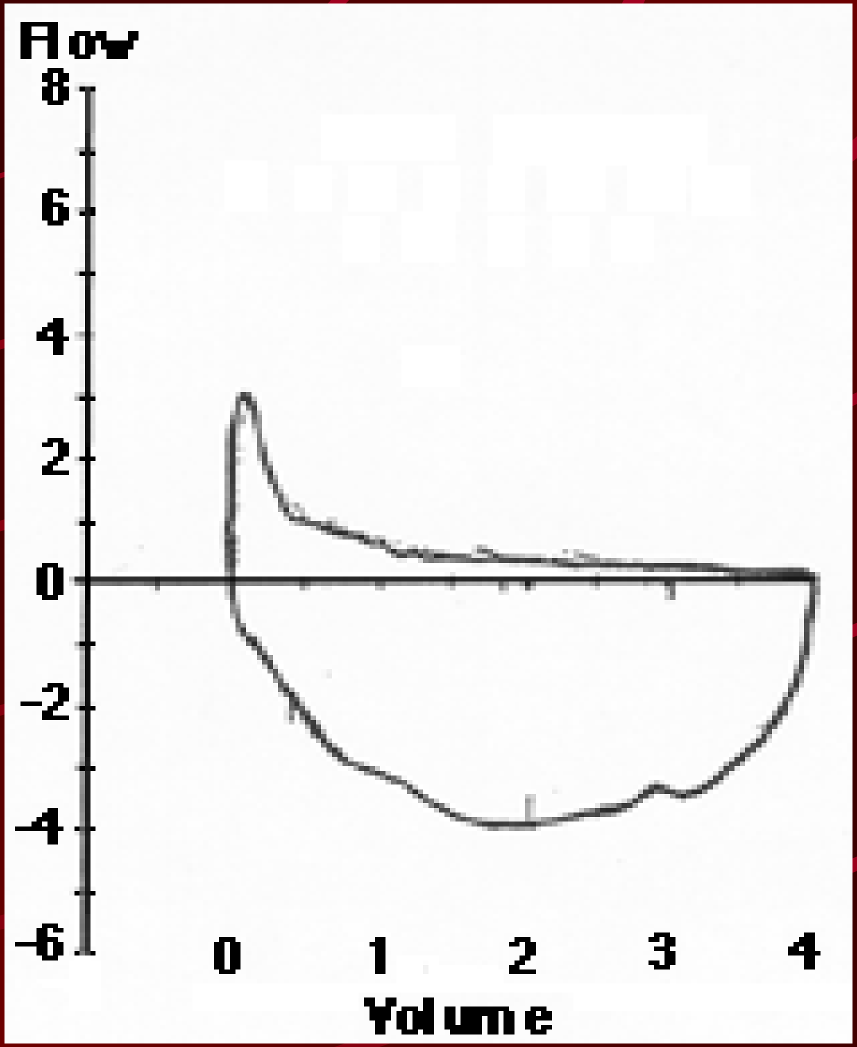
FVC





Note changes in lung volume and ↓ in flow in COPD and ↑ relative flow in restrictive disease

Severe obstruction

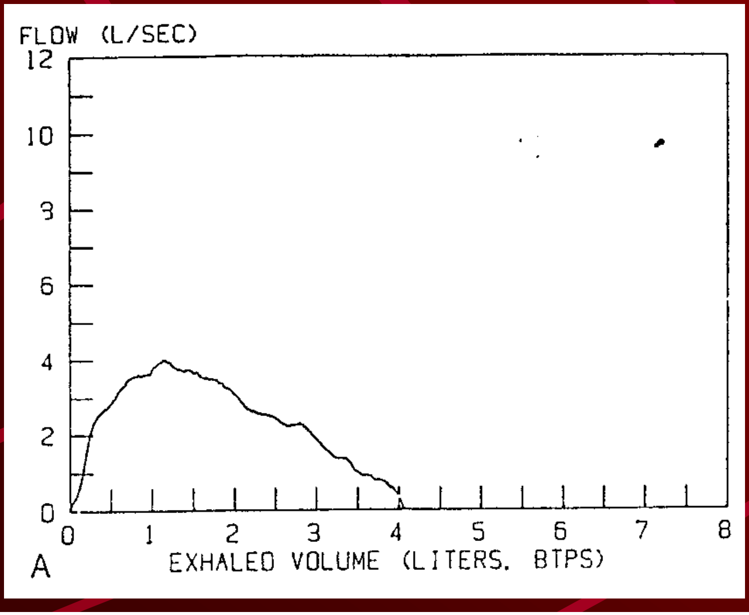


Flow-volume loops

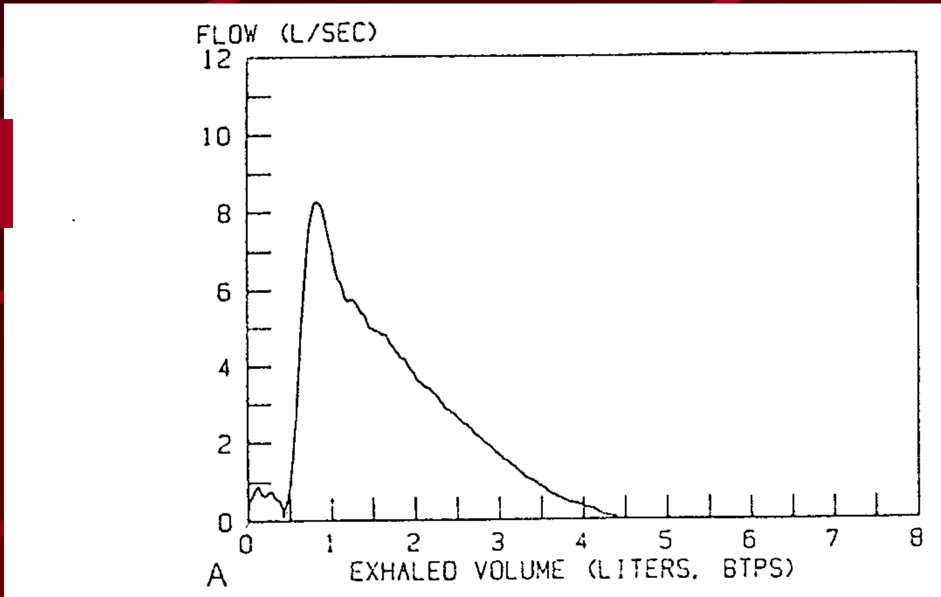
- The measure of the volume that can be forced in from RV in 1 second (FIV1) will always be greater than that which can be forced out from TLC in 1 second (FEV1). Thus, the ratio of FEV1 to FIV1 is below 1.
- The only exception to this occurs when there is significant obstruction to the airways outside the thorax, such as with a tumour mass in the upper part of the trachea. Under these circumstances expiratory airway narrowing is prevented by the tracheal resistance (a situation similar to pursing the lips) and expiratory airflow becomes more effort-dependent. During forced inspiration this same resistance causes such negative intraluminal pressure that the trachea is compressed by the surrounding atmospheric pressure. Inspiratory flow thus becomes less effort-dependent, and the ratio of FEV1 to FIV1 becomes greater than 1. This phenomenon, and the characteristic flow-volume loop, is used to diagnose extrathoracic airways obstruction. When obstruction occurs in large airways within the thorax (lower end of trachea and main bronchi), expiratory flow is impaired more than inspiratory flow but a characteristic plateau to expiratory flow is seen.

Unacceptable Curves

Delayed expiration

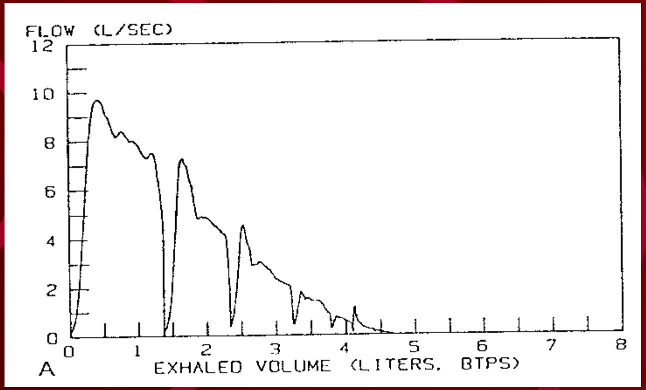


Suboptimal initial blow

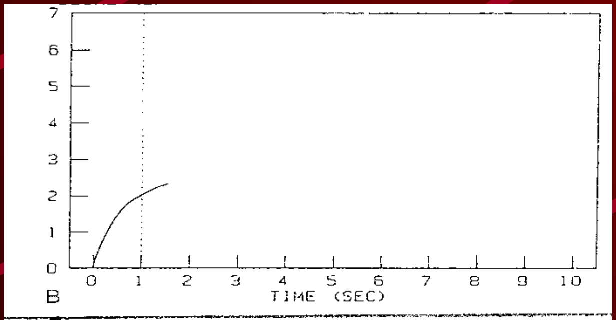


Unacceptable curves

- Cough



- Premature termination
  - (volume-time curve shown)



- Variable effort

Figure A3a. Unacceptable volume–time spirogram due to variable effort and early termination.

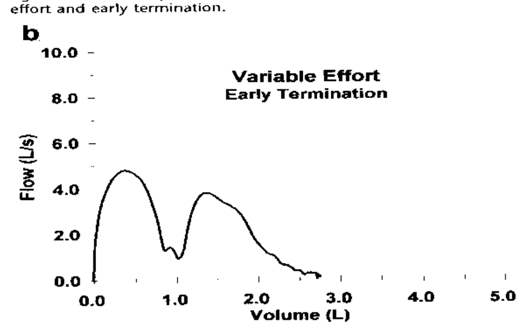
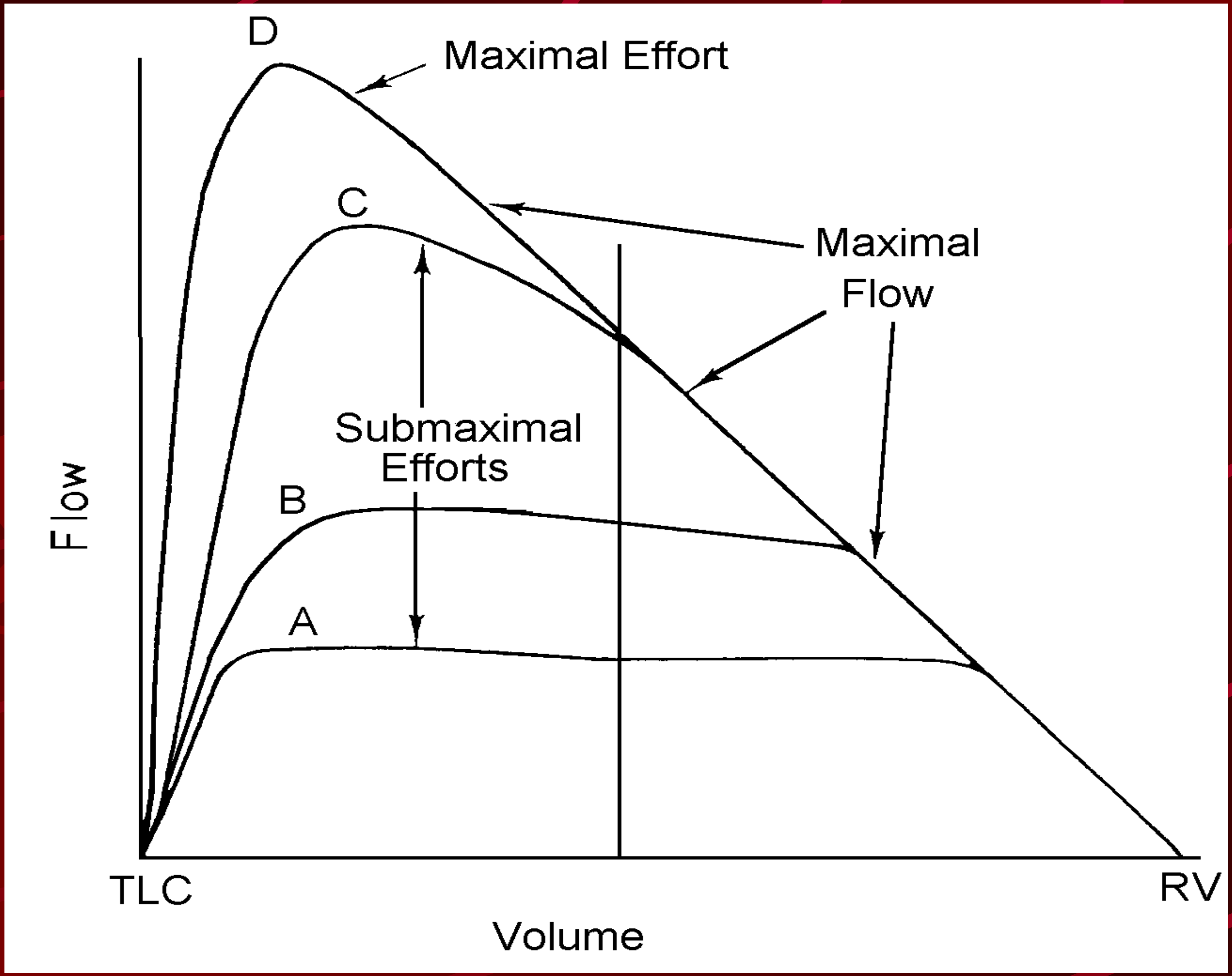


Figure A3b. Unacceptable flow–volume spirogram due to variable effort and early termination.

Scale effort



Obstructive and restrictive diseases

obstructive
- bronchial asthma (alergic, non-alergic), chron. bronchitis, emphysema, COPD

restrictive
- idiopatic pulmonary fibrosis, sarcoidosis, professional intersticial diseases, pleural diseases, pneumothorax, scoliosis, neuromuscular disorders, pneumonia

Scales of obstruction

Grade	***FEV1/FVC***	***FEV1(ml)***
Very Severe	<0.3	<600
Severe	0.3-0.4	600-1000
Moderate	0.4-0.6	1000-2000
Mild	0.6-0.7	2000-3000
Very Mild	0.7-pred. value	>3000

Scales of restrictive disorders

Grade	*VC% predicted*	*TLC% predicted*
Very Mild	>80	>90
Mild	60-80	70-90
Moderate	30-60	50-70
Severe	<30	<50

22 –year-old patient

- Pre-bronchodilator
- FVC 4.0(80%)
- FEV1 2.0 (66%)
- FEV1/FVC 50%
- MVV 70L (665)

- Post-bronchodilator
- FVC 4.2
- FEV1 3.0 (100%)
- FEV1/FVC 73%
- MVV 105 L

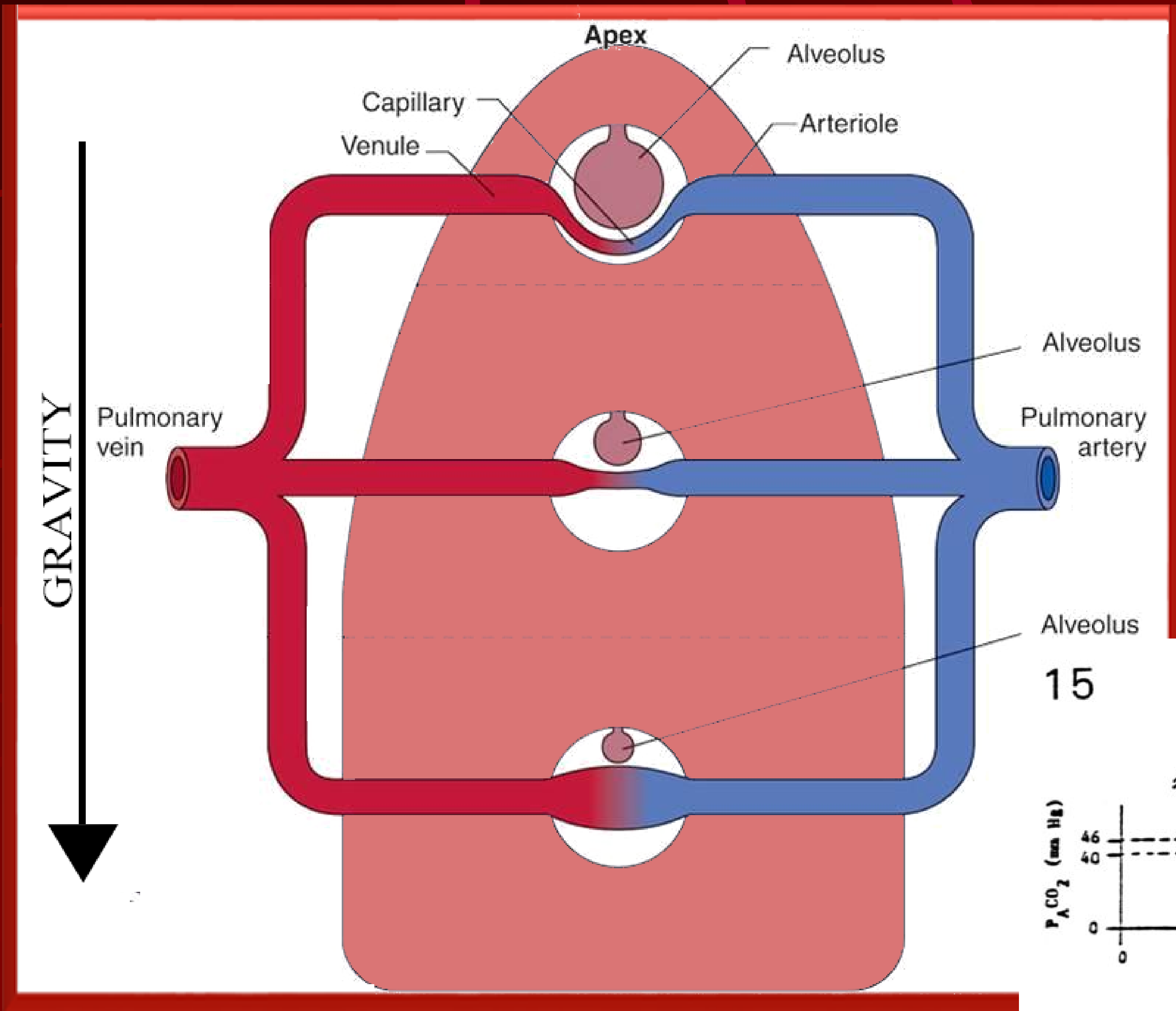
Arterial Blood Gases

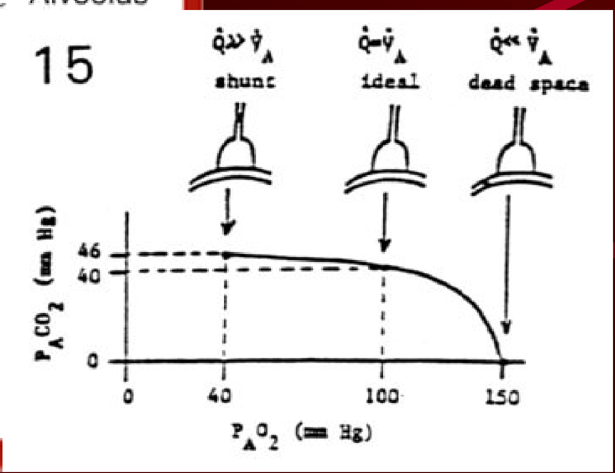
- Measurement of pH, pCO_2 and pO_2 on room air is often done along with spiro, lung volumes and diffusing capacity to help characterize the severity of disease

Ventilation-perfusion

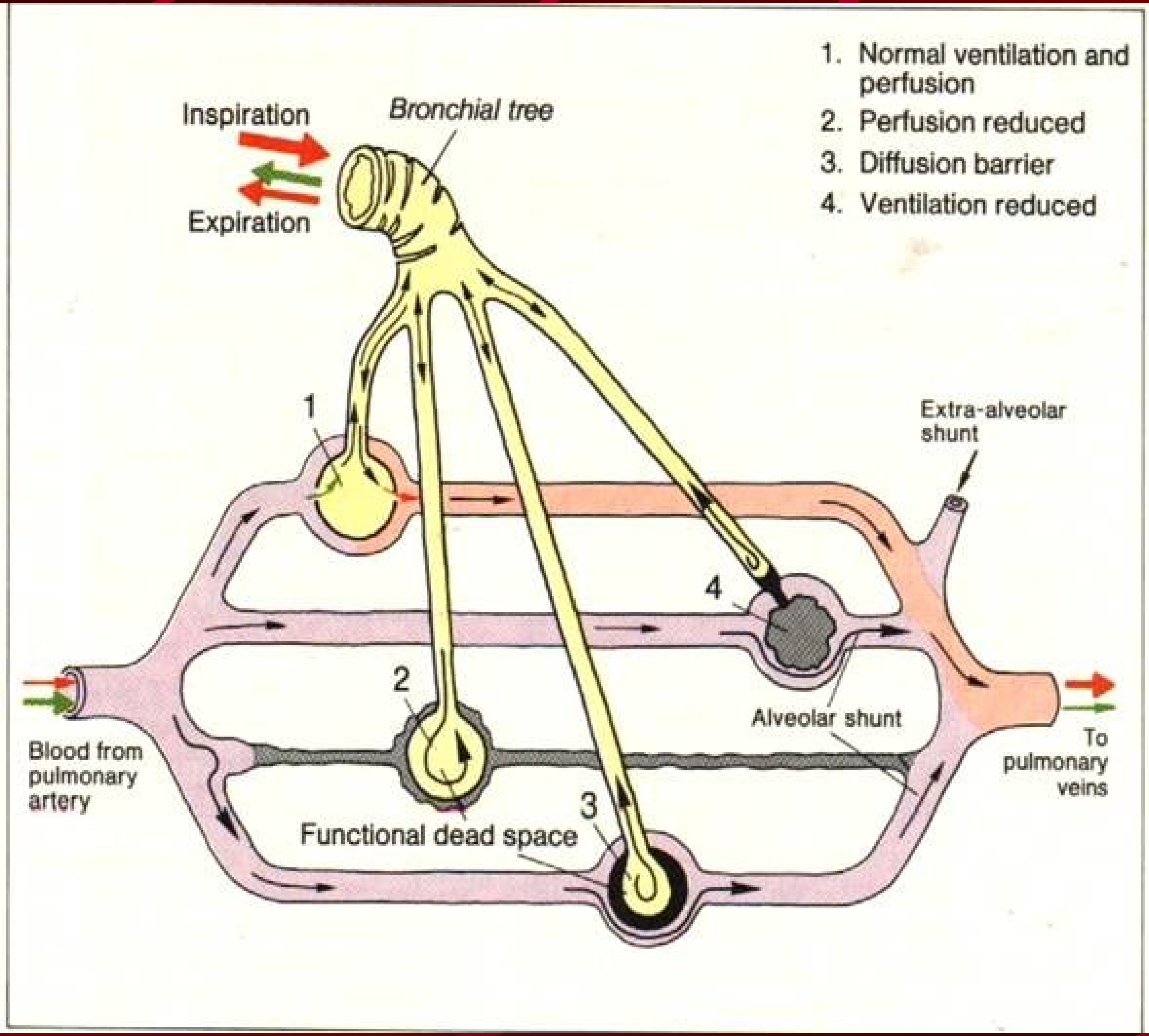
- For efficient gas exchange it is important that there is a match between ventilation of the alveoli ([Vdot]A) and their perfusion ([Qdot]).
- There is a wide variation in the [Vdot]A/[Qdot] ratio throughout both normal and diseased lung. In the normal lung the extreme relationships between alveolar ventilation and perfusion are:
- ventilation with reduced perfusion (physiological deadspace)
- perfusion with reduced ventilation (physiological shunting).
- In normal lungs there is a tendency for ventilation not to be matched by perfusion towards the apices, with the reverse occurring at the bases.

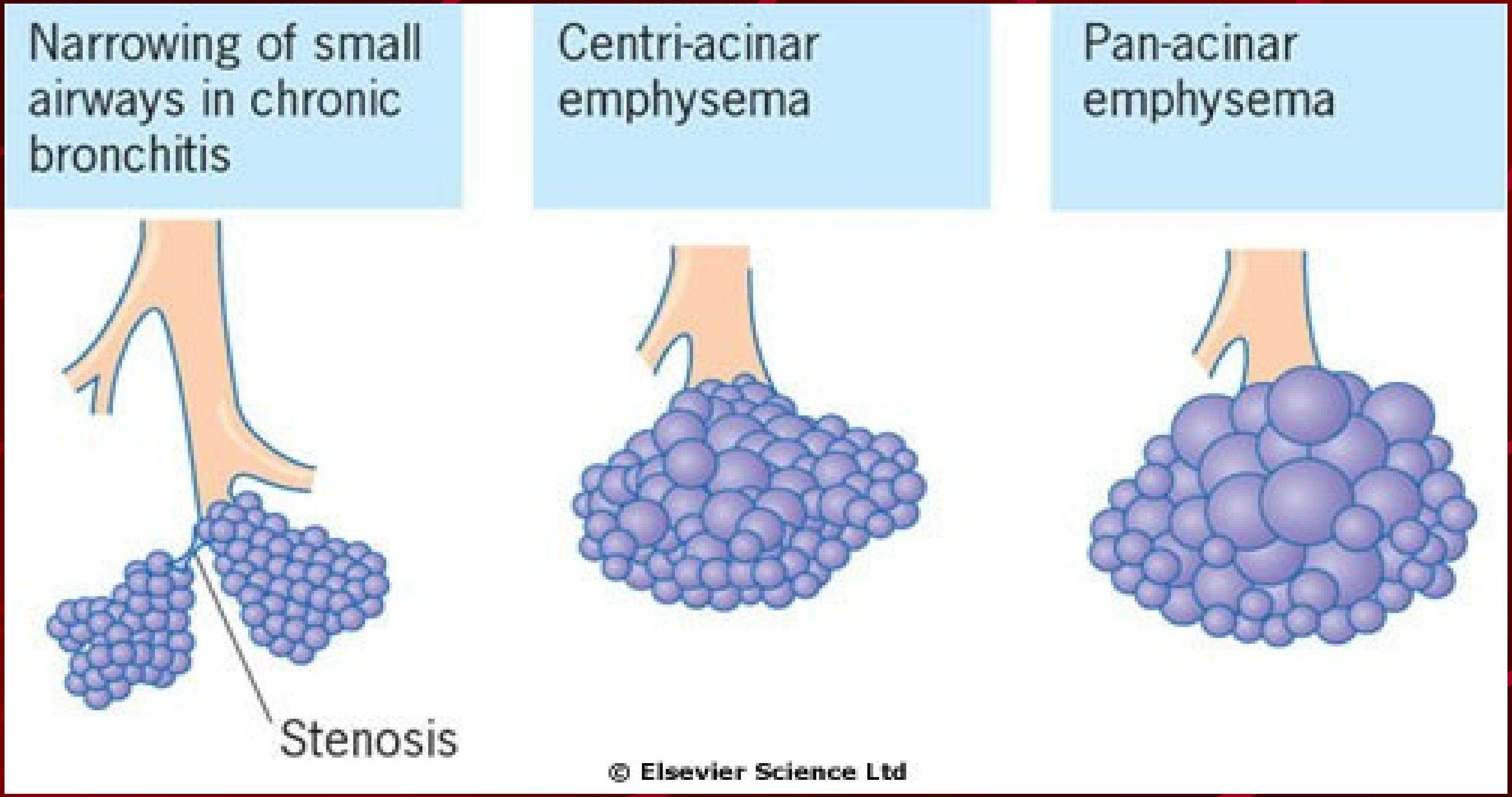
Physiological shunt and dead space





Shunt and dead space

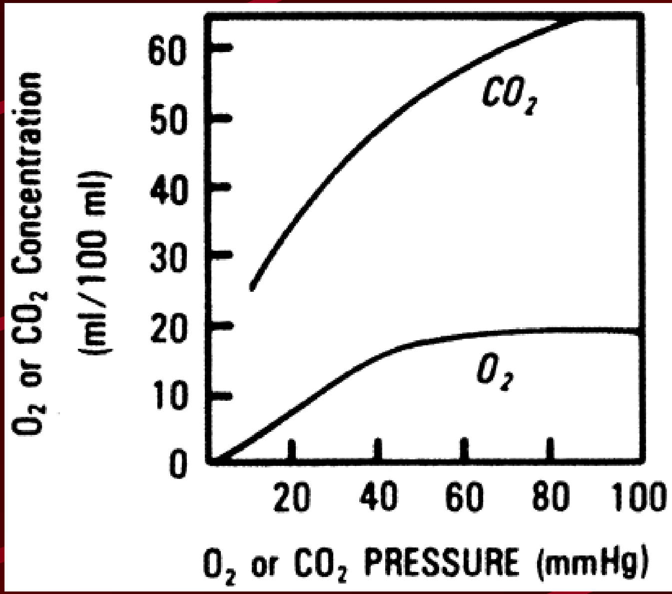




Pathological features of chronic bronchitis and emphysema.

Respiratory insufficiency

- Partial
  - Isolated hypoxia (the saturation of Hb may be lower, than in global insufficiency)
  - When only a part of lung is affected, the hypercapnia may be compensated by unaffected alveoli (this results from CO_2 dissociation curve)
  - On the other hand, the saturation by O_2 in a blod flowing out of healthy alveoli is almost 100% (97-99%), the hypoxia in one pulmonary segment can‘t be compensated by another
- Global
  - Hypoxia with hypercapnia
  - When whole respiretory system is affected, both O_2 and CO_2 exchange is affected



Chronic obstructive pulmonary disease

- Pink puffers
  - emphysema predominance
  - collapse of bronchioli and alveolar septa
  - ↑ dead space
  - respiratory insufficiency occur only in late stages, but high respiratory effort to overcome the dead space is necessary
  - α1-antitrypsin deficiency (hereditary or acquired - smoking)
- Blue bloaters
  - chronic bronchitis predominance
  - obstruction of bronchioli
  - ↑ shunt
  - partial, later global respiratory insufficiency
  - hypercapnia occur only in later stages – respiratory effort does not occur
  - severe hypoxia– increased deoxyHb
  - mostly smokers

pink puffers vs. blue bloaters