Human Phys PCB4701

Respiration part 1

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Analysis of Internal Transport in an Organism:

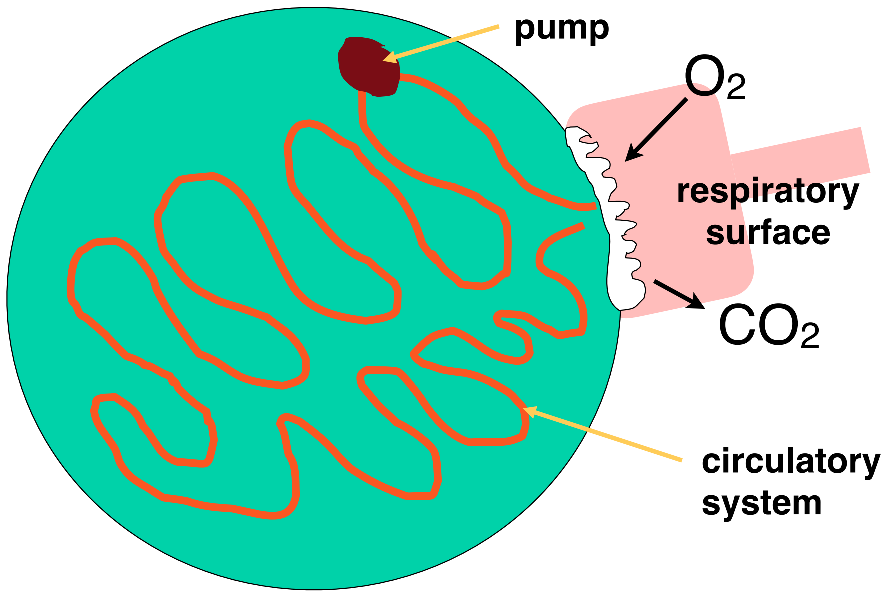
Movement of chemicals from external environment into the body, and between organs of the body.

Tranported Chemicals can be essential metabolic nutrients (O_2, glucose) or toxic waste products (CO_2, N (urea), heme (bilirubin))

1. What is the internal transport system that carries the chemicals from the exchange surface to target tissues?
2. What provides & controls the force to move chemicals through the system?
3. What are the exchange surfaces?
4. How do the chemicals enter/exit the cells of the exchange surface?
5. How are the chemicals unloaded by the transport system and taken up by the target cells?

Respiration:

Get O_2 from outside environment into deep tissues,;
get CO_2 out of tissues



Cellular Respiration:
O_2 used by tissues in oxidative phosphorylation;
CO_2 produced as waste product by glycolysis.

Analysis of Internal Transport in an Organism:

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Tranported Chemicals can be essential metabolic nutrients (O_2, glucose) or toxic waste products (CO_2, N (urea), heme (bilirubin))

1. What is the internal transport system that carries the chemicals from the exchange surface to target tissues? lungs, circulation
2. What provides & controls the force to move chemicals through the system? breathing (suction), regulation by brainstem and pH
3. What are the exchange surfaces? alveoli, capillaries
4. How do the chemicals enter/exit the cells of the exchange surface?

   diffusion
5. How are the chemicals unloaded by the transport system and taken up by the target cells? hemoglobin, O_2 levels, pH

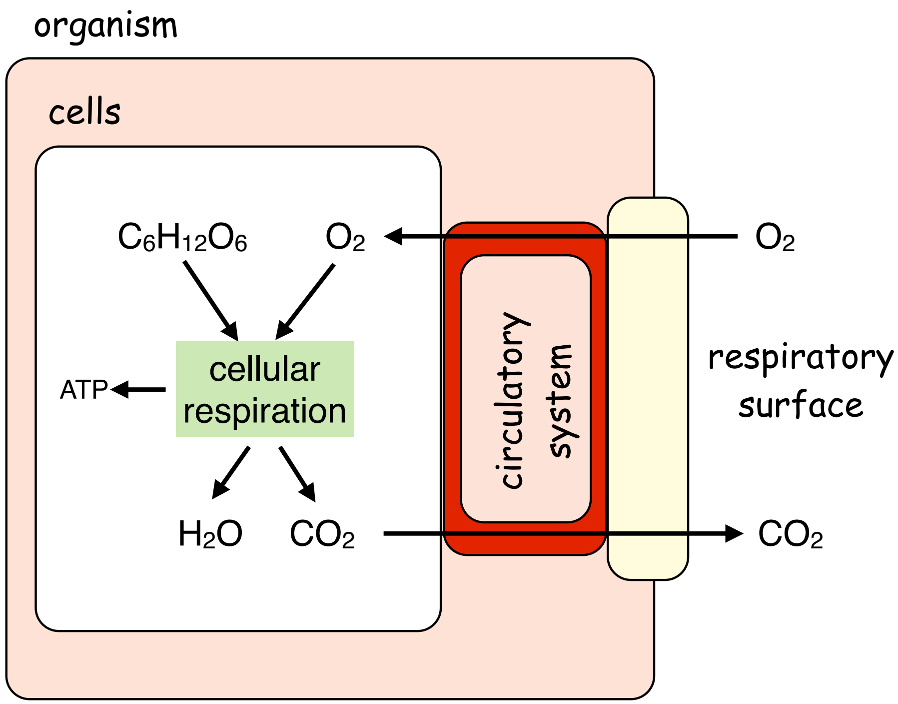
Respiration

Tuesday
Anatomy of Lungs
Mechanics of Breathing
Lung Volume
Gas Concentrations
Control of Breathing

Thursday:
Role of CO_2 and HCO_3 (bicarbonate) as buffer
Hemoglobin
Transport of CO_2
Fetal Circulation

Respiration:

Get O_2 from outside environment into deep tissues;
get CO_2 out of tissues



Cellular Respiration:

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CO_2 produced as waste product by glycolysis.

Basic Anatomy of the Lungs

glottis
larynx
trachea
right & left bronchus
bronchiole
alveolar sac
alveoli
Type 1 alveolar cell
Type 2 alveolar cell

diaphragm - divides **thoracic** cavity from **abdominopelvic** space

intrapleural space **-** space between outer lining of lungs and inner lining of thorax

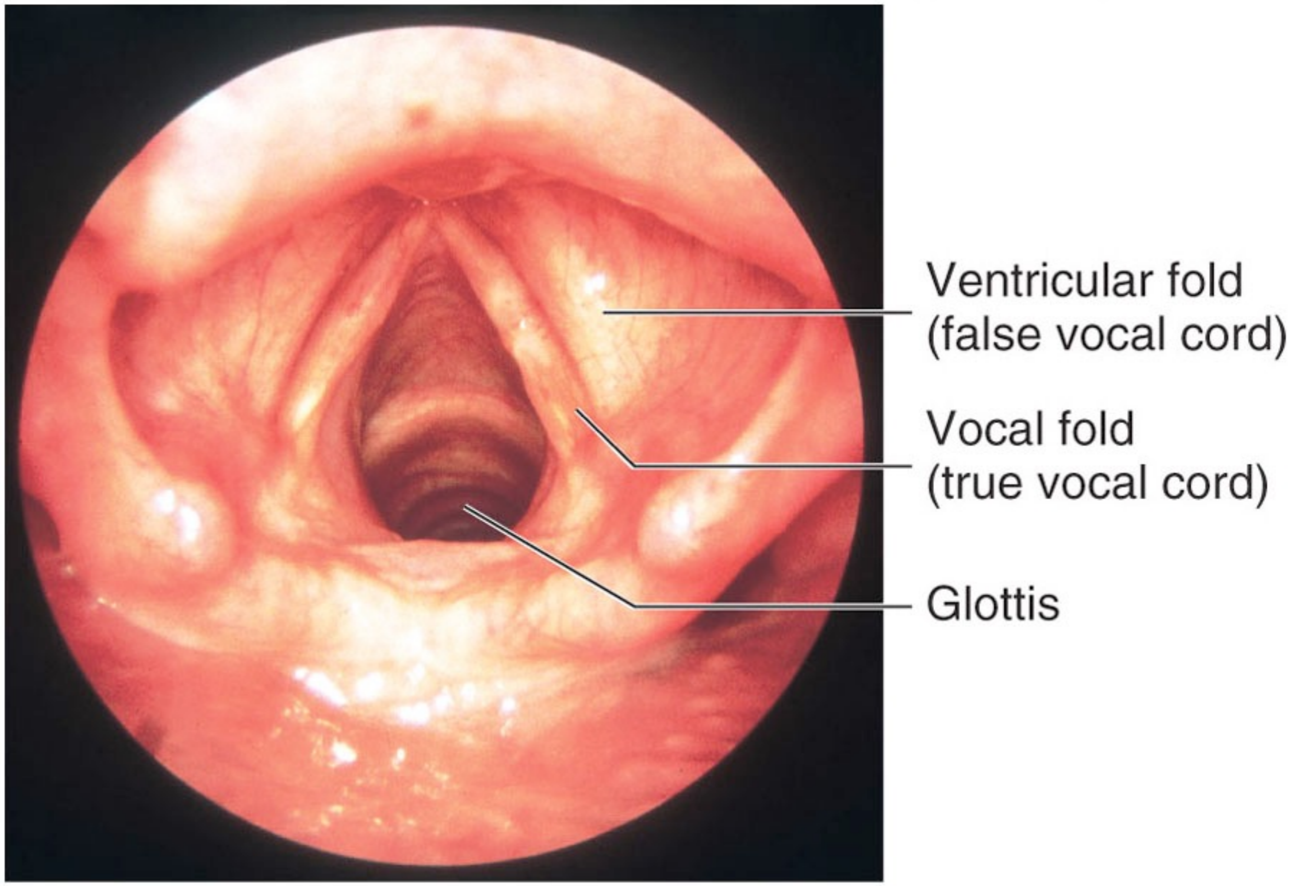

Ventricular fold
(false vocal cord)
Vocal fold
(true vocal cord)
Glottis

Figure 16.6

Figure 16.5a

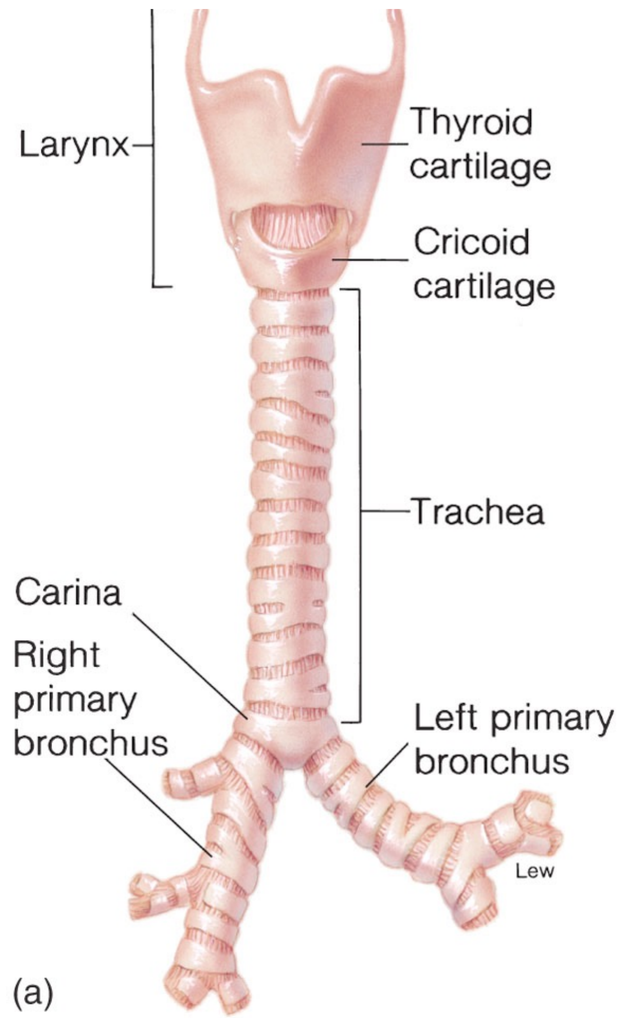

Larynx
Thyroid cartilage
Cricoid cartilage
Trachea
Carina
Right primary bronchus
Left primary bronchus
Lew
(a)

Figure 16.7

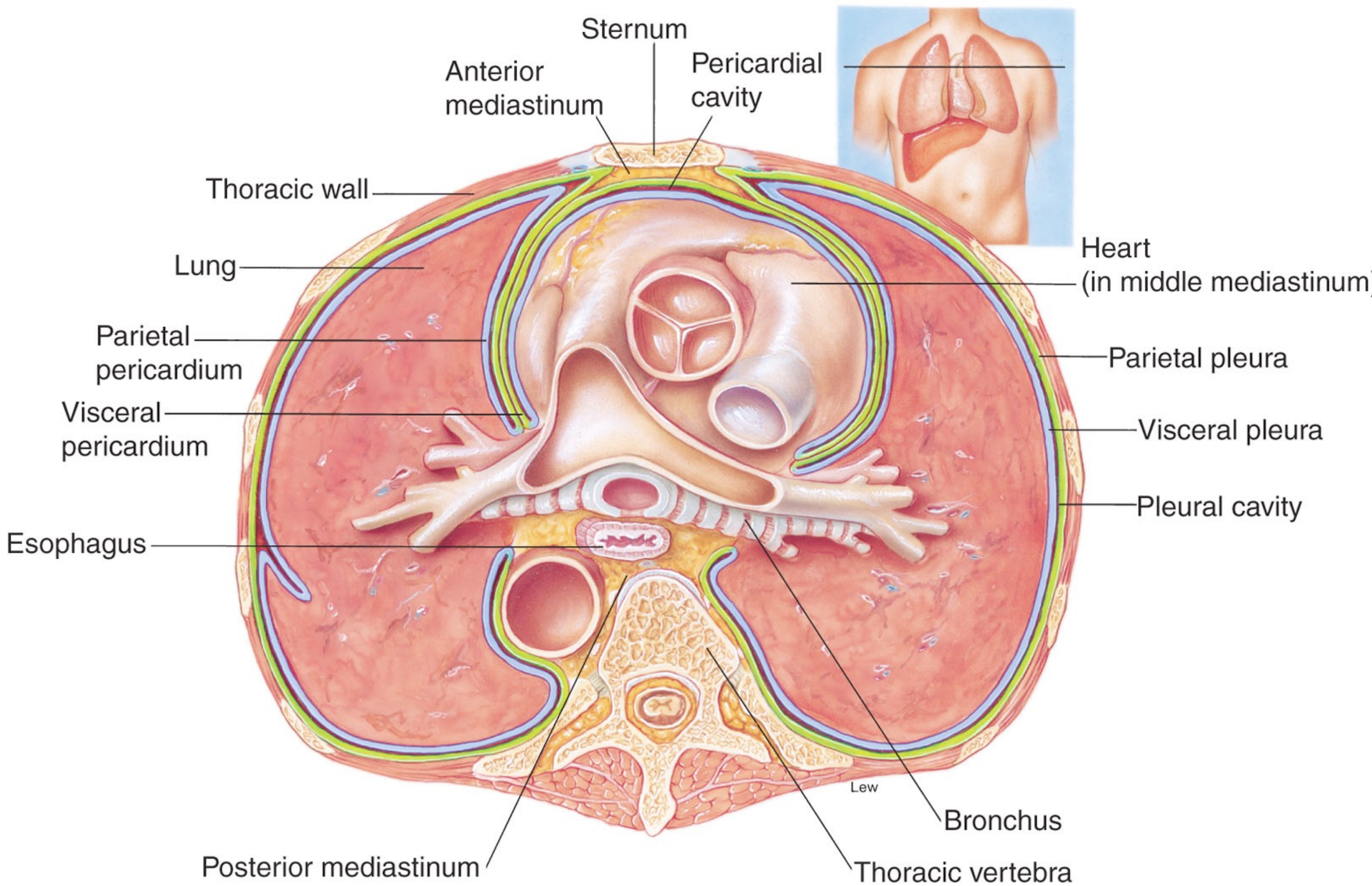
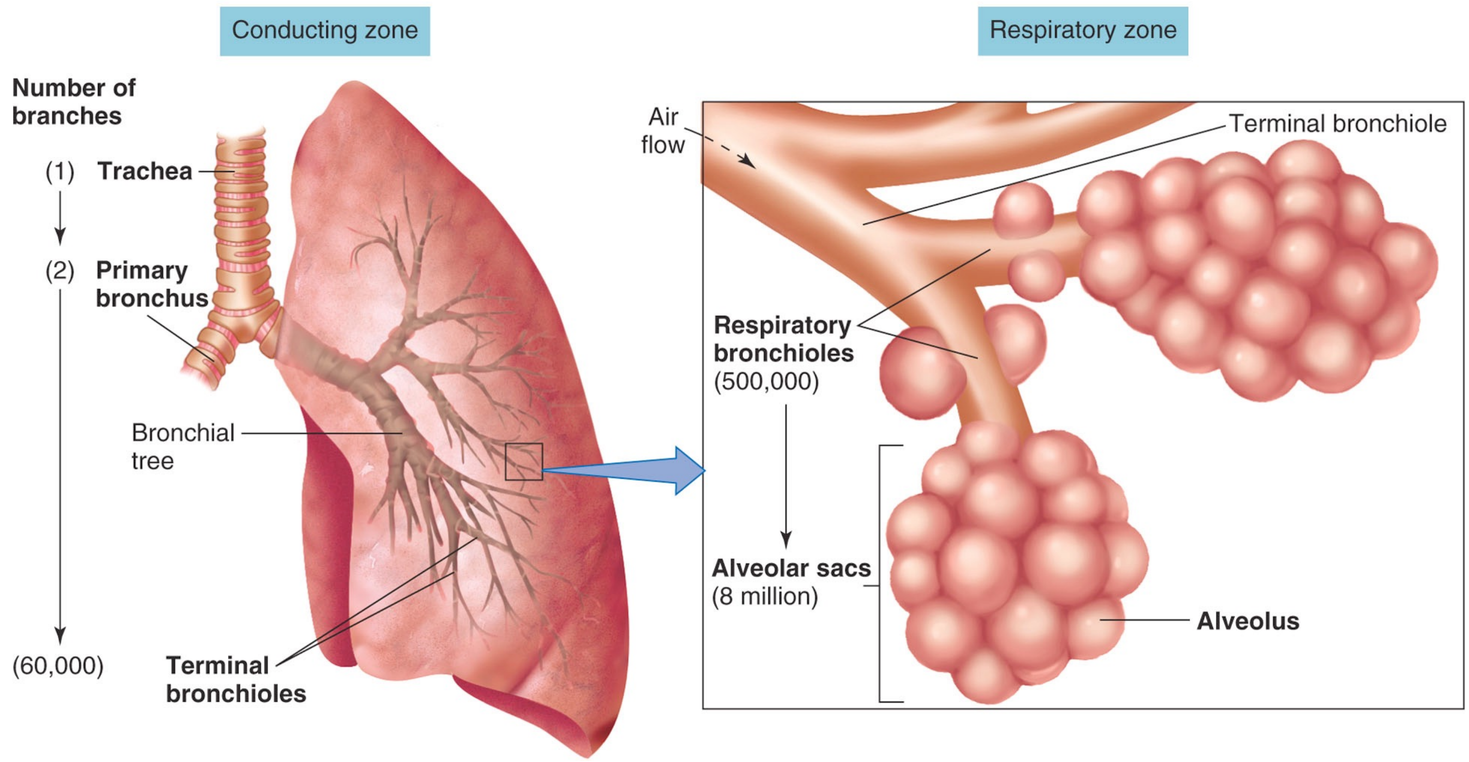

Sternum
Anterior mediastinum
Pericardial cavity
Thoracic wall
Lung
Parietal pericardium
Visceral pericardium
Esophagus
Heart (in middle mediastinum)
Parietal pleura
Visceral pleura
Pleural cavity
Lew
Bronchus
Posterior mediastinum
Thoracic vertebra

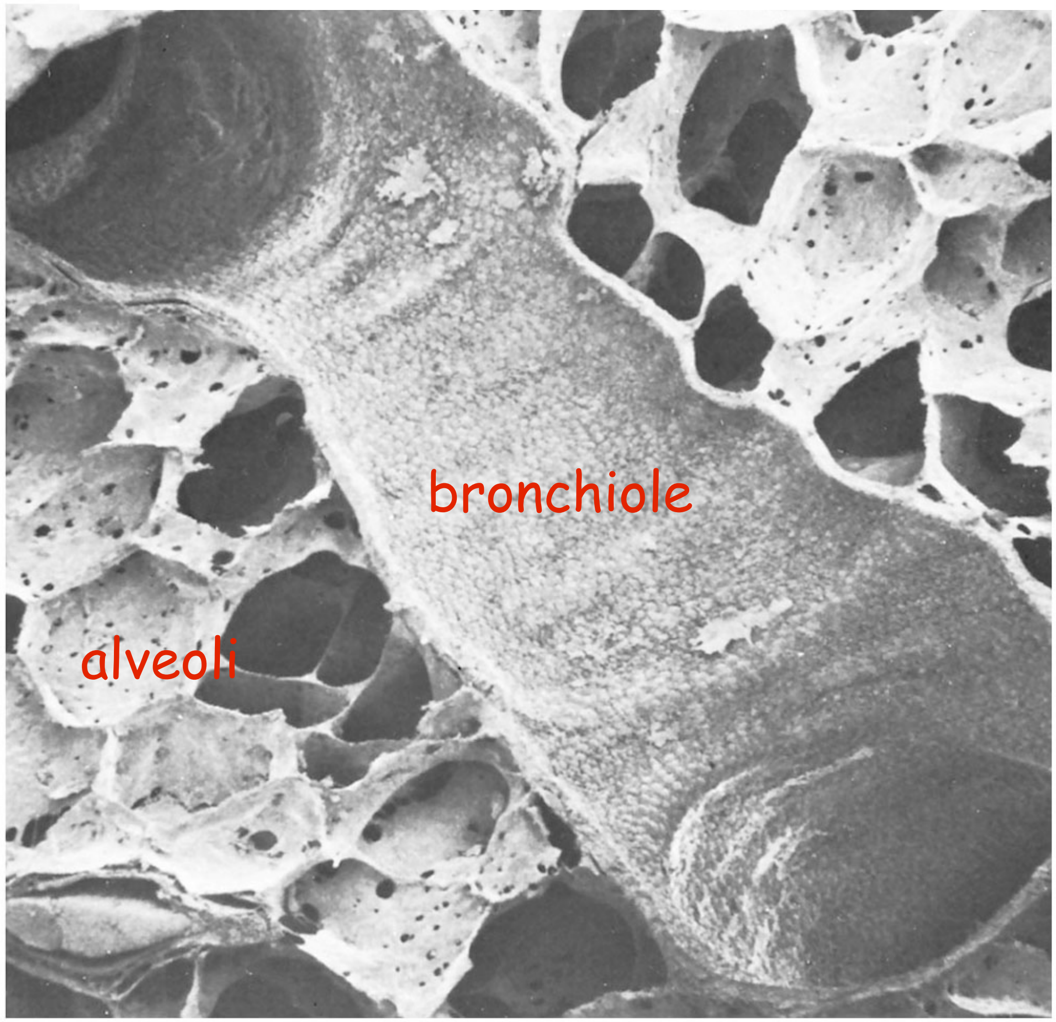
Figure 16.4



Tubes for bulk transport –> many small spheres for increased surface area

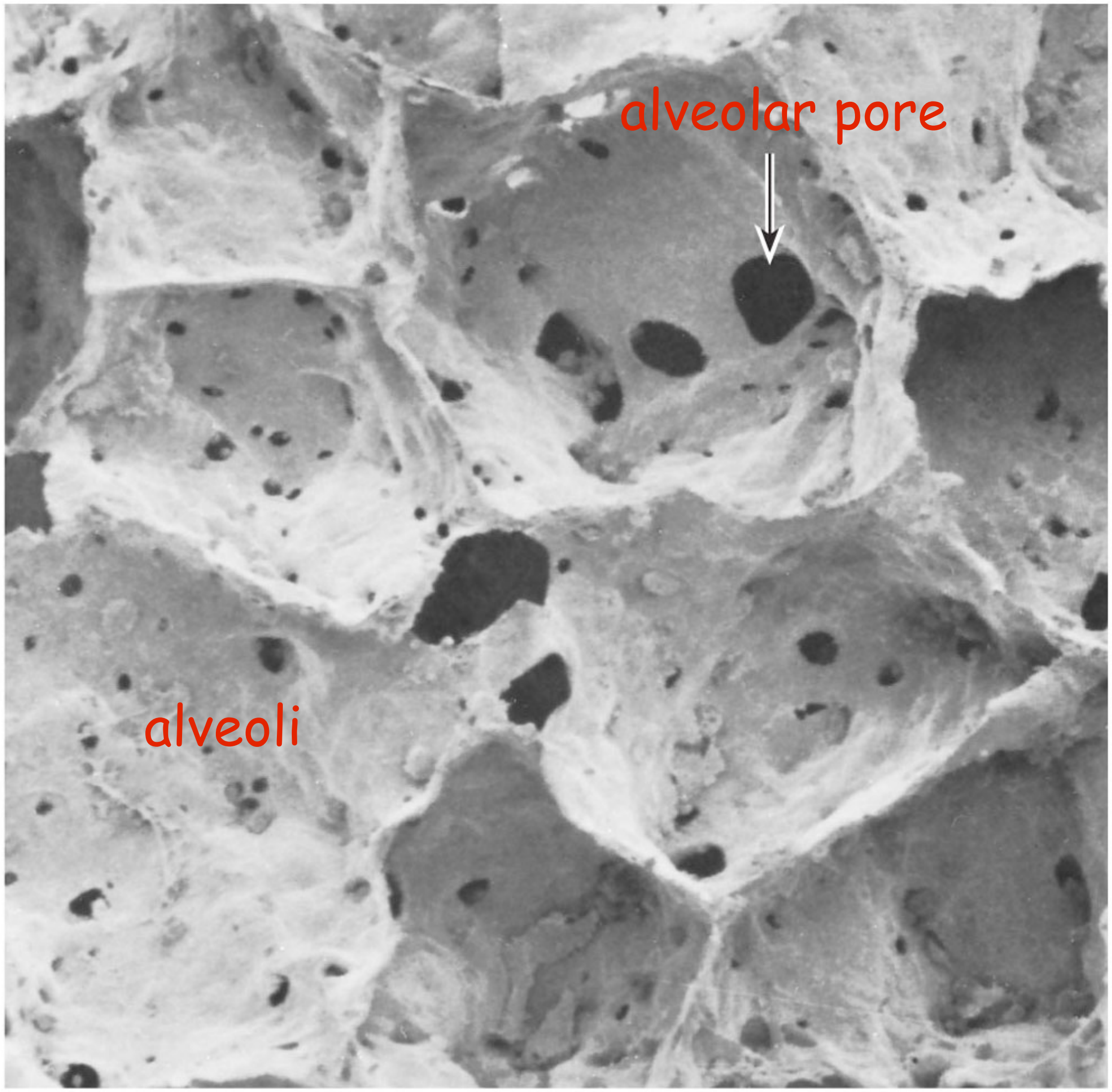
1 trachea –> 8 million alveoli

Figure 16.3a



(a)

Figure 16.3b



(b)

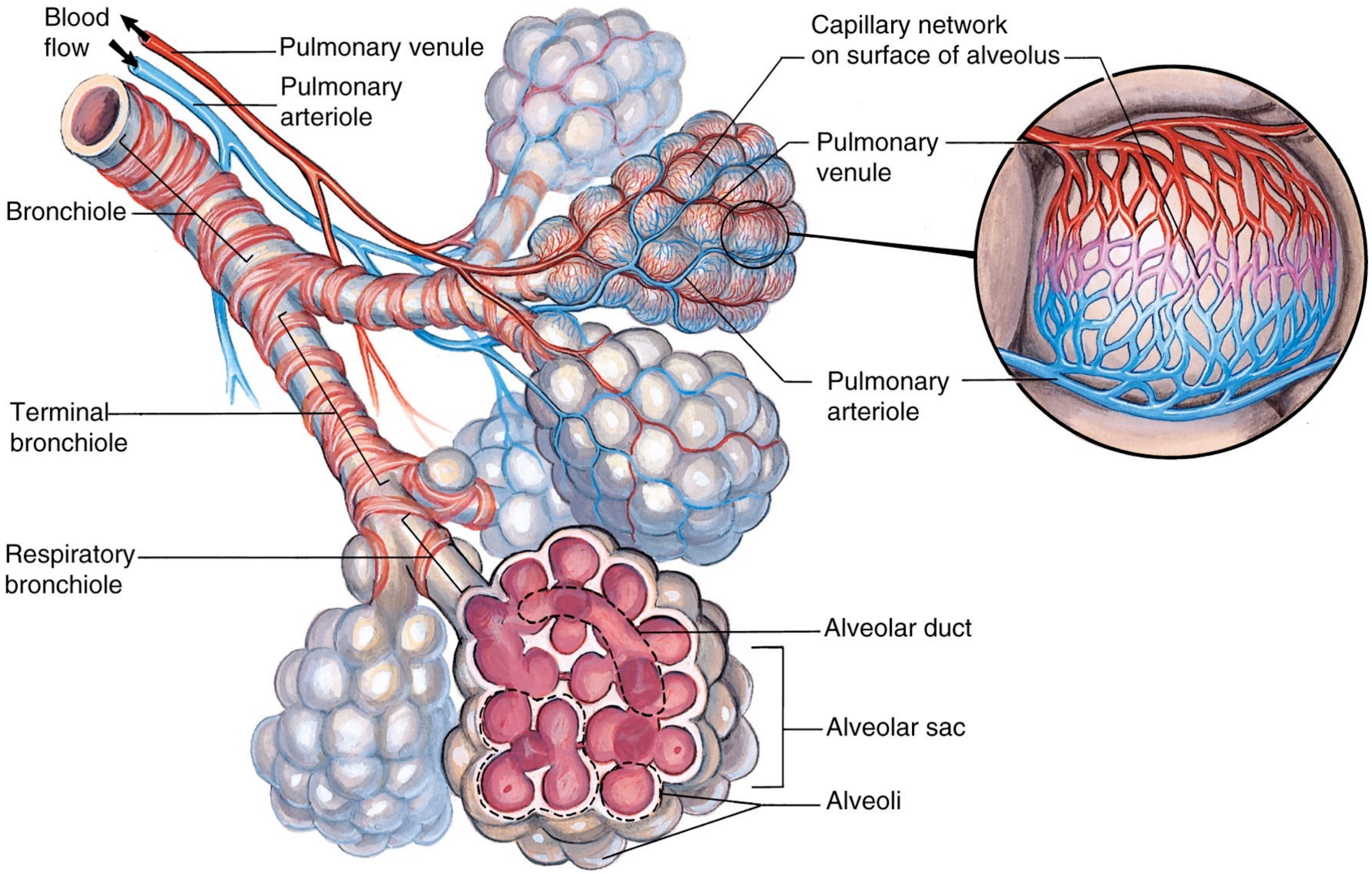

Blood
flow
Pulmonary venule
Pulmonary
arteriole
Capillary network
on surface of alveolus
Pulmonary
venule
Bronchiole
Pulmonary
arteriole
Terminal
bronchiole
Respiratory
bronchiole
Alveolar duct
Alveolar sac
Alveoli

Figure 16.20

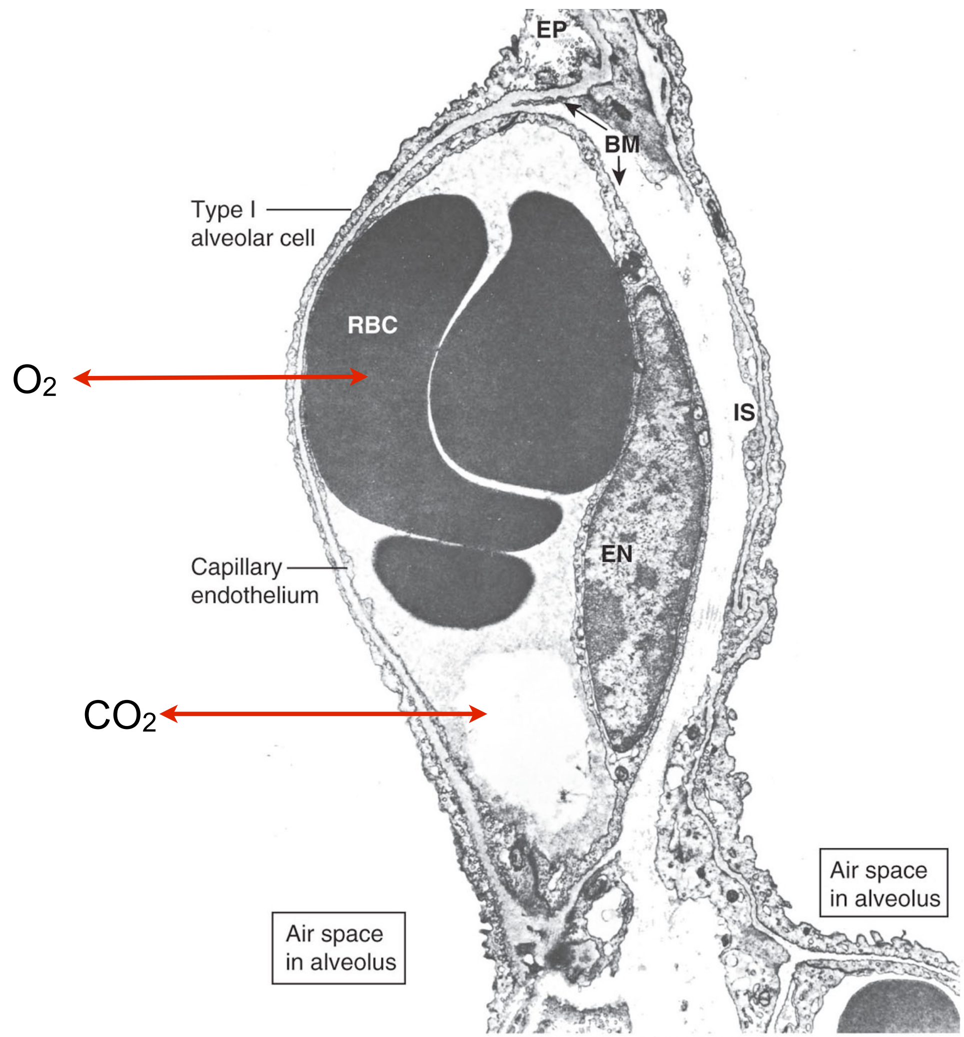

EP
BM
Type I alveolar cell
RBC
O_2
IS
EN
Capillary endothelium
CO_2
Air space in alveolus
Air space in alveolus

Figure 16.2

Physical Properties of Lungs

Compliance
amount that lung inflates with given pressure change

Elasticity
amount that lung resists inflation and recoils back to resting state (loungs stuck to chest wall, so always in elastic tension)

Surface Tension
thin film of fluid on inside of alveoli has surface tension (attraction of water molecules), tending to collapse the alveoli. Type 2 alveolar cells produce
surfactant a phospholipid-protein detergent that breaks surface tension.

Disorders of surface tension:
cystic fibrosis: genetic defect of a Cl- transporter causes lack of fluid secretion, so airway fluid is excessively viscous

acute respiratory distress syndrome (ARDS): inflammation in lungs leads to excessive accumulation of fluid & reduced surfactant release

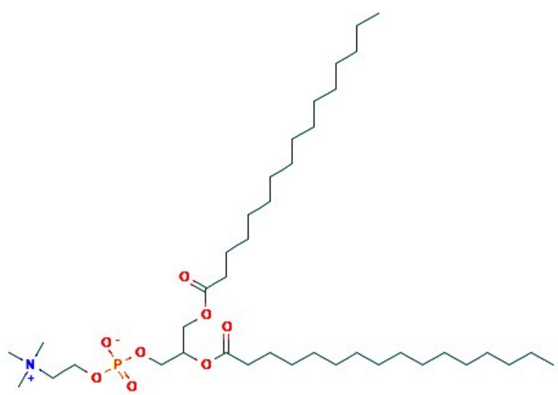
premature infant: surfactant not produced until late in gestation (just before birth), so premature infants have collapsed alveoli unless exogenous surfactant administered.

The smaller the radius, the stronger the pressure exerted by surface tension

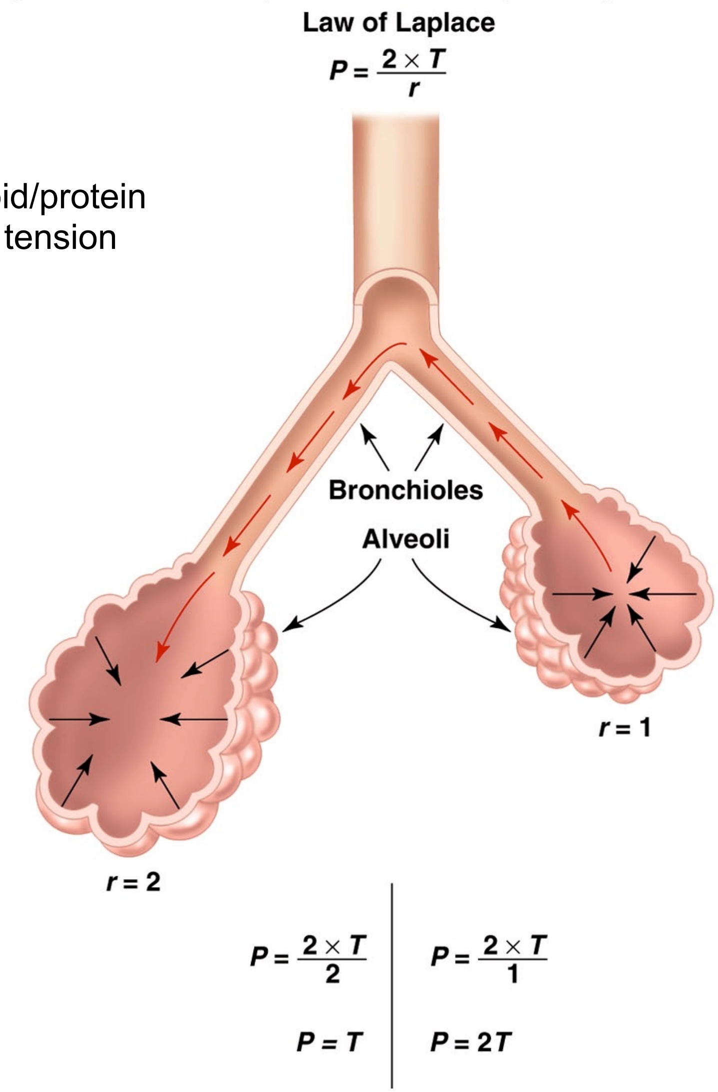
Small, fluid coated alveoli tend to close up because of surface-tension of fluid

surfactant (“surface active agent”) phospholipid/protein acts as natural detergent to break up surface tension make it easier to inflate alveoli.

Synthesized by Type II cells in late fetal life, so pre-term babies may not have enough.



1,2-Dipalmitoylphosphatidylcholine



Surfactant & premature infant

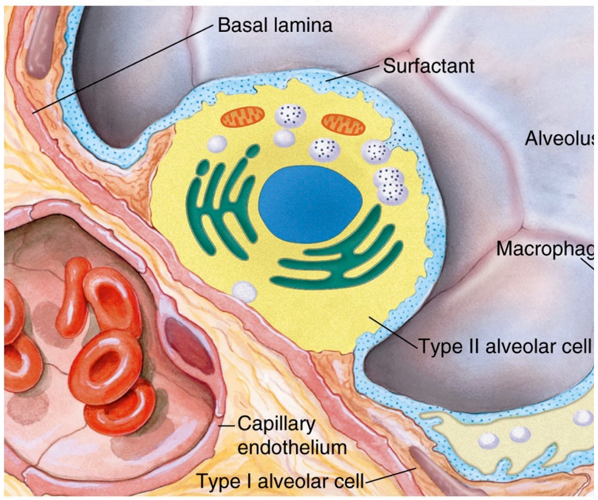
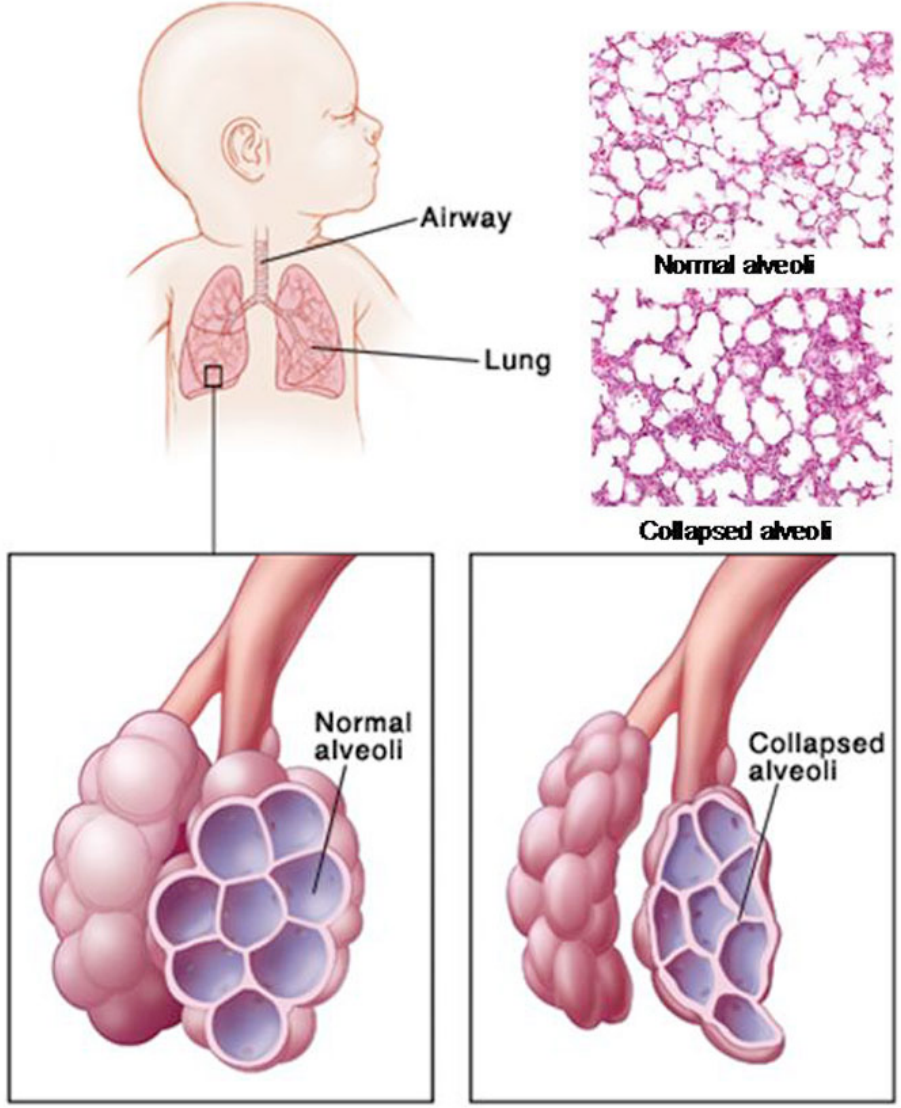


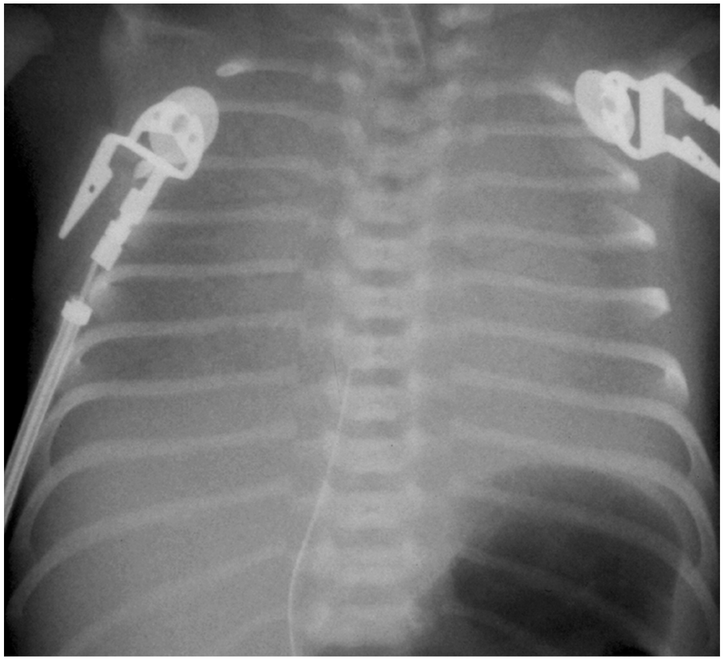
Figure 16.11

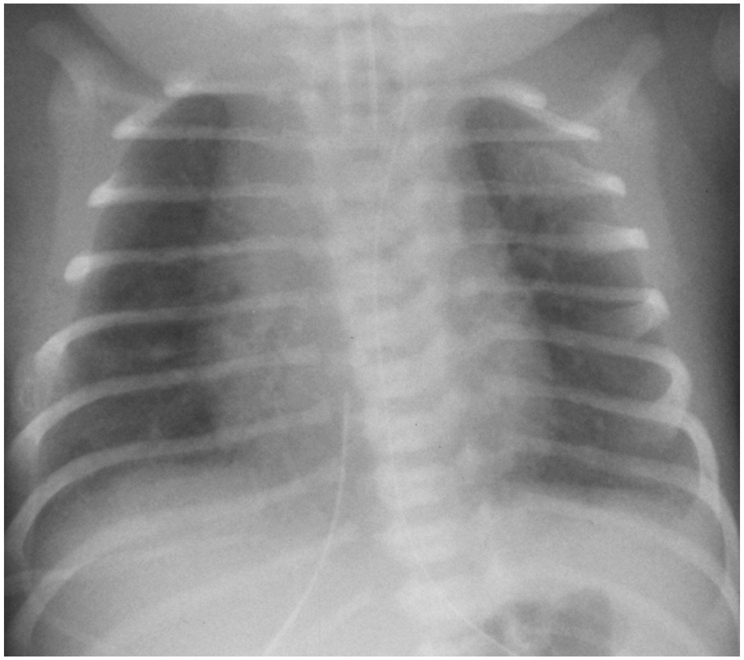


http://www2.hawaii.edu/~yzuo/research1-surfactant.html

Symmetric surfactant effect in a 36-week-gestational age infant of a diabetic mother.

Pretreatment radiograph shows diminished lung expansion, diffuse bilateral reticulogranular opacities, and air bronchograms, findings consistent with severe RDS





Repeat radiograph, obtained 6 hours after endotracheal administration of one dose of surfactant, reveals marked improvement in lung aeration and vascular definition.

Agrons G A et al. Radiographics 2005;25:1047-1073

Inspiration & Expiration

Intrapulmonary - inside the lungs
Intrapleural - inside the space between the lungs and the chest wall.

At rest, intrapulmonary pressure = atmospheric pressure.

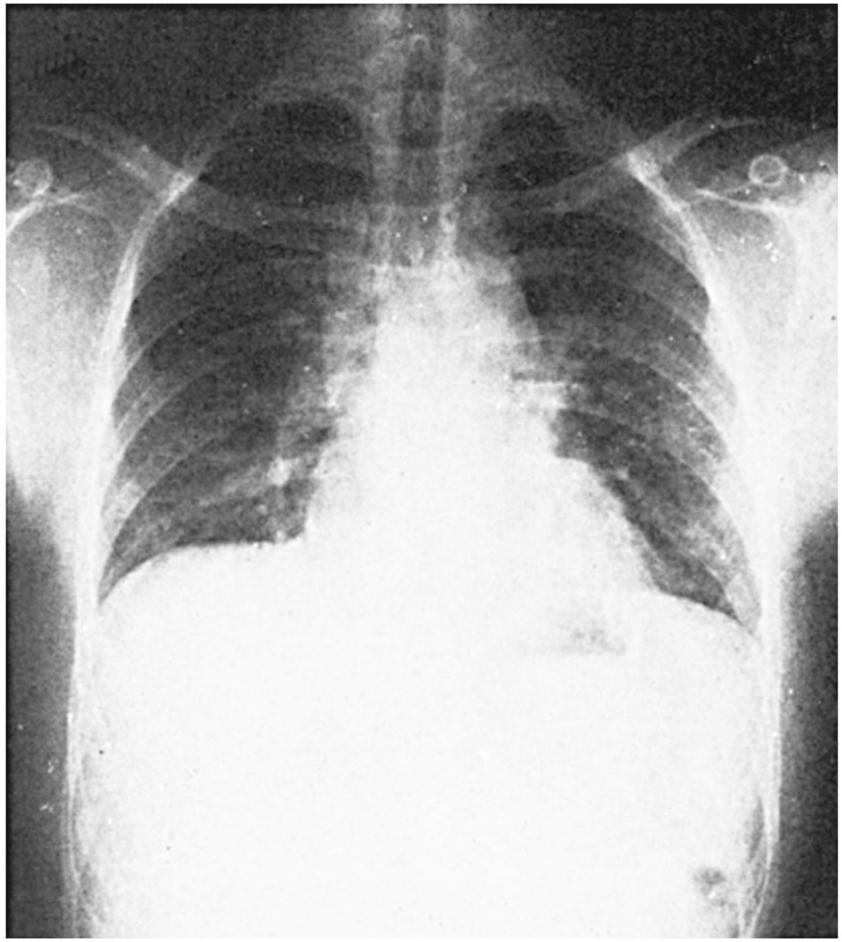
Intrapleural pressure less than atmospheric pressure, so lungs kept inflated up against the inside of chest wall.

Boyle's Law - pressue drops as volume increases; so increase in lung voume decreases intrapulmonary pressure -> sucks air into lungs

Lowering diaphragm causes drop in intrapulmonary pressure to less than atmospheric.

Raising diaphragm causes increase in intrapulmonary pressure, forces air out.

Figure 16.12a

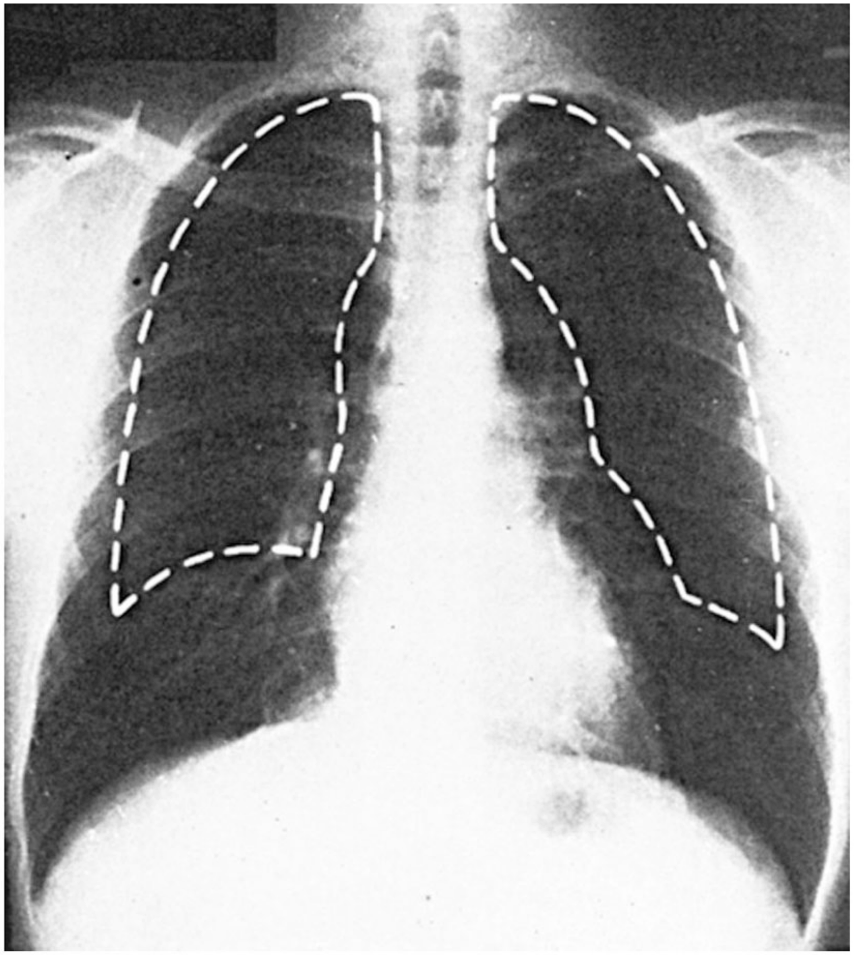


(a)

Expiration

From J.H. Comroe, Jr., Physiology of Respiration: An Introductory Text, 2nd editon, 1974

Figure 16.12b

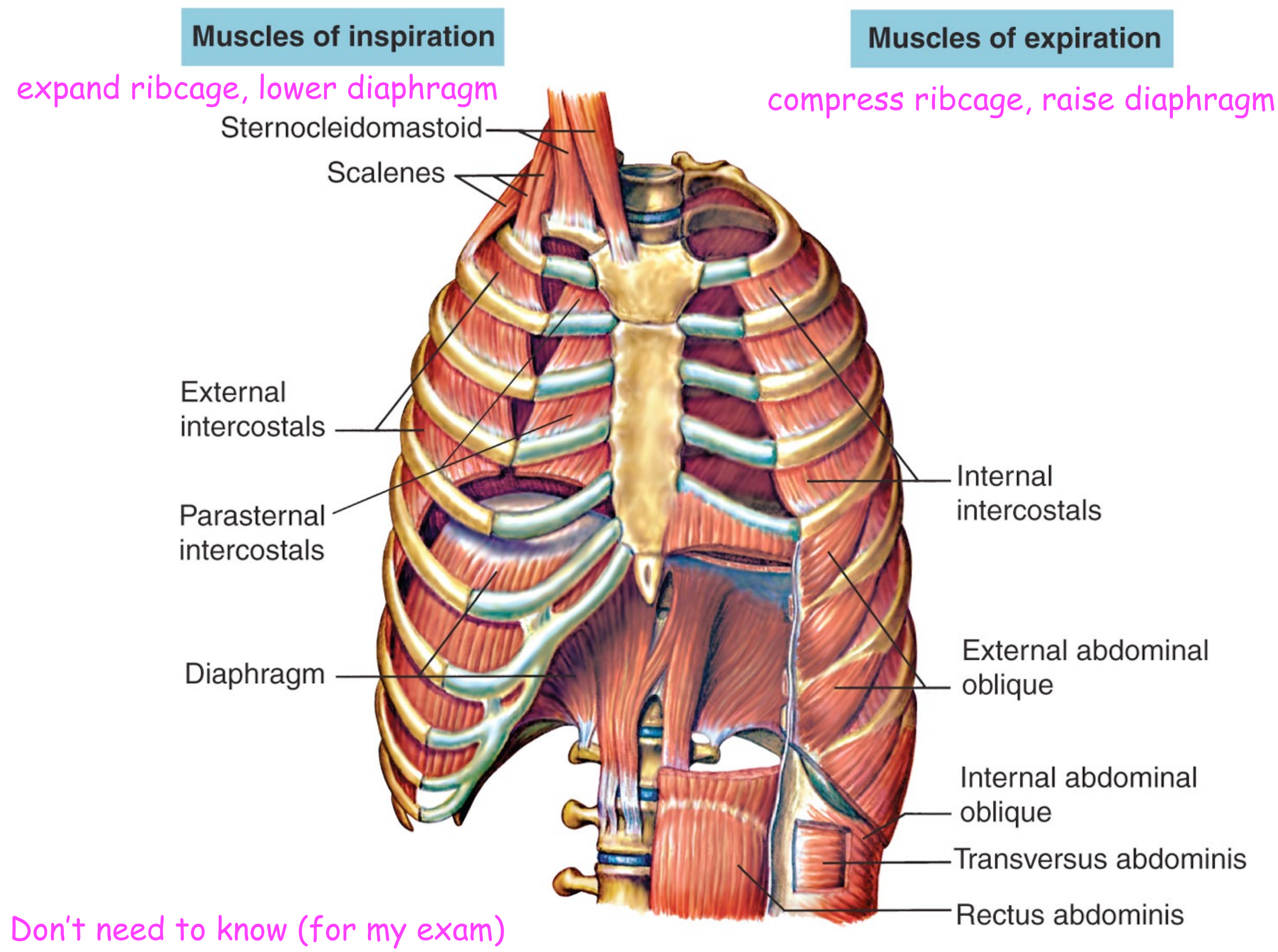


(b)

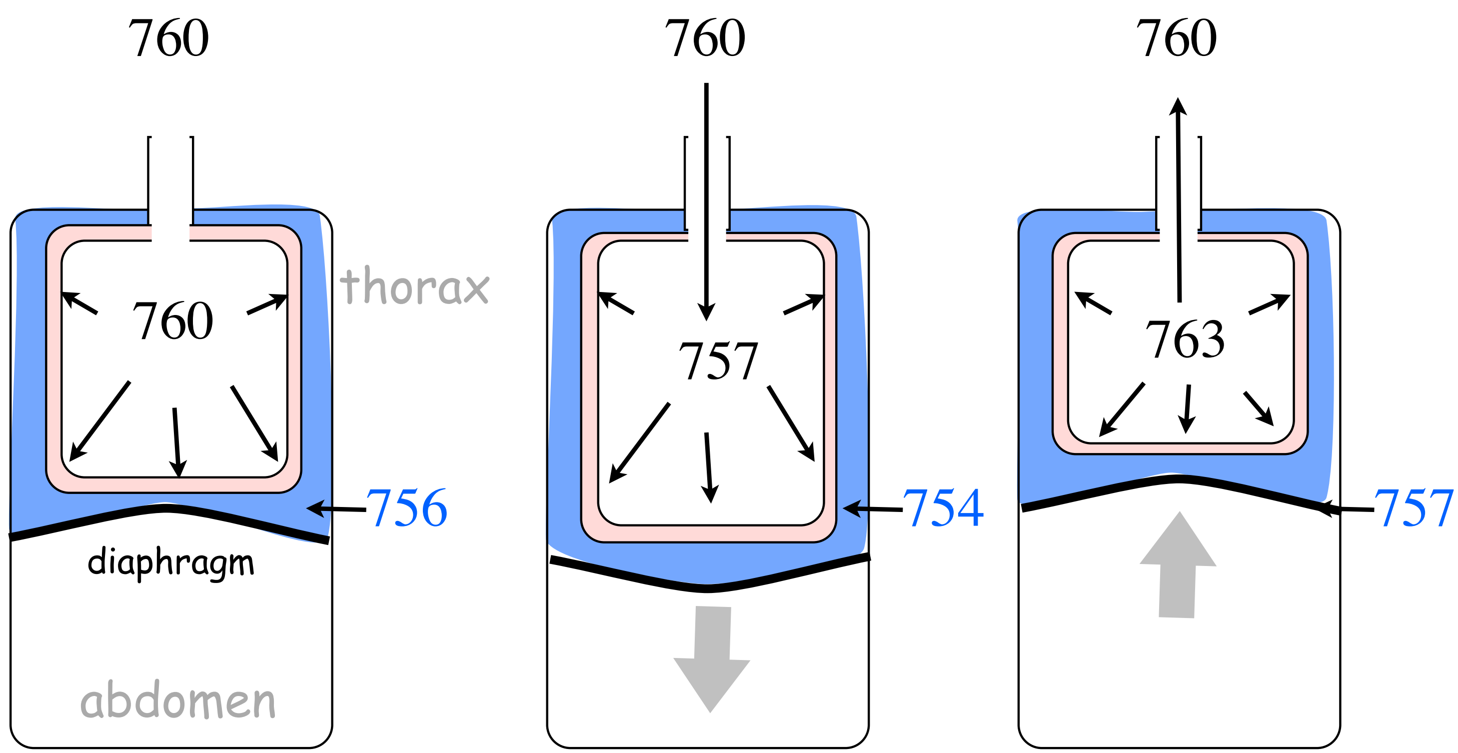
Inspiration

From J.H. Comroe, Jr., Physiology of Respiration: An Introductory Text, 2nd editon, 1974

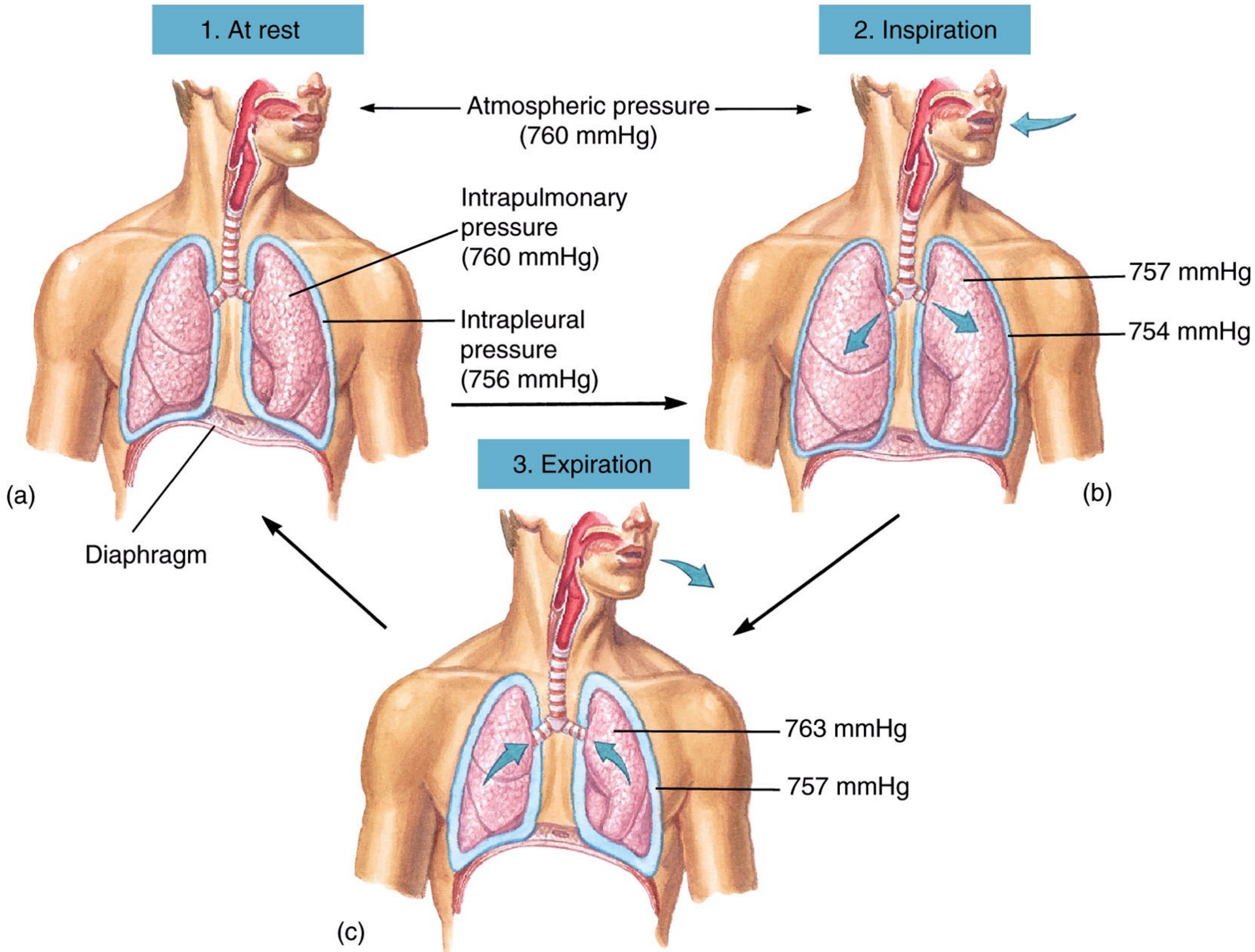
Figure 16.13


Muscles of inspiration
expand ribcage, lower diaphragm
Sternocleidomastoid
Scalenes
External intercostals
Parasternal intercostals
Diaphragm
Muscles of expiration
compress ribcage, raise diaphragm
Internal intercostals
External abdominal oblique
Internal abdominal oblique
Transversus abdominis
Rectus abdominis
Don't need to know (for my exam)

Pressures on the lungs



what sets up negative pressure in pleural cavity?
venous return, lymphatic drainage

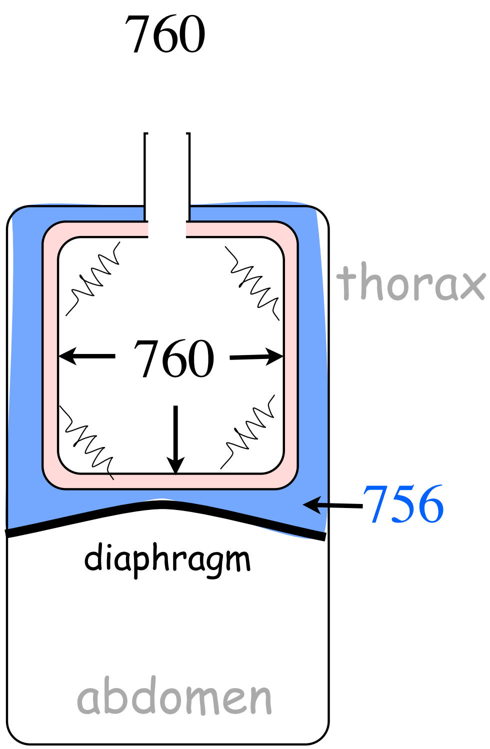


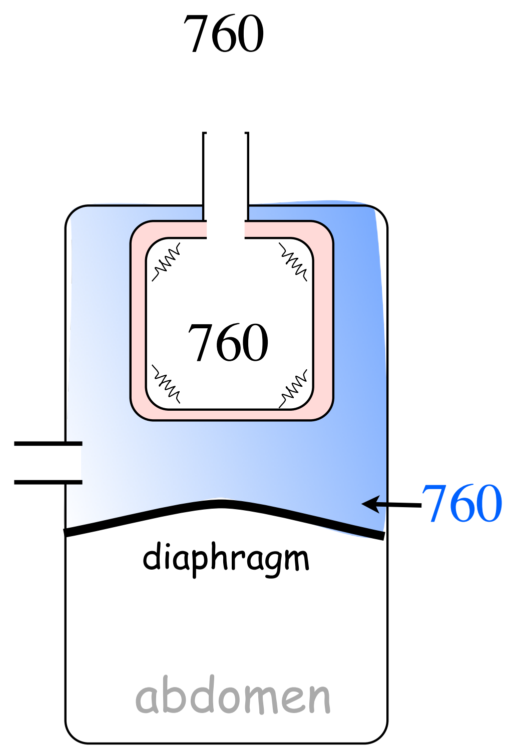
1. At rest
2. Inspiration
Atmospheric pressure
(760 mmHg)
Intrapulmonary
pressure
(760 mmHg)
Intrapleural
pressure
(756 mmHg)
757 mmHg
754 mmHg
3. Expiration
763 mmHg
757 mmHg
(a)
(b)
(c)
Diaphragm

Figure 16.14

Intrapleural pressure keeps elastic lung inflated.

Pneumothorax: air enters intrapleural space, lung collapses
(also, because lung no longer pulled against diaphragm, can't pull air in during inspiration.)





Pneumothorax - “air chest”

air enters intrapleural space
and lung collapses

more air on right (so darker),
more blood on left

Treatment: insert catheter
(small tube) and suck air out
of intrapleural space.

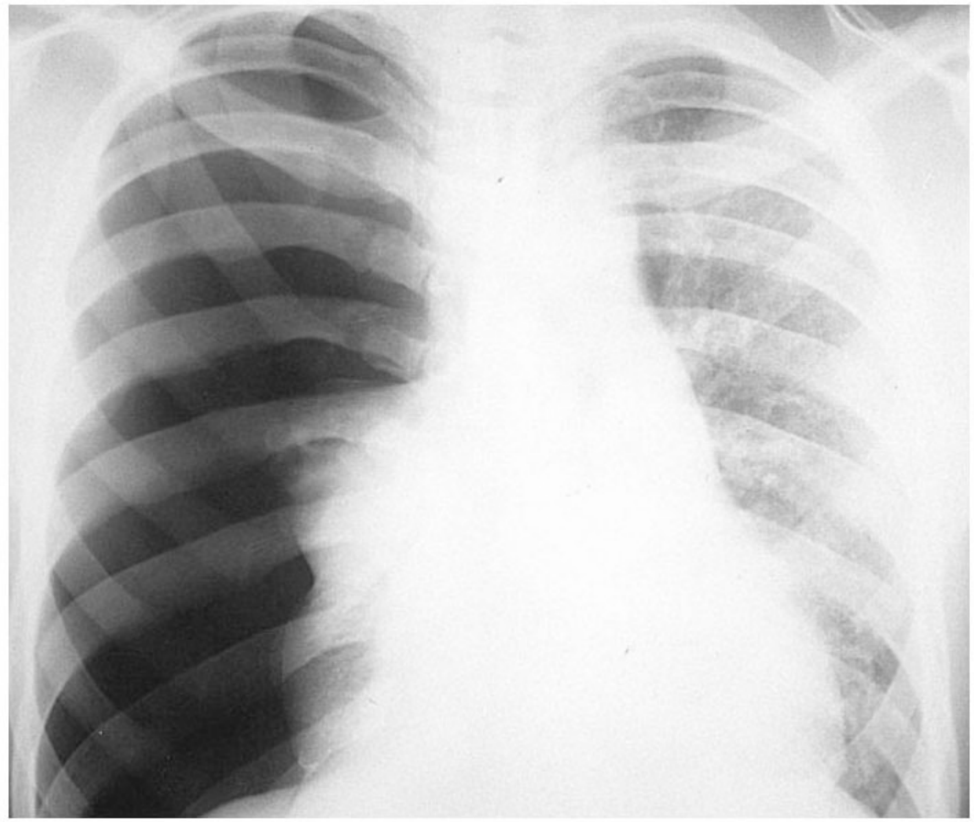


Figure 16.9

Pneumothorax - “air chest”

pre-term infant with pneumothorax & correction with catheter to remove air from pleural space.

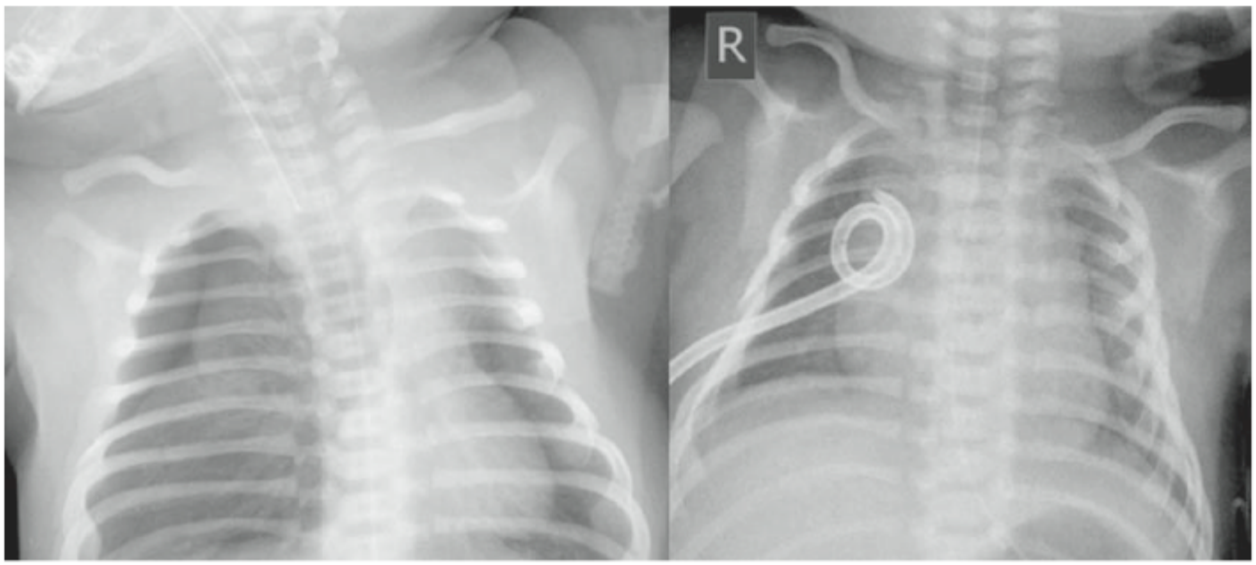


Figure 3 (A) Right sided pneumothorax. (B) Insertion of pigtail catheter showing resolution of the pneumothorax.

Miall L , Wallis S Arch Dis Child Educ Pract Ed doi:10.1136/ adc.2010.189712

Algorithm for the management of respiratory distress in the moderately preterm infant in the newborn period.

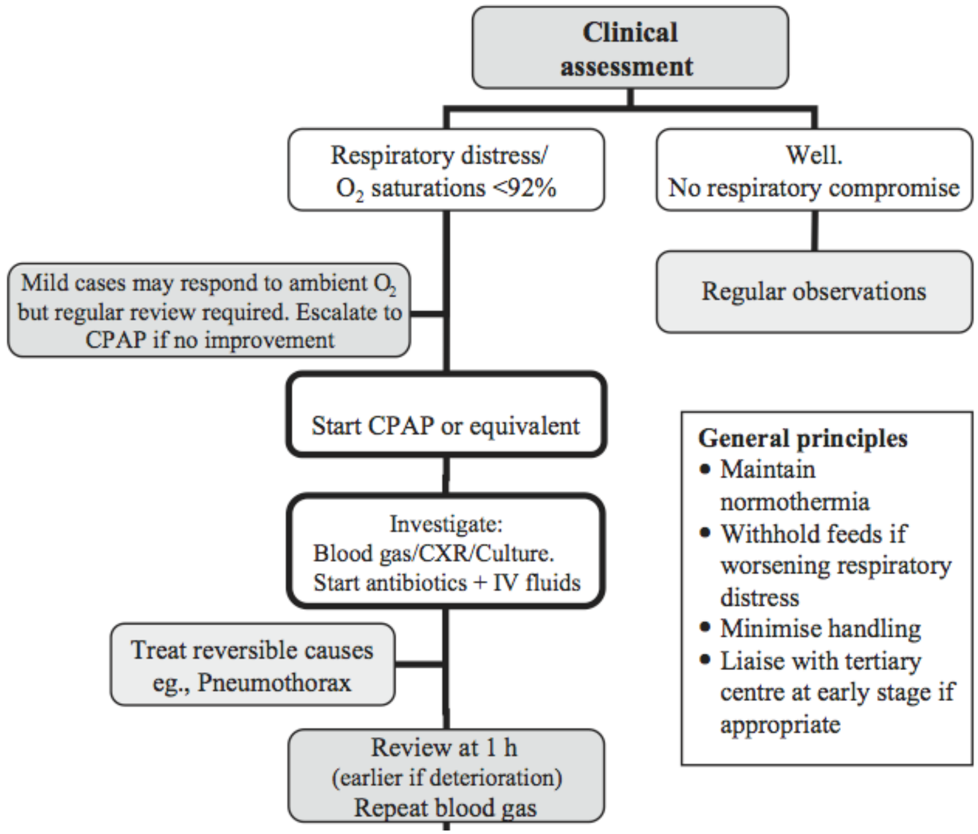


Figure 4 Algorithm for the management of respiratory distress in the moderately preterm infant in the newborn period. CPAP, continuous positive airflow pressure; CXR, chest x-ray; RDS, respiratory distress syndrome.

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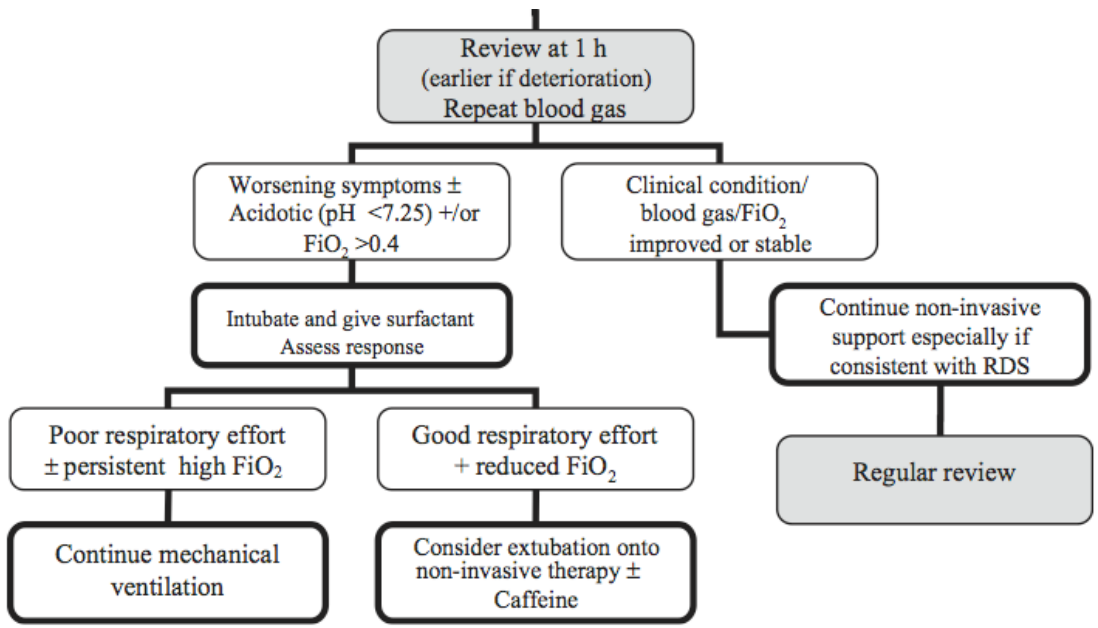

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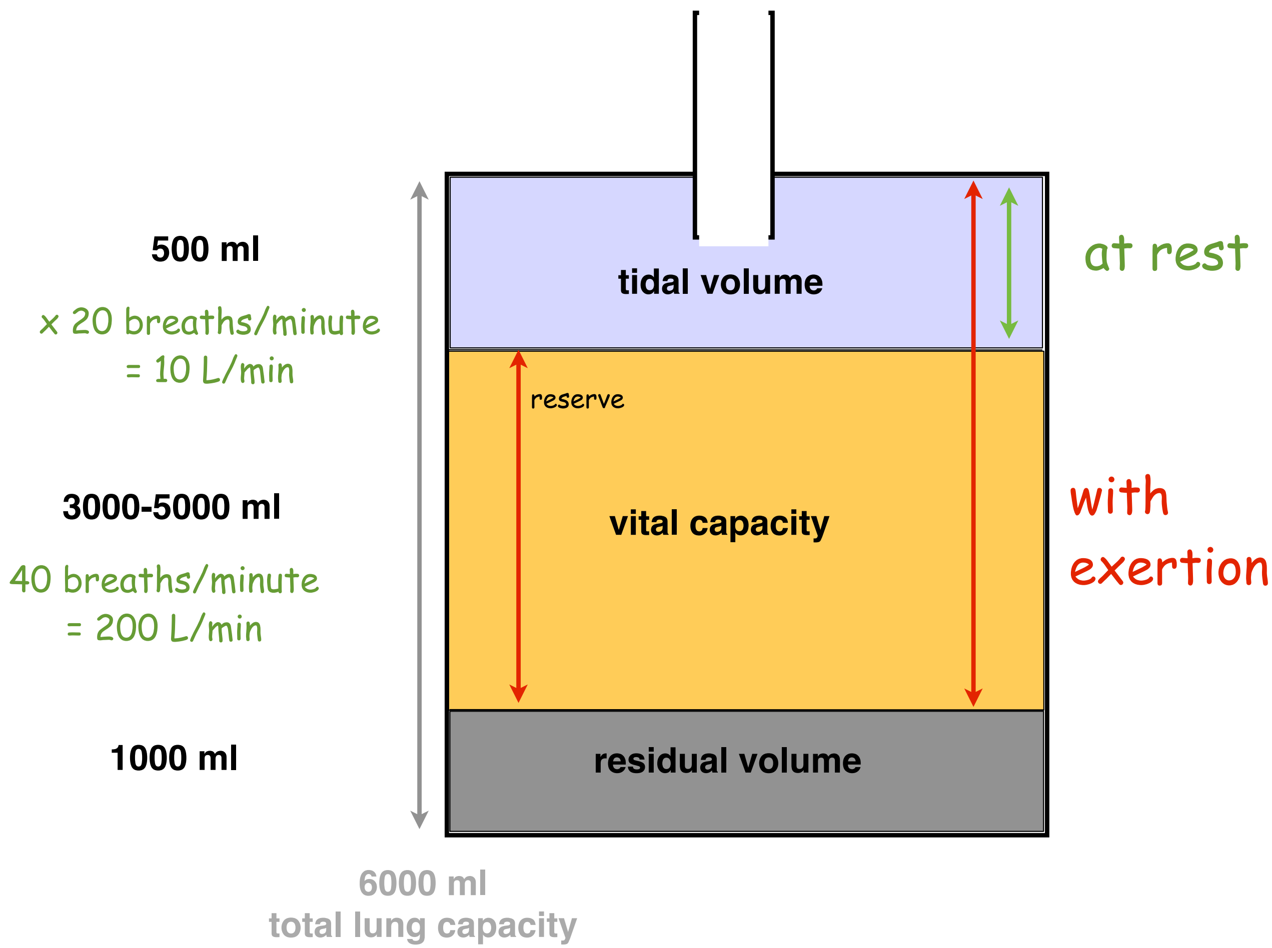
Miall L , Wallis S Arch Dis Child Educ Pract Ed doi:10.1136/adc.2010.189712

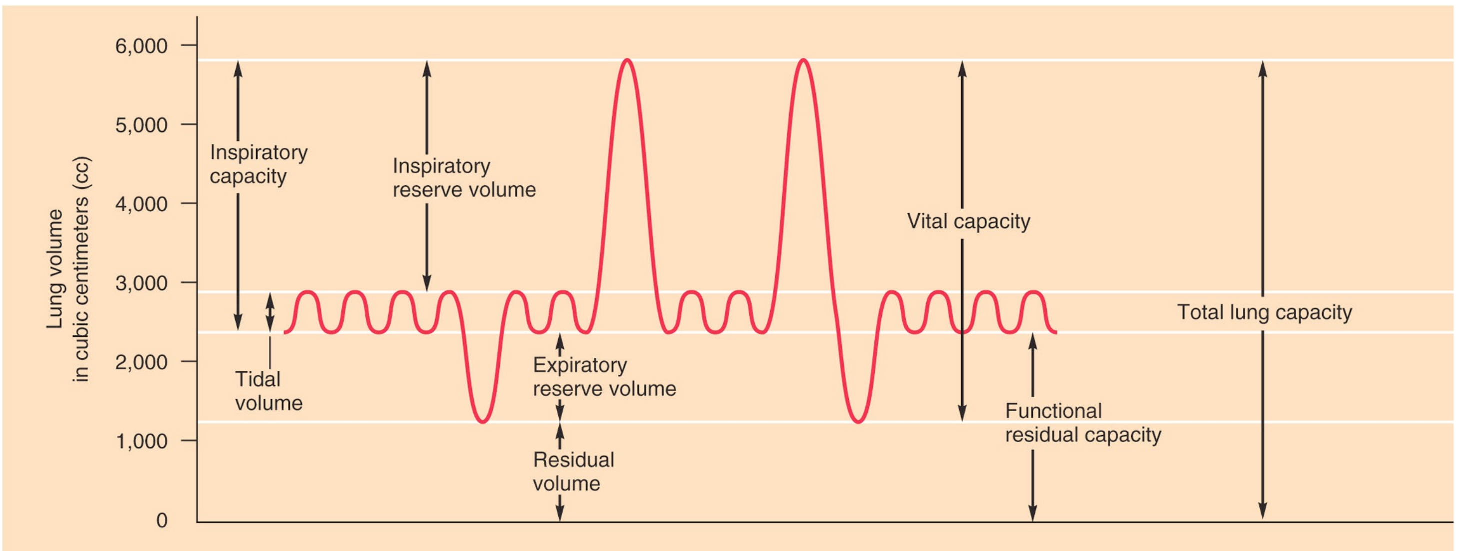
Lung Volumes & Capacities

Total Lung Capacity - gas in lungs after maximum expansion
Tidal Volume - gas breathed in and out at rest
Vital Capacity - gas breathed in and out at maximum inspiration
Residual Volume - gas left in lungs after maximum expiration

Table 16.3 | **Terms Used to Describe Lung Volumes and Capacities**

Term	Definition
Lung Volumes	The four nonoverlapping components of the total lung capacity
Tidal volume	The volume of gas inspired or expired in an unforced respiratory cycle
Inspiratory reserve volume	The maximum volume of gas that can be inspired during forced breathing in addition to tidal volume
Expiratory reserve volume	The maximum volume of gas that can be expired during forced breathing in addition to tidal volume
Residual volume	The volume of gas remaining in the lungs after a maximum expiration
Lung Capacities	Measurements that are the sum of two or more lung volumes
Total lung capacity	The total amount of gas in the lungs after a maximum inspiration
Vital capacity	The maximum amount of gas that can be expired after a maximum inspiration
Inspiratory capacity	The maximum amount of gas that can be inspired after a normal tidal expiration
Functional residual capacity	The amount of gas remaining in the lungs after a normal tidal expiration


500 ml
x 20 breaths/minute
= 10 L/min
3000-5000 ml
40 breaths/minute
= 200 L/min
1000 ml
tidal volume
reserve
vital capacity
residual volume
at rest
with exertion
6000 ml
total lung capacity



Lung volume
in cubic centimeters (cc)
6,000
5,000
4,000
3,000
2,000
1,000
0
Inspiratory
capacity
Tidal
volume
Inspiratory
reserve volume
Expiratory
reserve volume
Residual
volume
Vital capacity
Functional
residual capacity
Total lung capacity

Figure 16.15

Disease state (like emphysema)

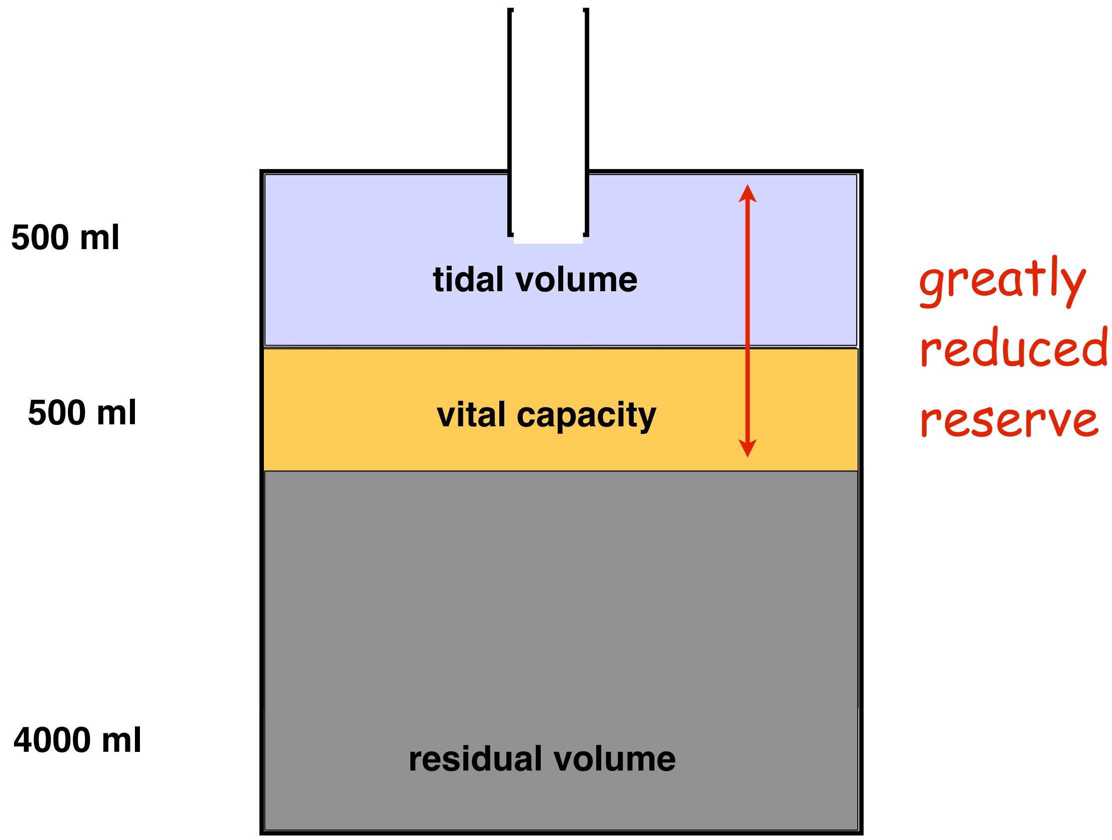
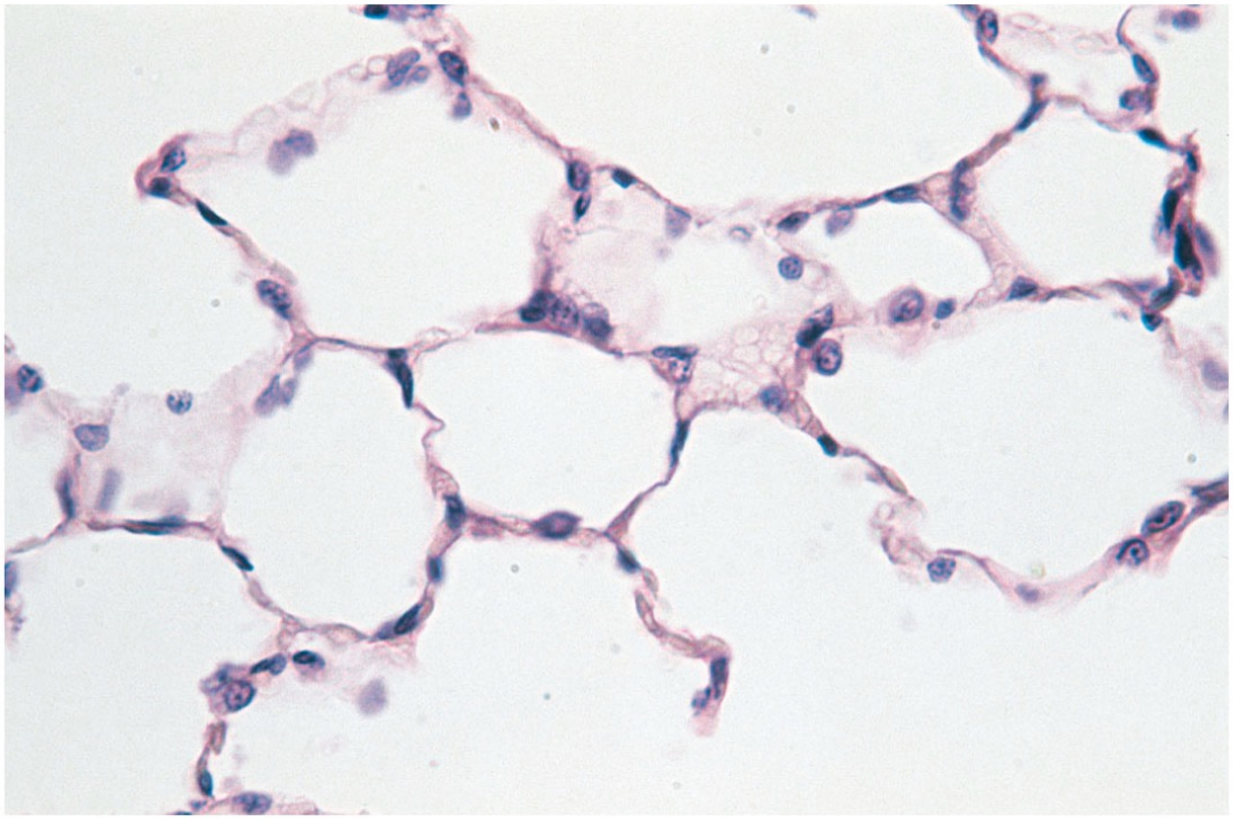


Figure 16.17a

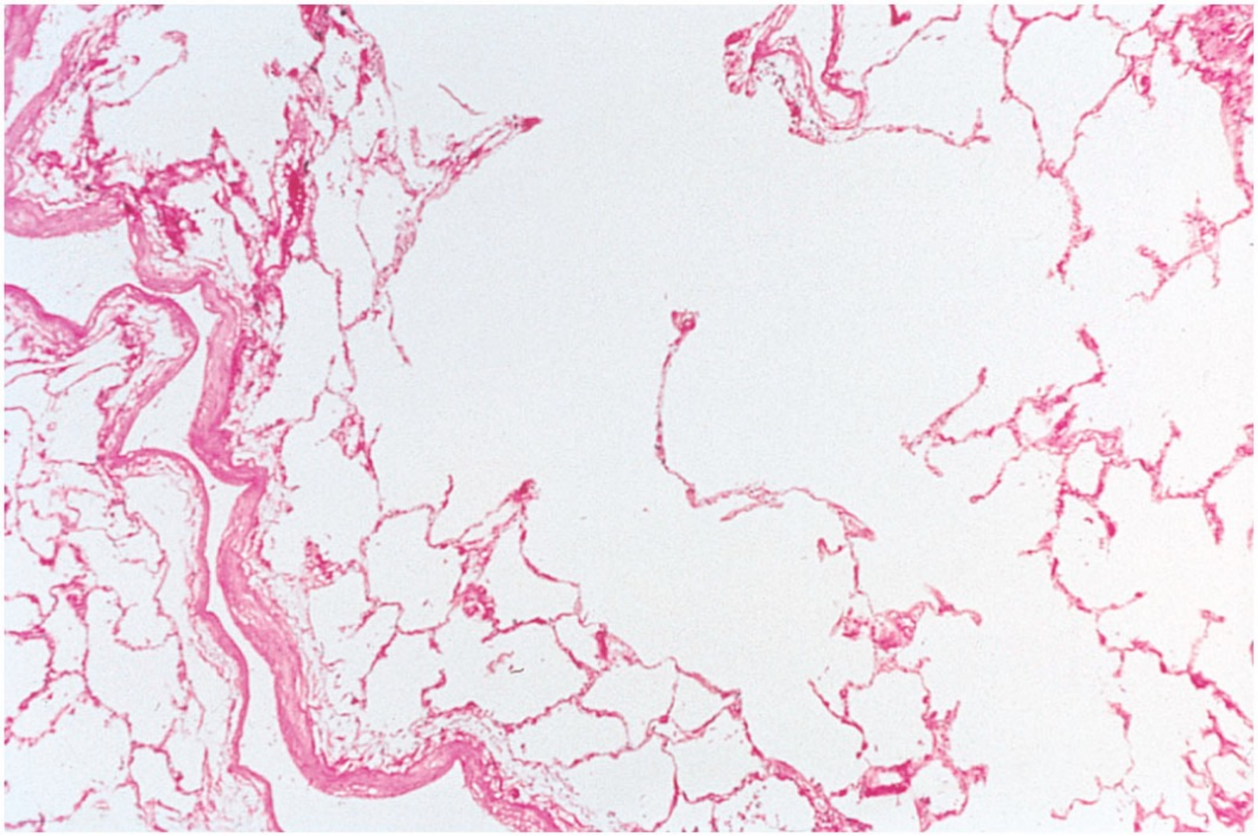
(a)



Normal Lung

Figure 16.17b

(b)



Lung w/ Emphysema

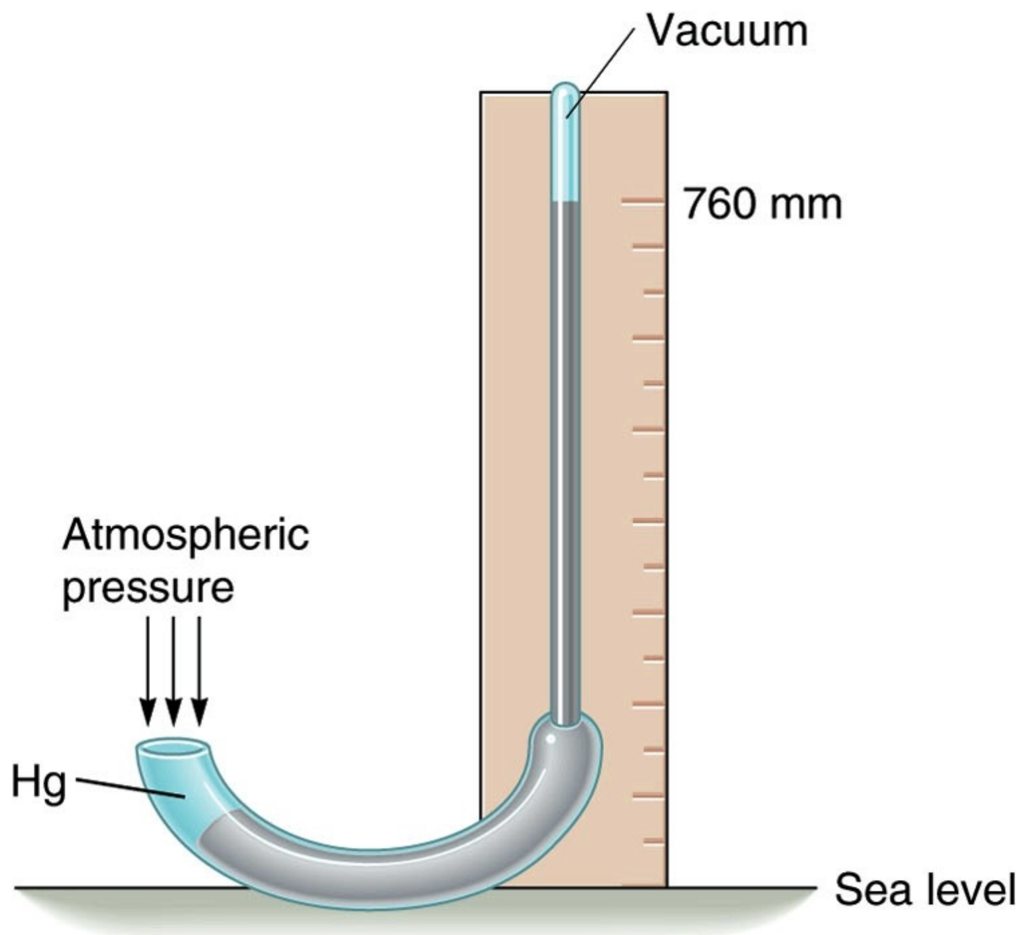


Figure 16.18

	Inspired air	Alveolar air
H_2O	Variable	47 mmHg
CO_2	000.3 mmHg	40 mmHg
O_2	159 mmHg	105 mmHg
N_2	601 mmHg	568 mmHg
Total pressure	760 mmHg	760 mmHg

Figure 16.19

Measure P_{O2} with oxygen sensitive electrode

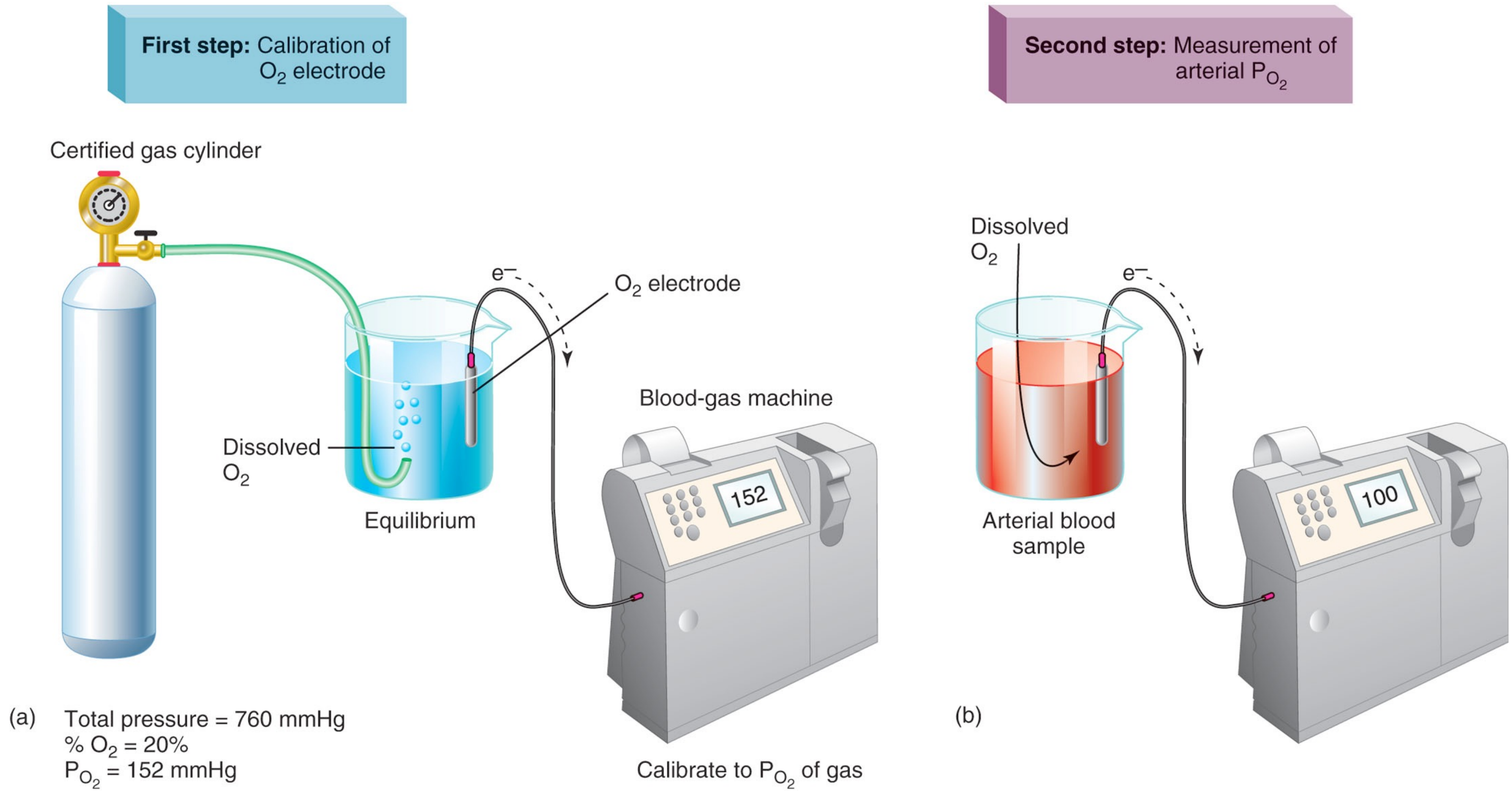


Figure 16.21

Partial Pressure of O_2 (P_{O2}) in air, lungs and blood

Control of Breathing

Role of CO_2 / carbonic acid / bicarbonate and pH

Hemoglobin and Hemoglobin Dissociation Curves

Measuring Gas Concentration in Air & Blood

Pressure measured in mmHg. At sea level, atmosphere - 760 mmHg.

Gas Concentration is measured as **partial pressure** = fraction of total pressure exerted by particular gas.

for example:

atmospheric pressure is **760 mmHg**
O_2 is 20% of atmosphere
P_{O2} = 0.20 x 760 = **152 mmHg**

Partial pressure determines how much O2 dissolves in alveolar fluid & diffuses into blood.

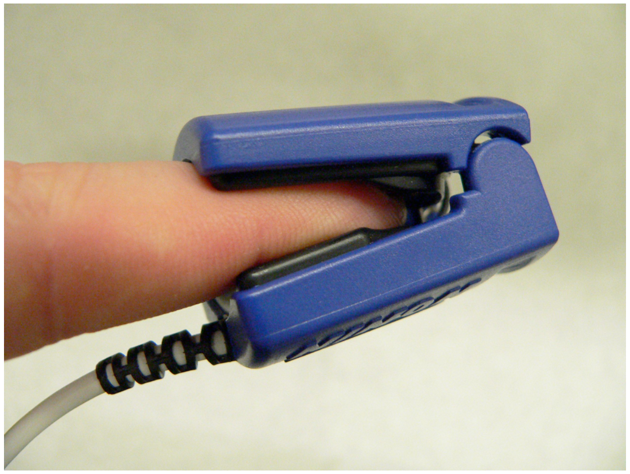
O_2 diffuses from higher P_{O2} to lower P_{O2}

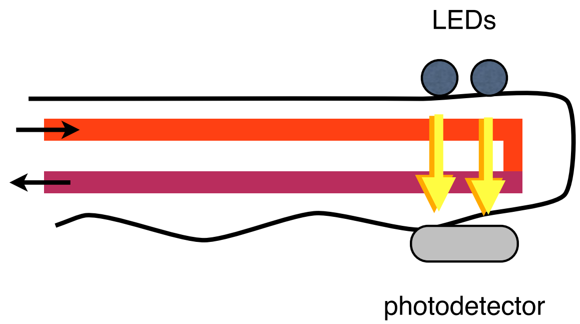
Because O_2 is leaving lungs into blood, and CO_2 is entering lungs from blood, P_{O2} is lower in lungs than in atmosphere, and P_{CO2} is higher in lungs than in blood.

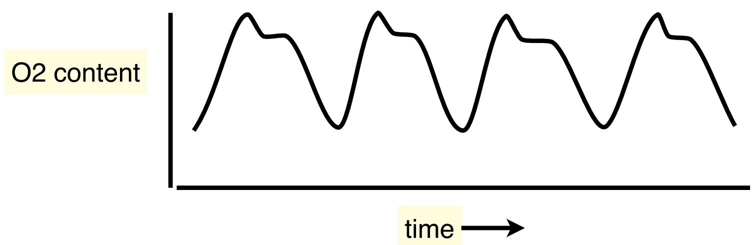
Gas concentration in blood is also measured in partial pressure = pressure required to dissolve that much of the gas in the blood.

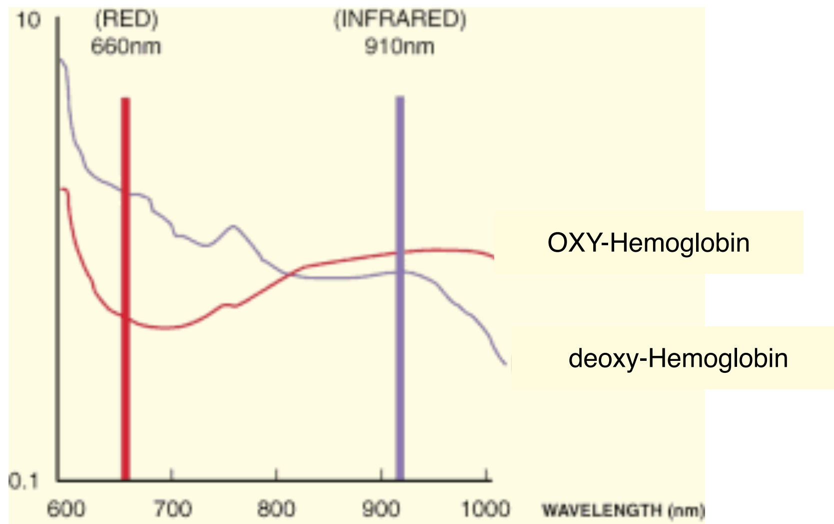
P_{O2} is high in blood leaving lungs, P_{O2} low in blood leaving tissue.
P_{CO2} is low in blood leaving lungs, P_{CO2} is high in blood leaving tissure.

Pulse Oximetry









https://www.youtube.com/watch?v=4pZZ5AEEmek

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Skin colour affects the accuracy of medical oxygen sensors

COVID-19 broadened the use of pulse oximeters for rapid blood-oxygen readings, but it also highlighted the fact that skin pigmentation alters measurements. Two groups of researchers analyse this issue, and its effects on people with dark skin.

Matthew D. Keller, Brandon Harrison-Smith, Chetan Patil & Mohammed Shahriar Arefin

MATTHEW D. KELLER & BRANDON HARRISON-SMITH: Pulse-oximetry errors affect patient outcomes

The pulse oximeter is a device that estimates a person's oxygen saturation level, a measure of the oxygen concentration in their blood, by shining light through their tissue, typically a fingertip or an earlobe (Fig. 1). As highlighted by the COVID-19 pandemic, accurate pulse-

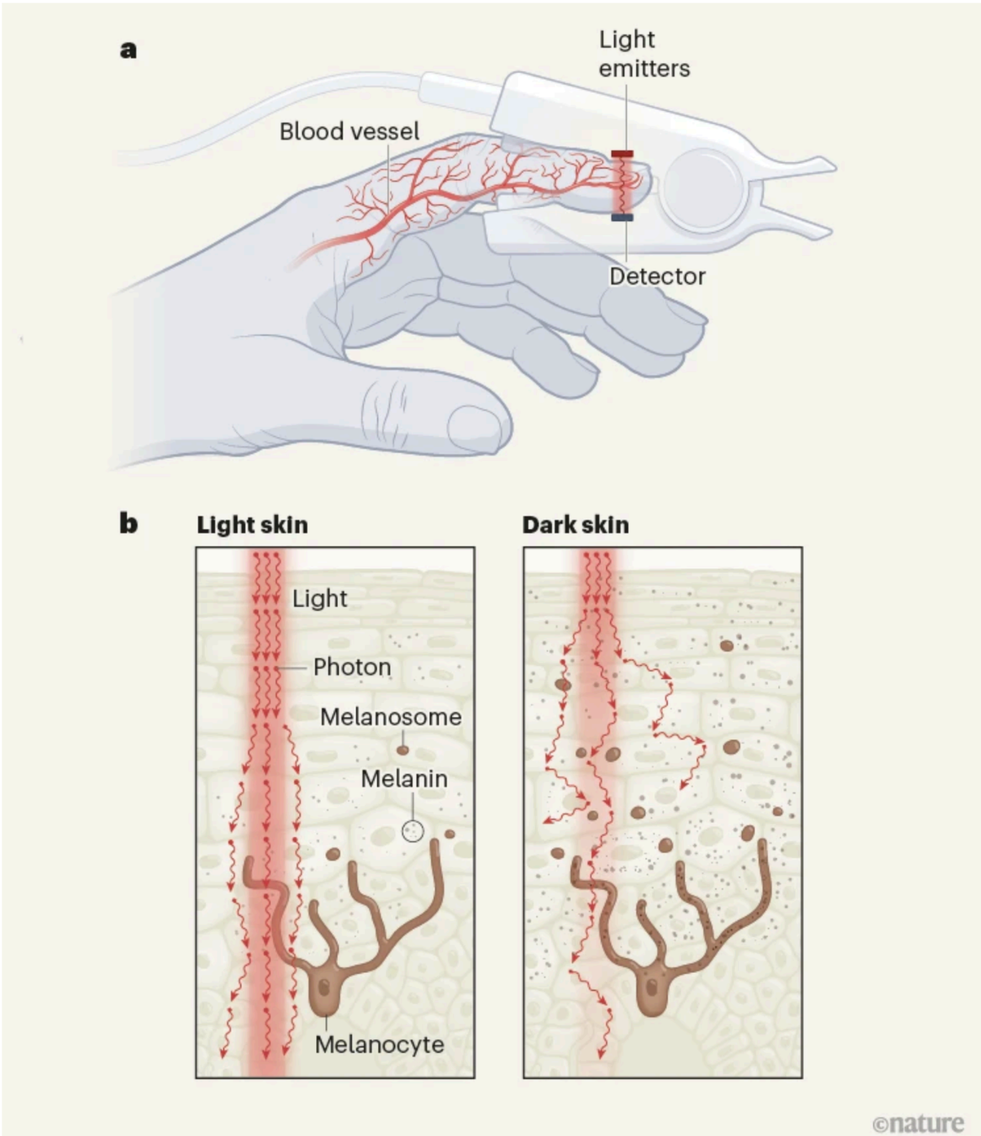
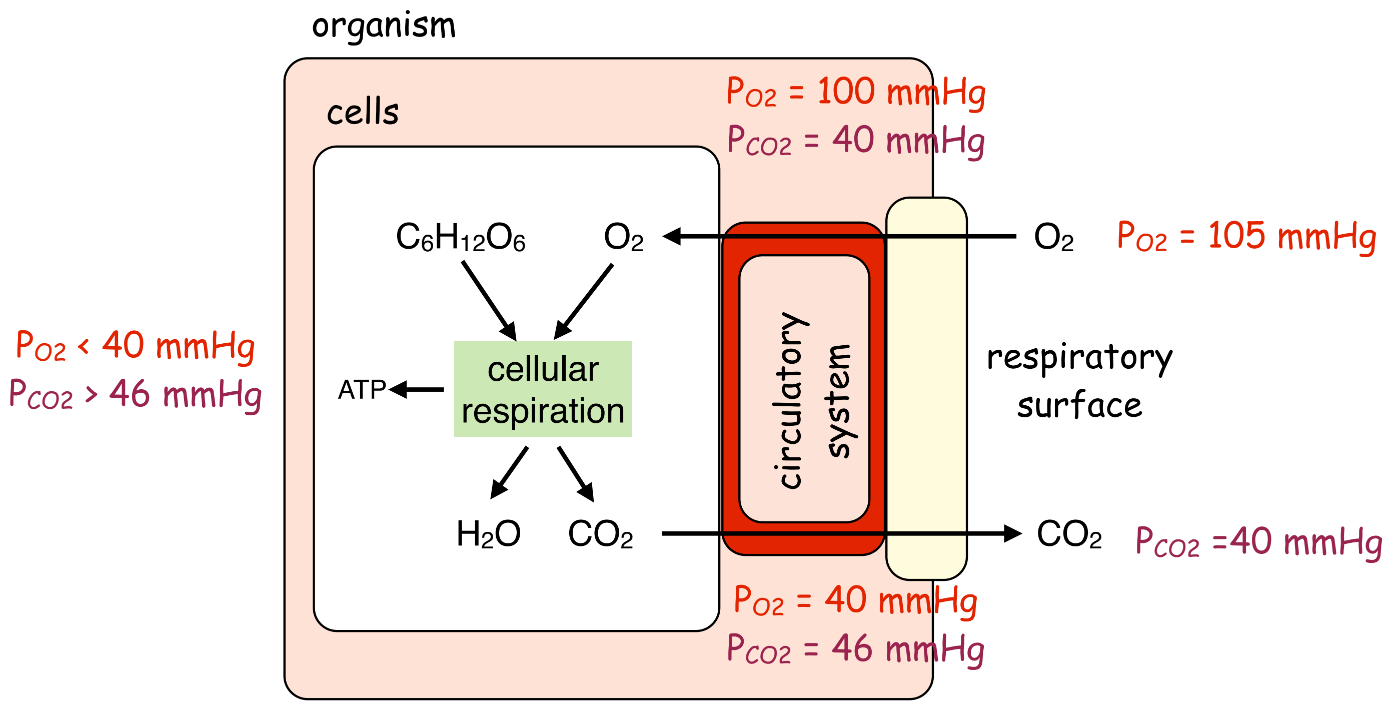


Figure 1 | Pulse-oximetry accuracy varies with skin tone. a, Devices known as pulse oximeters estimate the oxygen concentration in a person's blood by shining red and infrared light through their fingertip. Oxygenated haemoglobin absorbs infrared light more efficiently than it does red light, whereas the opposite is true for deoxygenated haemoglobin. **b**, These signals are affected by melanin, which is distributed through the skin in structures, known as melanosomes, that are produced by cells called melanocytes. Melanosomes in dark skin are both larger and more numerous than are those in light skin. Long-standing oximetry theory does not fully account for the way in which photons are scattered by the biomolecular content and structure of the tissue, and thus imprecisely corrects for the effect of pigmentation.

Driven by clinical experiences early in the pandemic, Sjoding *et al.*[1] published a retrospective report showing that pulse oximeters overestimate the true oxygen saturation of Black people. This inaccuracy means that diagnoses of hypoxaemia, the condition of having low levels of oxygen in one's blood, are approximately three times more likely to be missed in Black patients than in white patients. Misdiagnosed patients are said to have occult hypoxaemia when arterial blood-gas tests indicate oxygen saturation levels of less than 88% (signalling hypoxaemia), despite pulse oximeters measuring a healthy oxygenation of more than 92%.

Respiration:

Get O_2 from outside environment into deep tissues;
get CO_2 out of tissues



Cellular Respiration:
O_2 used by tissues in oxidative phosphorylation;
CO_2 produced as waste product by glycolysis.

0.36 g = 250 ml of oxygen / min

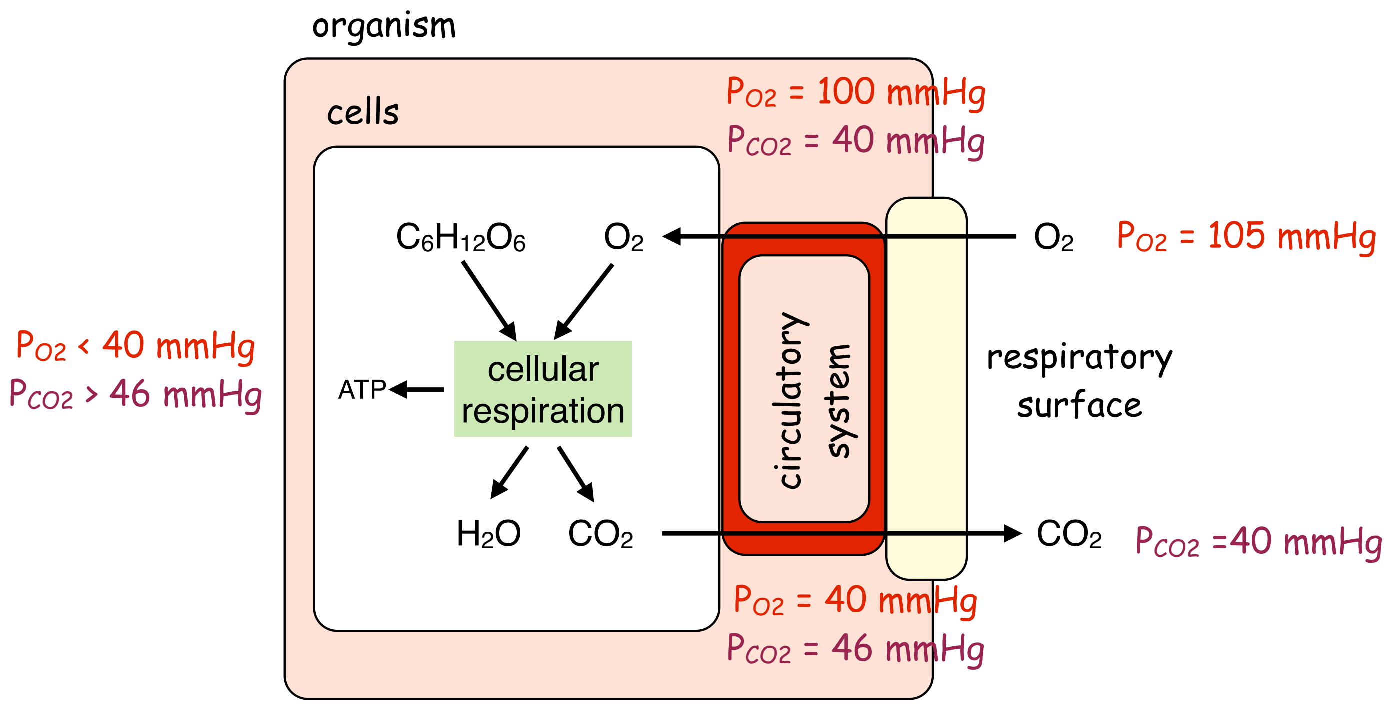
0.5 g = 250 ml of carbon dioxide / min

When a person loses weight (fat mass), where does the fat go?

top hat

Respiration:

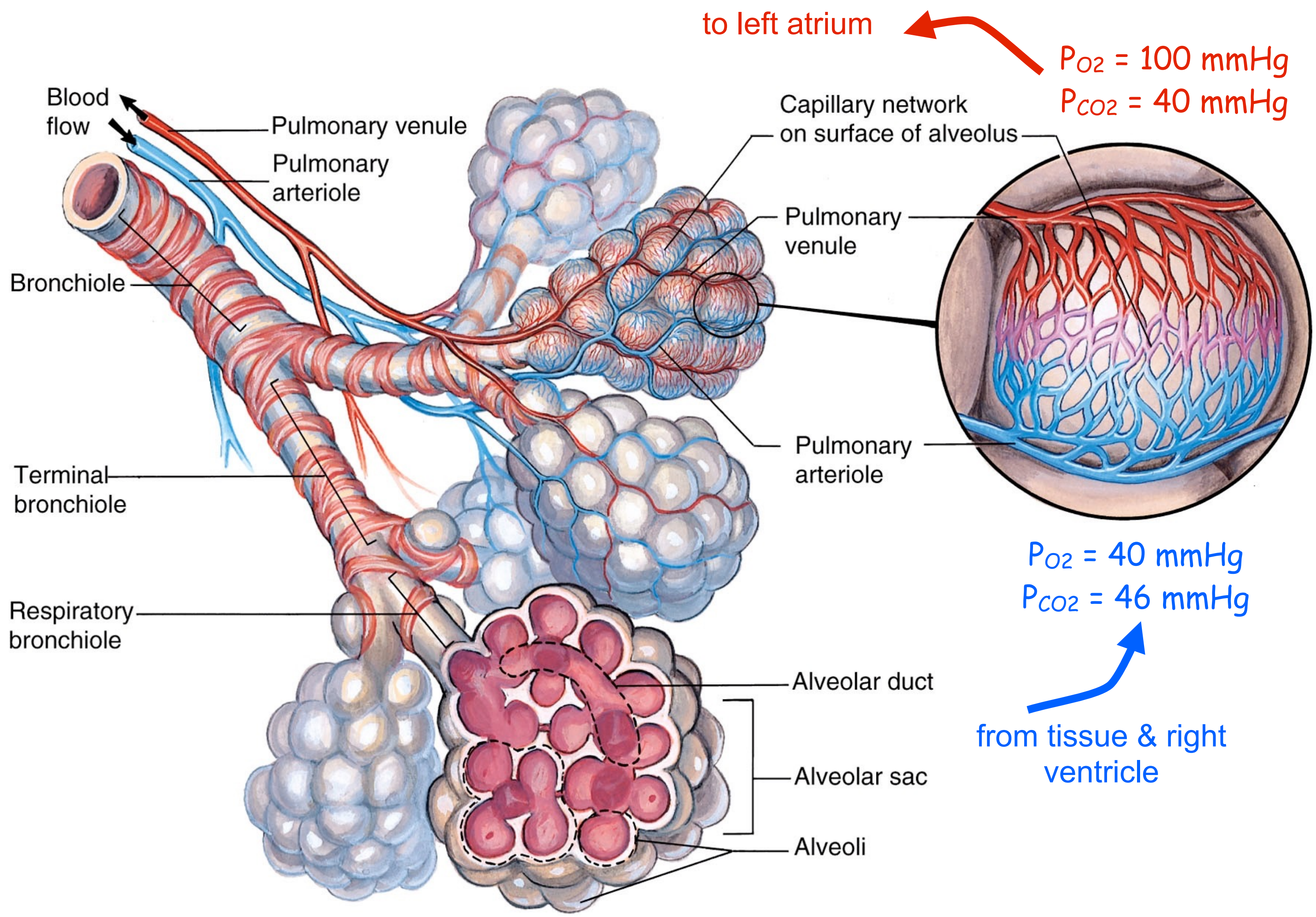
Get O_2 from outside environment into deep tissues;
get CO_2 out of tissues



Cellular Respiration:
O_2 used by tissues in oxidative phosphorylation;
CO_2 produced as waste product by glycolysis.

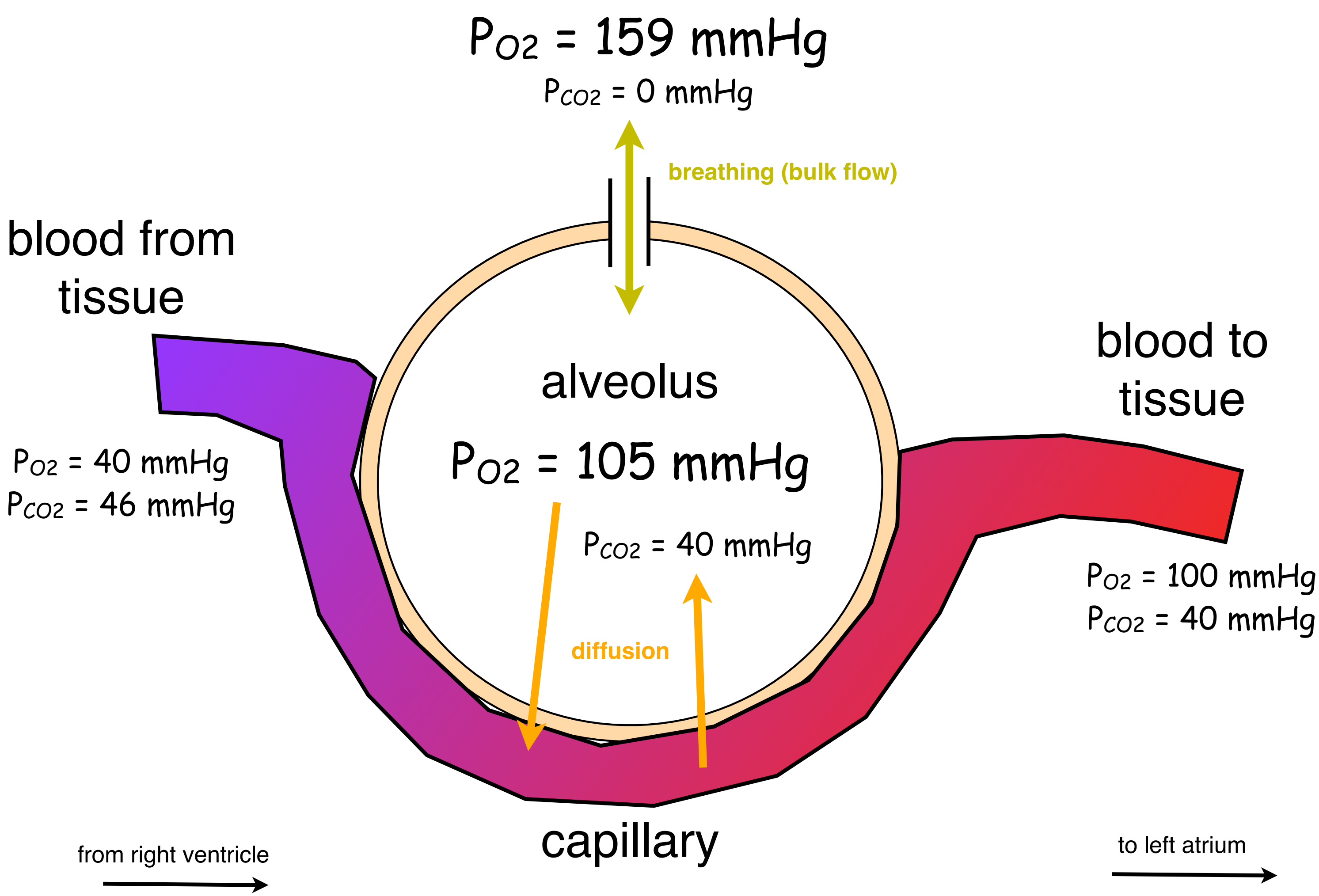
0.36 g = 250 ml of oxygen / min

0.5 g = 250 ml of carbon dioxide / min



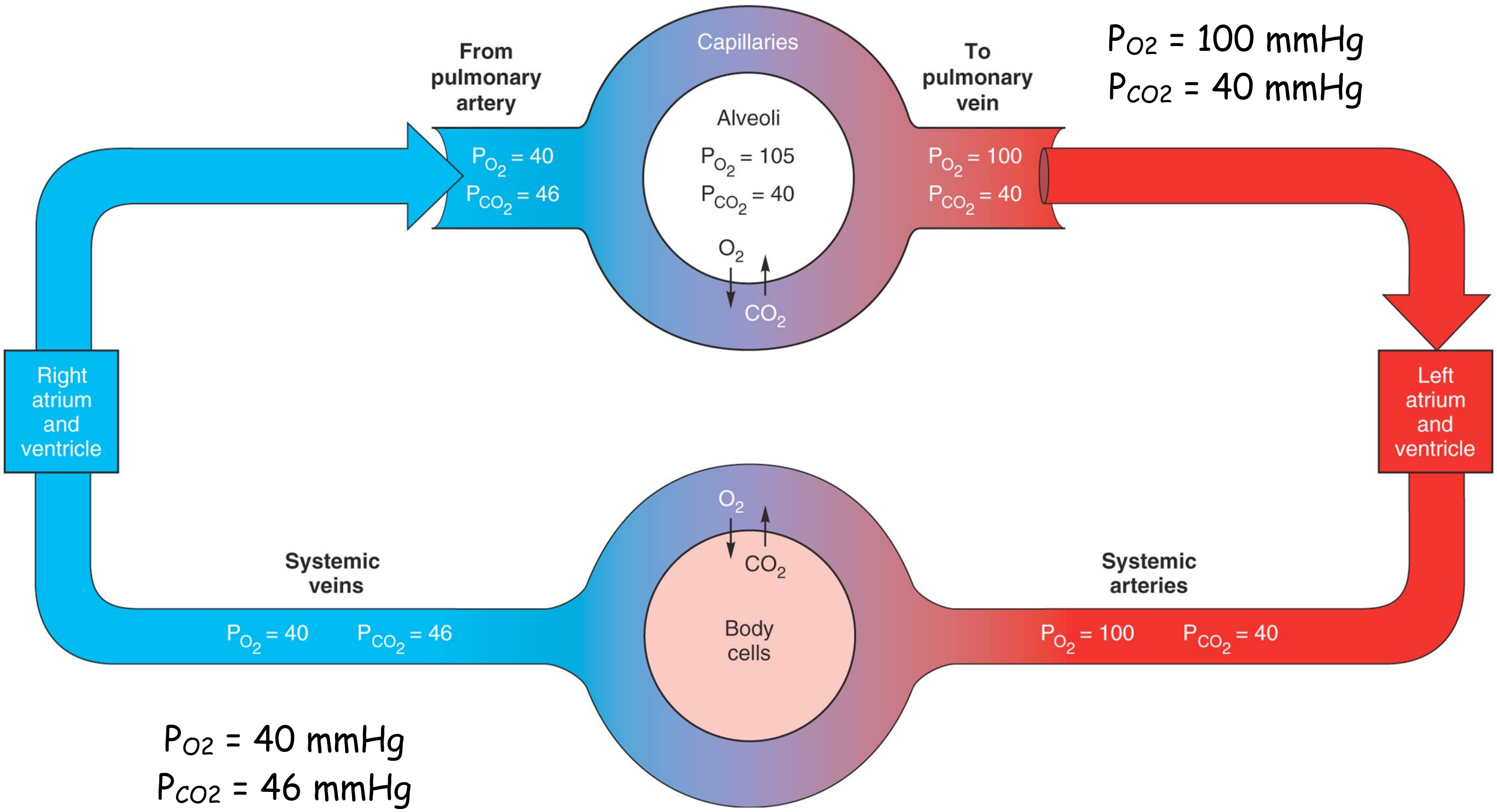
to left atrium
P_{O2} = 100 mmHg
P_{CO2} = 40 mmHg
Blood flow
Pulmonary venule
Pulmonary arteriole
Capillary network on surface of alveolus
Pulmonary venule
Bronchiole
Pulmonary arteriole
Terminal bronchiole
Respiratory bronchiole
P_{O2} = 40 mmHg
P_{CO2} = 46 mmHg
Alveolar duct
from tissue & right ventricle
Alveolar sac
Alveoli

Figure 16.20



P_{O2} = 159 mmHg
P_{CO2} = 0 mmHg
breathing (bulk flow)
blood from tissue
P_{O2} = 40 mmHg
P_{CO2} = 46 mmHg
alveolus
P_{O2} = 105 mmHg
P_{CO2} = 40 mmHg
diffusion
blood to tissue
P_{O2} = 100 mmHg
P_{CO2} = 40 mmHg
capillary
from right ventricle
to left atrium

Figure 16.22



From pulmonary artery
P_{O_2} = 40
P_{CO_2} = 46
Capillaries
Alveoli
P_{O_2} = 105
P_{CO_2} = 40
O_2
CO_2
To pulmonary vein
P_{O_2} = 100
P_{CO_2} = 40
P_{O2} = 100 mmHg
P_{CO2} = 40 mmHg
Right atrium and ventricle
Left atrium and ventricle
O_2
CO_2
Body cells
Systemic veins
P_{O_2} = 40
P_{CO_2} = 46
Systemic arteries
P_{O_2} = 100
P_{CO_2} = 40
P_{O2} = 40 mmHg
P_{CO2} = 46 mmHg

Lower air pressure -> lower P_{O2} -> less diffusion of O_2 into blood

Table 16.5 | Effect of Altitude on Partial Oxygen Pressure (P_{O_2})

Altitude (Feet Above Sea Level)*		Atmospheric Pressure (mmHg)	P_{O_2} in Air (mmHg)	P_{O_2} in Alveoli (mmHg)	P_{O_2} in Arterial Blood (mmHg)
0	Florida	760	159	105	100
2,000		707	148	97	92
4,000		656	137	90	85
6,000	Colorado	609	127	84	79
8,000		564	118	79	74
10,000		523	109	74	69
20,000		349	73	40	35
30,000	Mt Everest	226	47	21	19

*For reference, Pike's Peak (Colorado) is 14,110 feet; Mt. Whitney (California) is 14,505 feet; Mt. Logan (Canada) is 19,524 feet; Mt. McKinley (Alaska) is 20,320 feet; and Mt. Everest (Nepal and Tibet), the tallest mountain in the world, is 29,029 feet.

P_{CO2} in atmosphere always 0, so always good diffusion of CO_2 ***out*** *of blood*

Table 16.5

Control of Breathing

1. **Restful Breathing:**
Rhythmicity area in brainstem sets up rhythm.

Periodic inhalation caused by rhythmic firing of **I motor neurons** -> lowering of diaphragm -> inspiration

Inhalation is terminated by feedback from lung stretch sensors that inhibit the I motor neurons, and excite **E motor neurons** -> expiration.

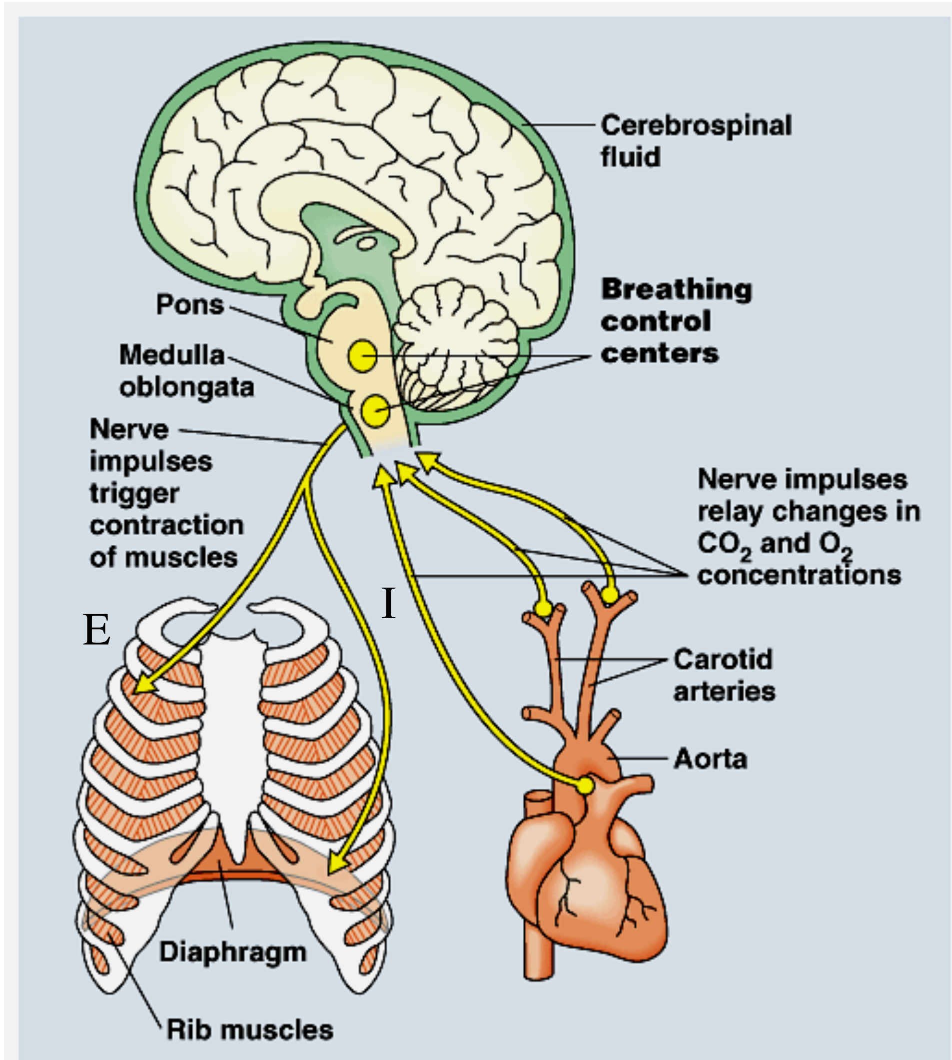
2. **Modulation:**
Rhythmic breathing modulated by:

- 2 centers in pons: **pneumotaxic** (inhibits I) & **apneustic** (sitimulates I)
- Voluntary control from cortex
- **Chemoreceptors** in aorta, carotid body, and brainstem.

If P_{CO2} gets too high, CO_2 -> H^+ HCO_3^- -> lower pH (more acidic).

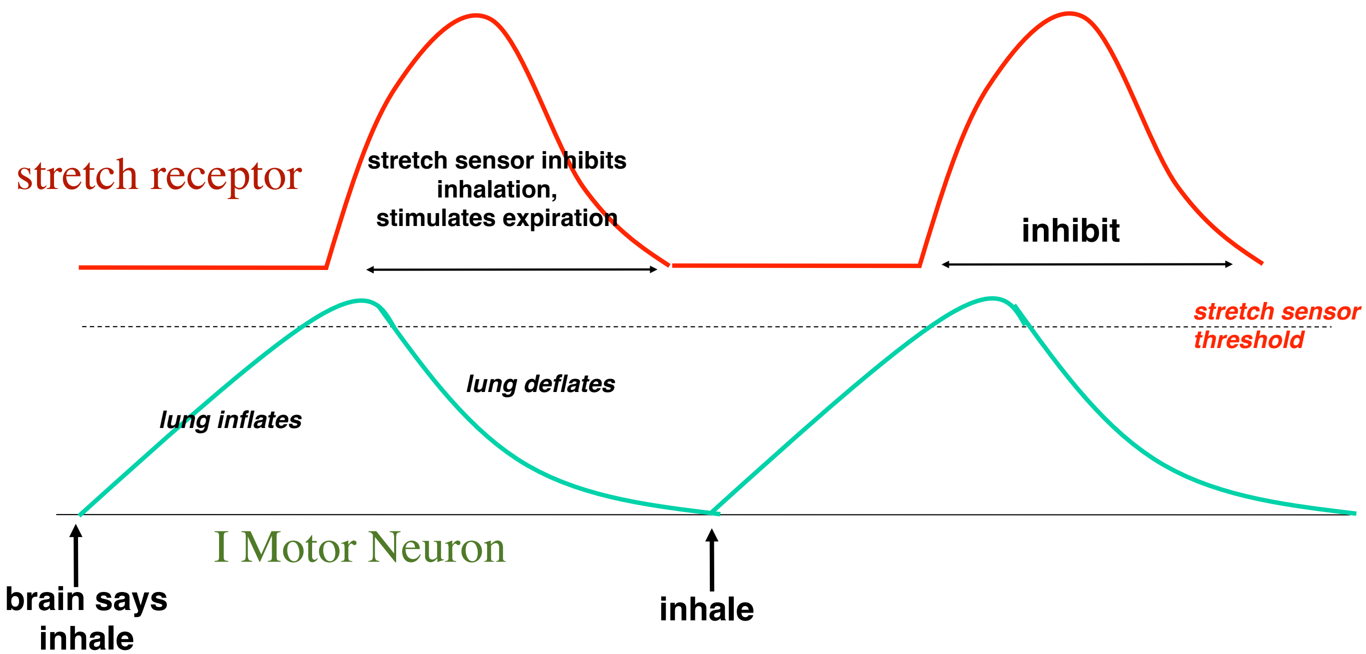
Drop in pH in brain makes respiratory control centers speed up breathing.

Figure 42.26 Automatic control of breathing



Control of Breathing

Control centers in brain set up rhythm:
periodic inhalation that is terminated by feedback from lung stretch sensors.



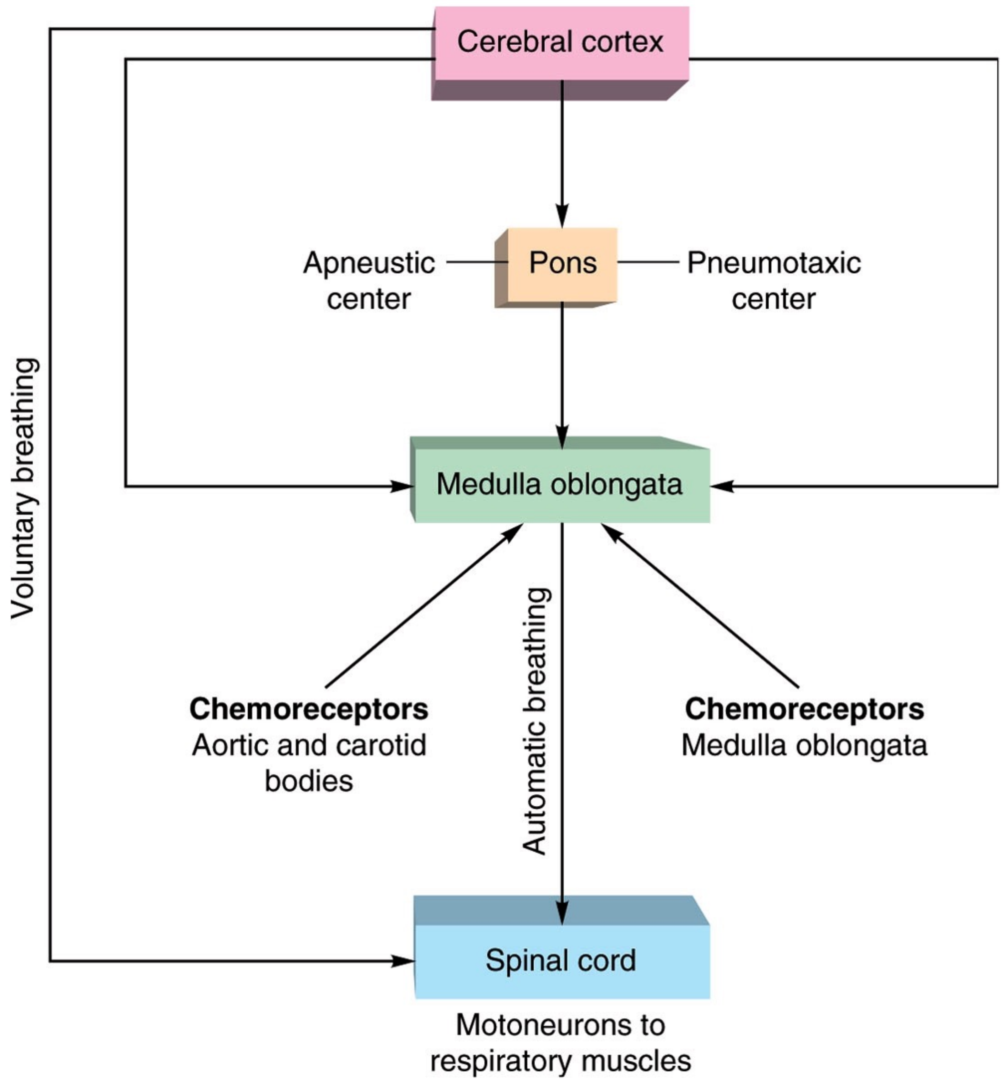
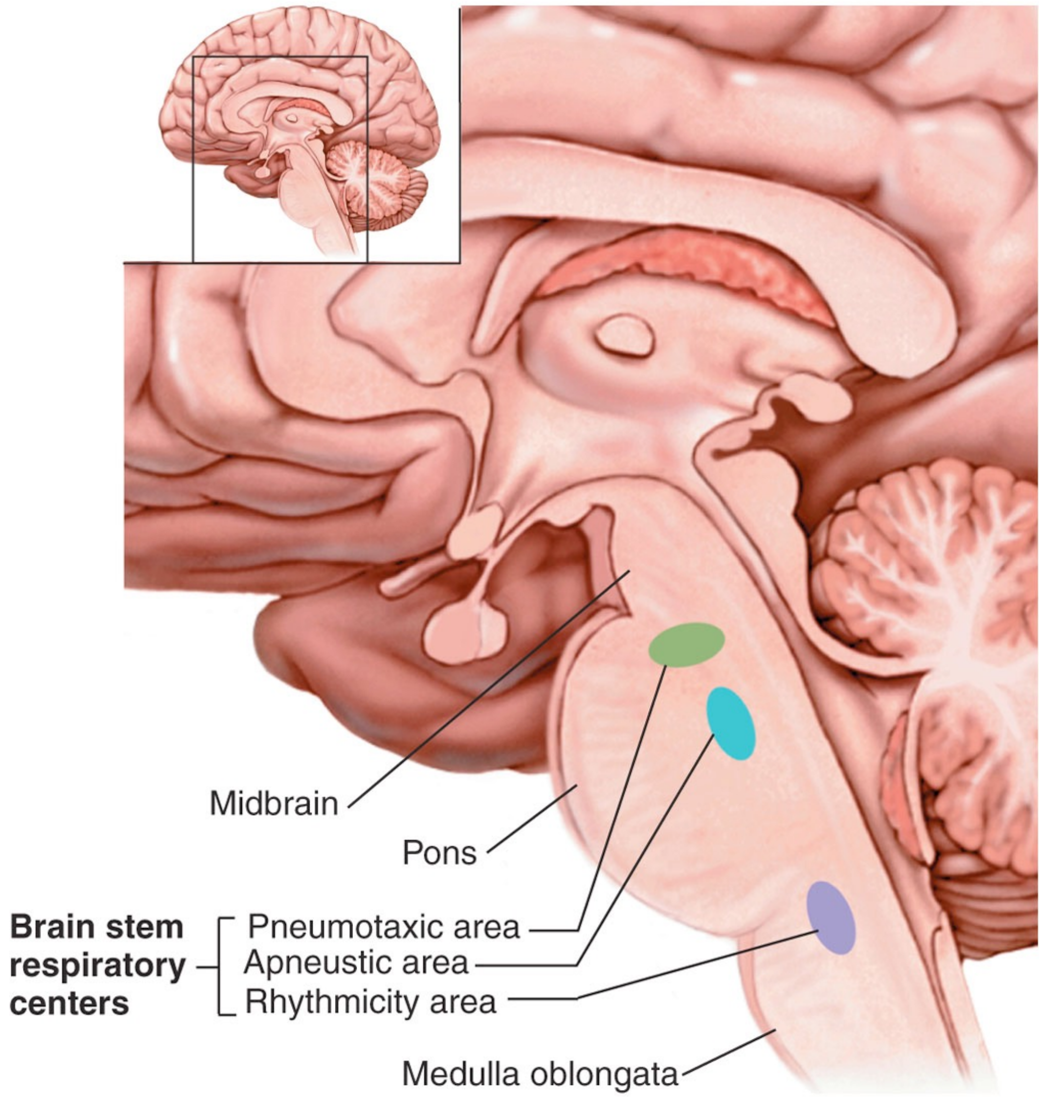

Cerebral cortex
Apneustic center
Pons
Pneumotaxic center
Medulla oblongata
Voluntary breathing
Automatic breathing
Chemoreceptors
Aortic and carotid bodies
Chemoreceptors
Medulla oblongata
Spinal cord
Motoneurons to respiratory muscles

Figure 16.26

Figure 16.24


Midbrain
Pons
Brain stem respiratory centers
Pneumotaxic area
Apneustic area
Rhythmicity area
Medulla oblongata

Breathing rate is regulated by blood pH and $C0_2$

breathing reduces plasma [CO_2]; plasma [CO_2] increases breathing.

- When $C0_2$ levels are high, breating rate increases to blow off $C0_2$
- In low $C0_2$ conditions, CO_2 is easily blown off, so breathing rate does not change (even if O_2 levels are dangerously low)

  Examples:

  1. breathing into a sealed container
  -> decreased O_2, increased CO_2 in the container
  -> faster, deeper breathing as body tries to blow off excess CO_2

  2. breathing into a sealed container **with CO_2 filter**
  -> decreased O_2, but no CO_2 in the container
  -> normal breathing, because brain does not detect elevated CO_2
  -> until body runs out of O_2

  3. pilots at high altitude: low O_2, low CO_2

Jonathan Miller, The Body in Question, part 4 (6/6)
https://youtu.be/yUBQjnQVJ4U?t=2589

Holding breath (hypoventilation) allows build up of CO_2.
Faster breathing (hyperventilation) blows off more CO_2 (lowers P_{CO2} in blood)

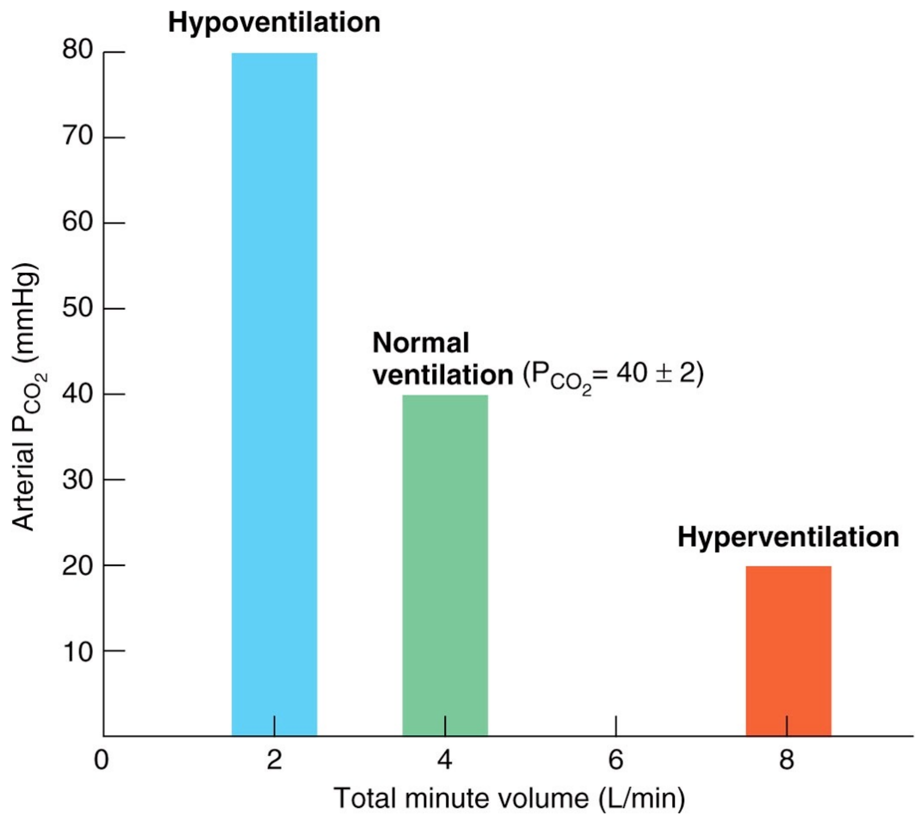


Figure 16.27

Chemoreceptors in:

Medulla

Carotid Bodies

Aortic Bodies

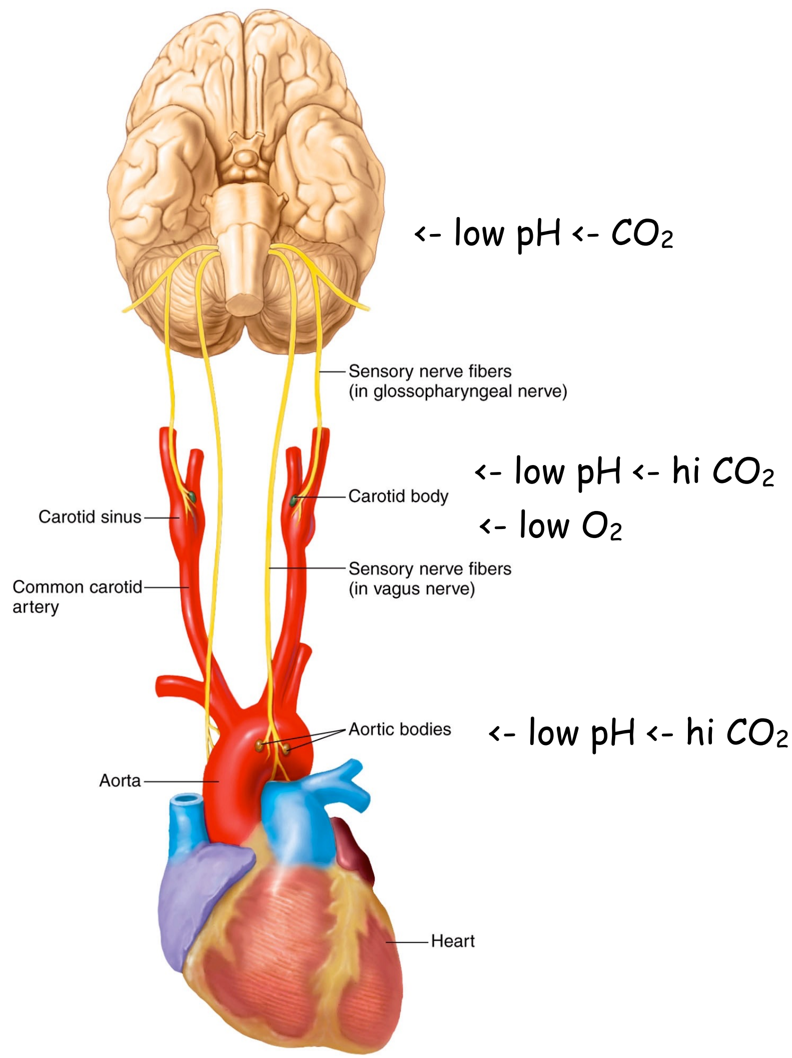
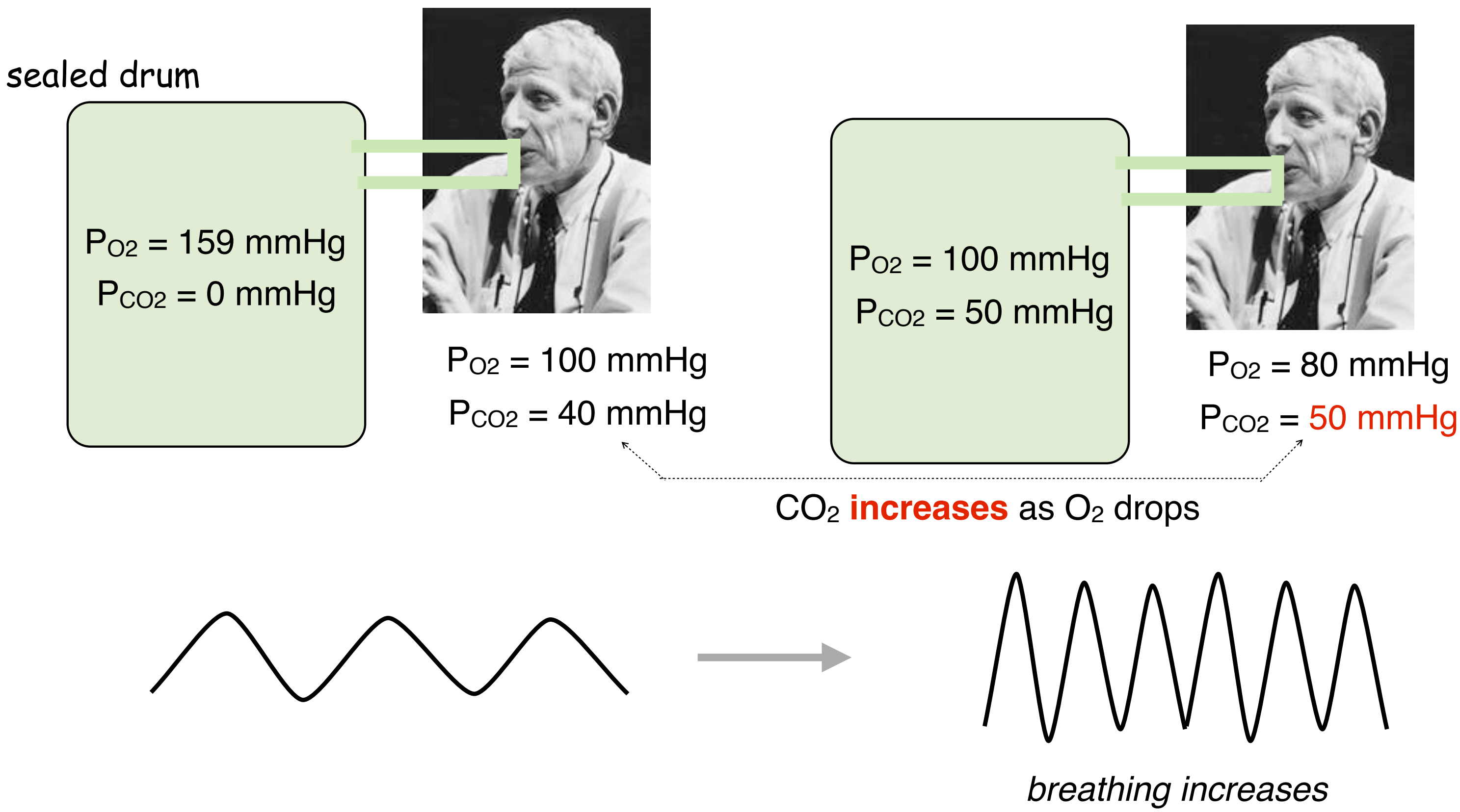


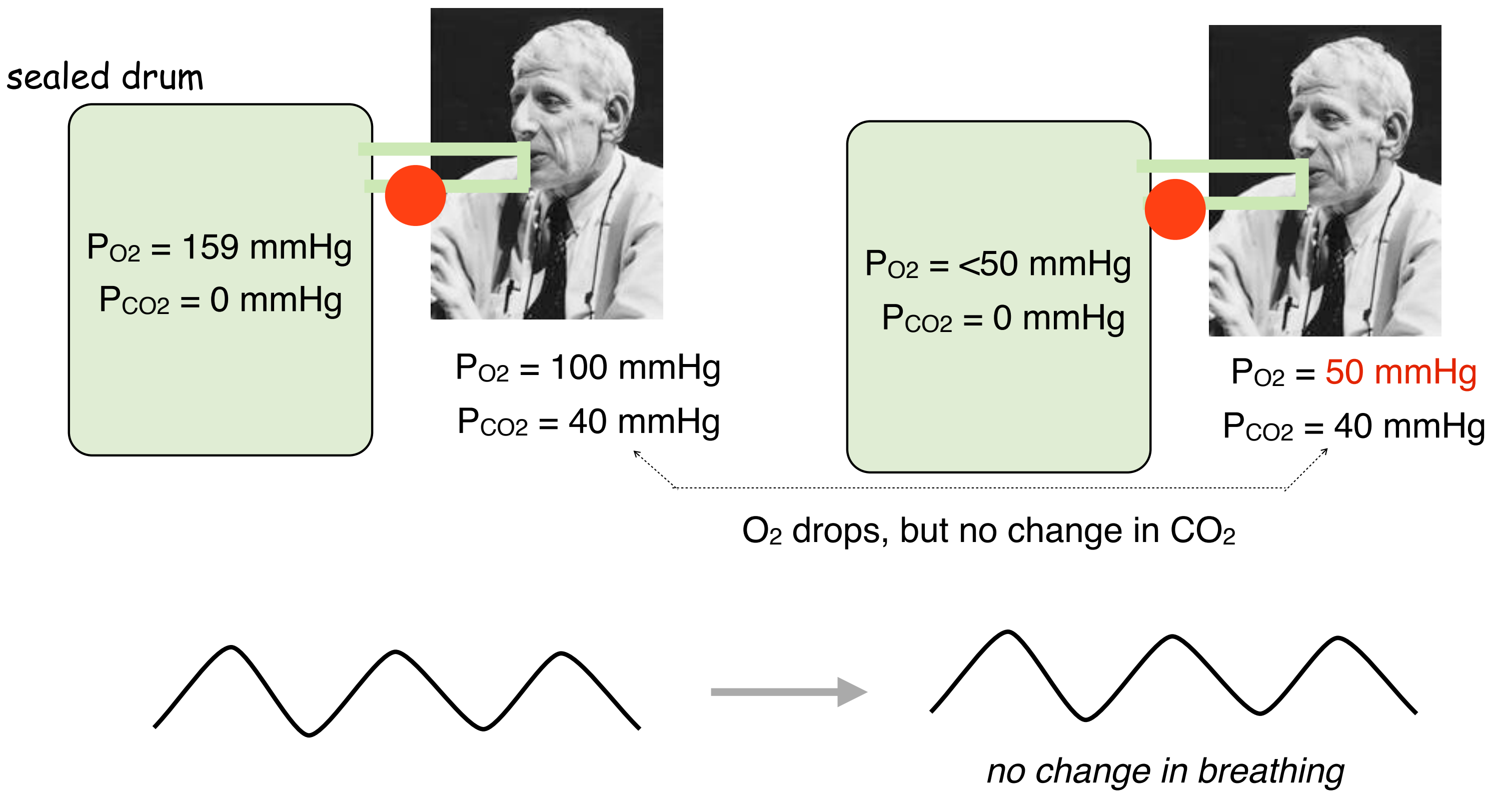
Figure 16.25

Jonathan Miller, The Body in Question, part 4

Rebreathing air: CO_2 **increases** as O_2 drops; breathing rate **increases**

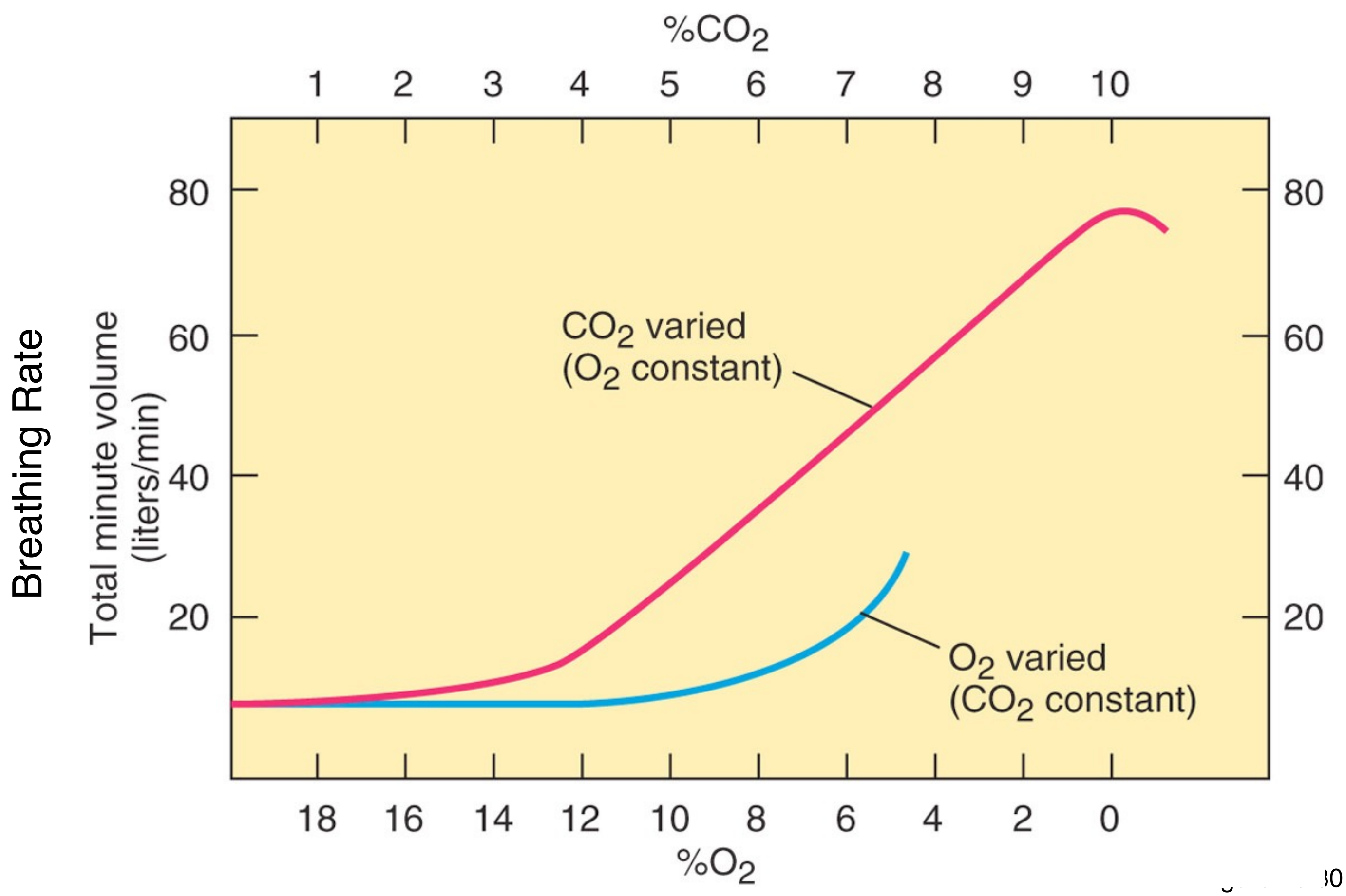


Rebreathing air with **CO_2 filter**: O_2 drops but CO_2 stays low.
Breathing rate does **not** increase, and brain runs out of oxygen



CO_2 levels control breathing:

Increasing CO_2 causes bigger change in breathing than lowering O_2



CO_2 and Bicarbonate act as a pH Buffer in the blood

Buffer - a chemical added to a solution to keep the pH constant by preventing rapid changes in [H^+]

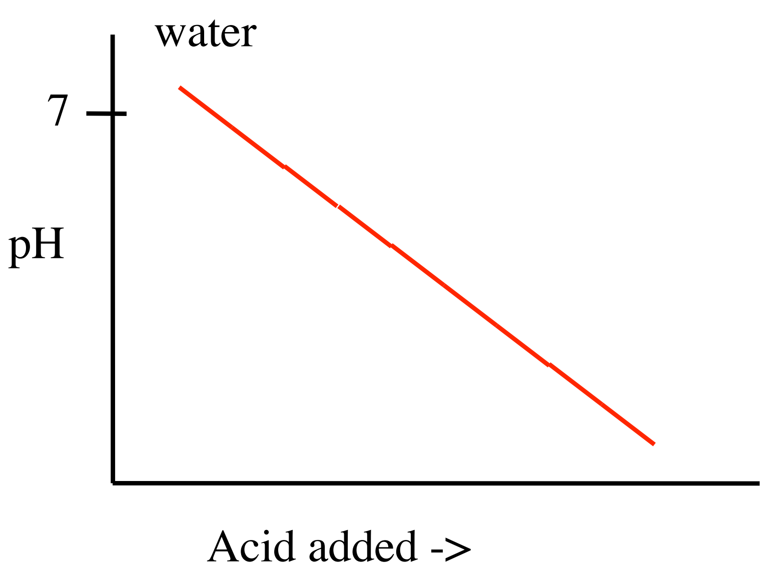
As acid is added to a buffer, it absorbs the new [H^+].
---> so little or no change in pH

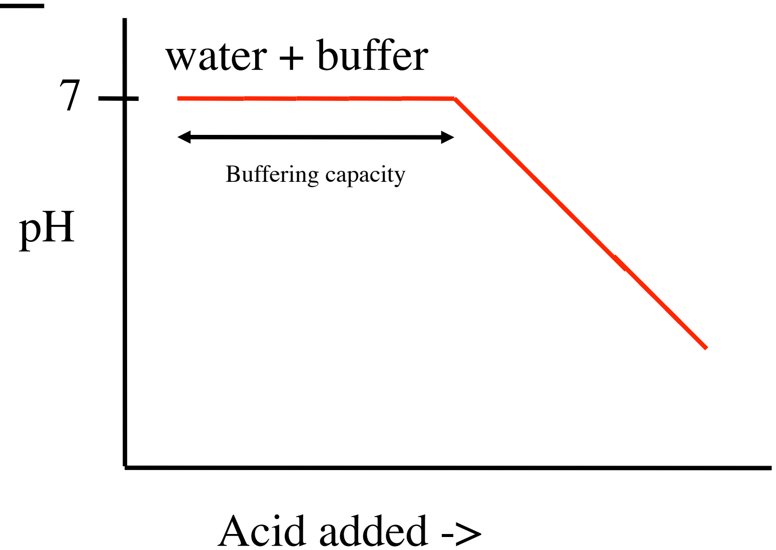
As base is added to a buffer, it gives up [H^+] to replace the ones sucked up by the base.
---> so little or no change in pH

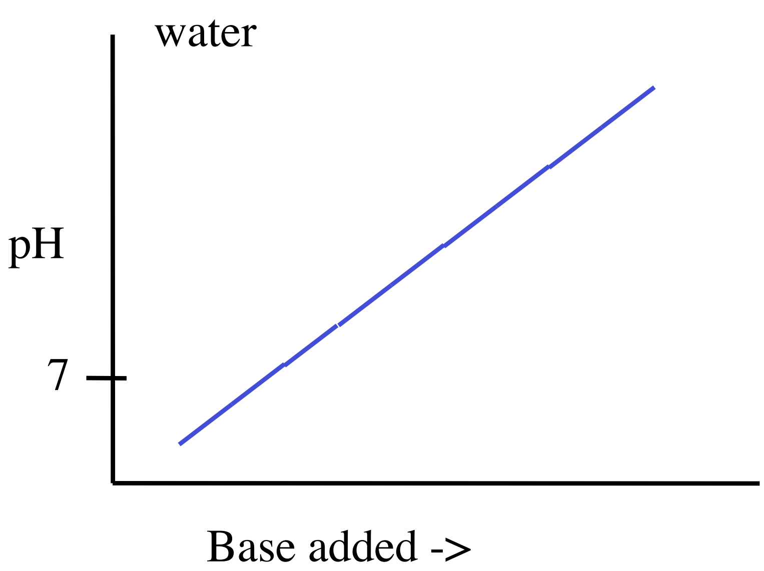
Carbonic acid *Bicarbonate*

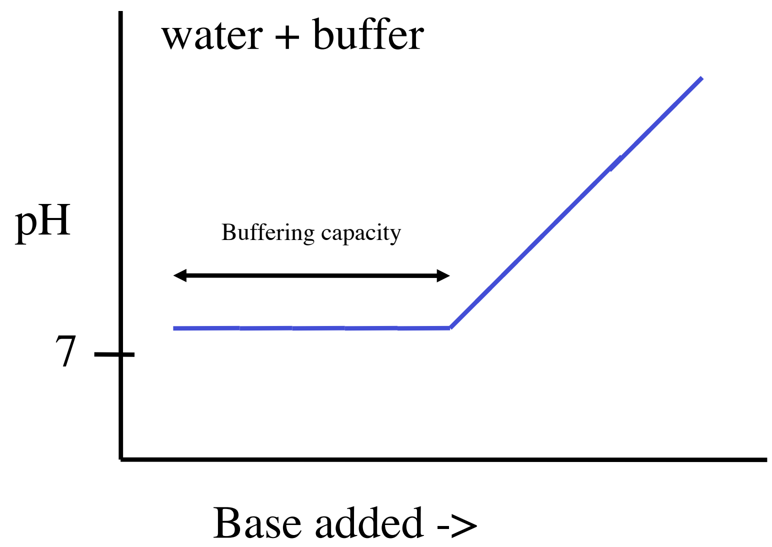
$$CO_2 + H_20 \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

If blood pH is too high (basic) breathe less to retain CO_2:
more CO_2 -> more bicarbonate + more H^+ -> more acid
If blood pH is too low (acidic) breathe more to blow off CO_2:
less CO_2 -> less bicarbonate + less H^+ -> less acid


water
7
pH
Acid added ->


water + buffer
7
Buffering capacity
pH
Acid added ->


water
pH
7
Base added ->


water + buffer
pH
Buffering capacity
7
Base added ->

Bicarbonate: The natural buffer in the blood

Carbonic acid (H_2CO_3) — *Bicarbonate* (HCO_3^-)

$$CO_2 + H_20 \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

if pH ↑ , then need more H^+

$$CO_2 + H_20 \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

$$CO_2 + H_20 \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

$$CO_2 + H_20 \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

So if blood pH rises (more basic), keep CO_2 -- *don't* breathe out *if pH* ↓ *, then need to absorb more H^+*

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

↓

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

↓

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

So if blood pH drops (more acidic), breath **off** CO_2

pH and Breathing

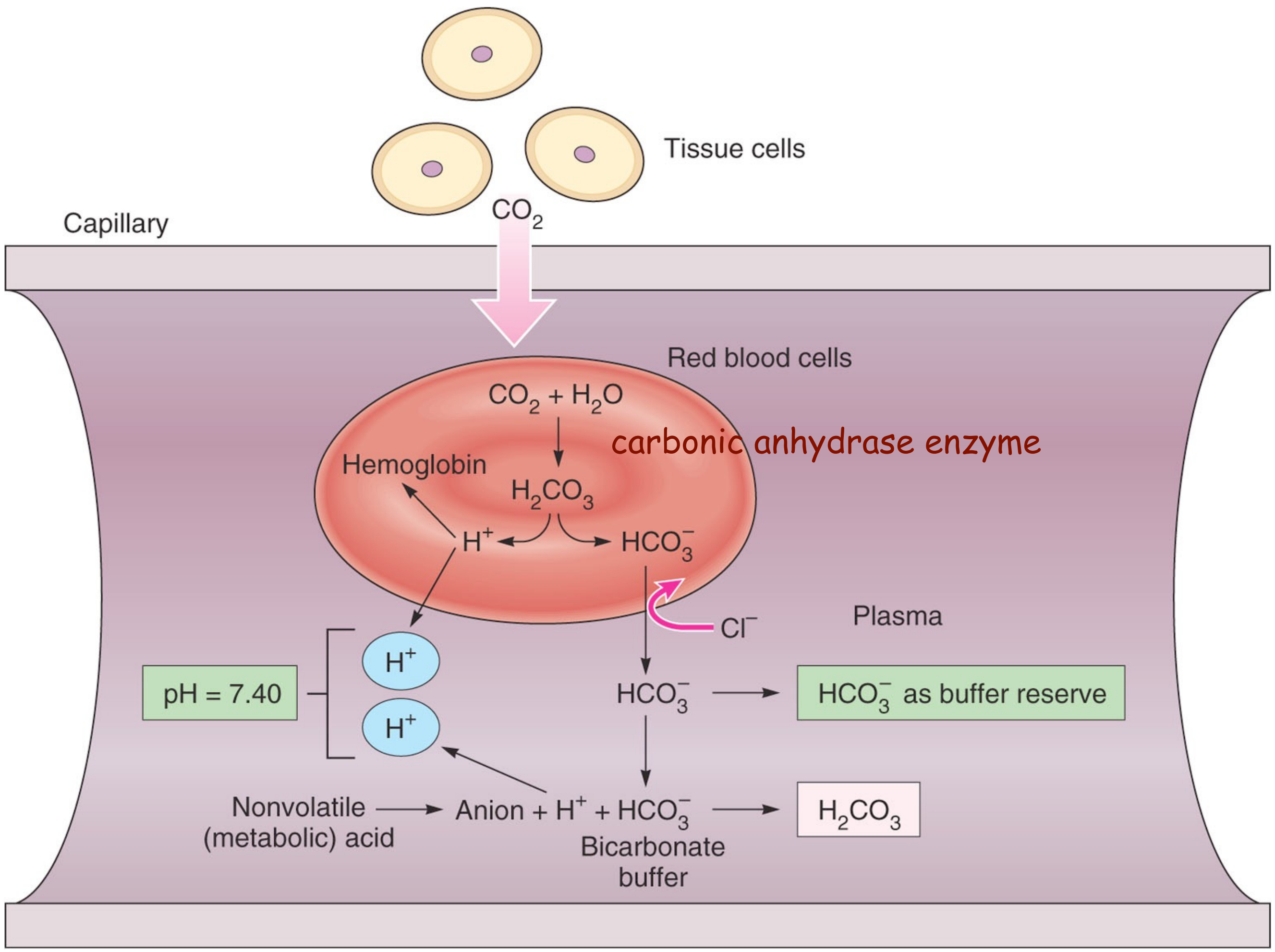
If blood pH is too high (basic) breathe less to retain CO_2:

more CO_2 -> more bicarbonate + more H^+ -> more acidic

If blood pH is too low (acidic) breathe more to blow off CO_2:

less CO_2 -> less bicarbonate + less H^+ -> less acidic

Figure 16.40



Tissue cells
CO_2
Capillary
Red blood cells
$CO_2 + H_2O$
carbonic anhydrase enzyme
Hemoglobin
H_2CO_3
H^+
HCO_3^-
Cl^-
Plasma
H^+
H^+
pH = 7.40
HCO_3^-
HCO_3^- as buffer reserve
Nonvolatile (metabolic) acid
Anion + H^+ + HCO_3^-
Bicarbonate buffer
H_2CO_3

Chemoreceptors in:

Medulla

Carotid Bodies

Aortic Bodies

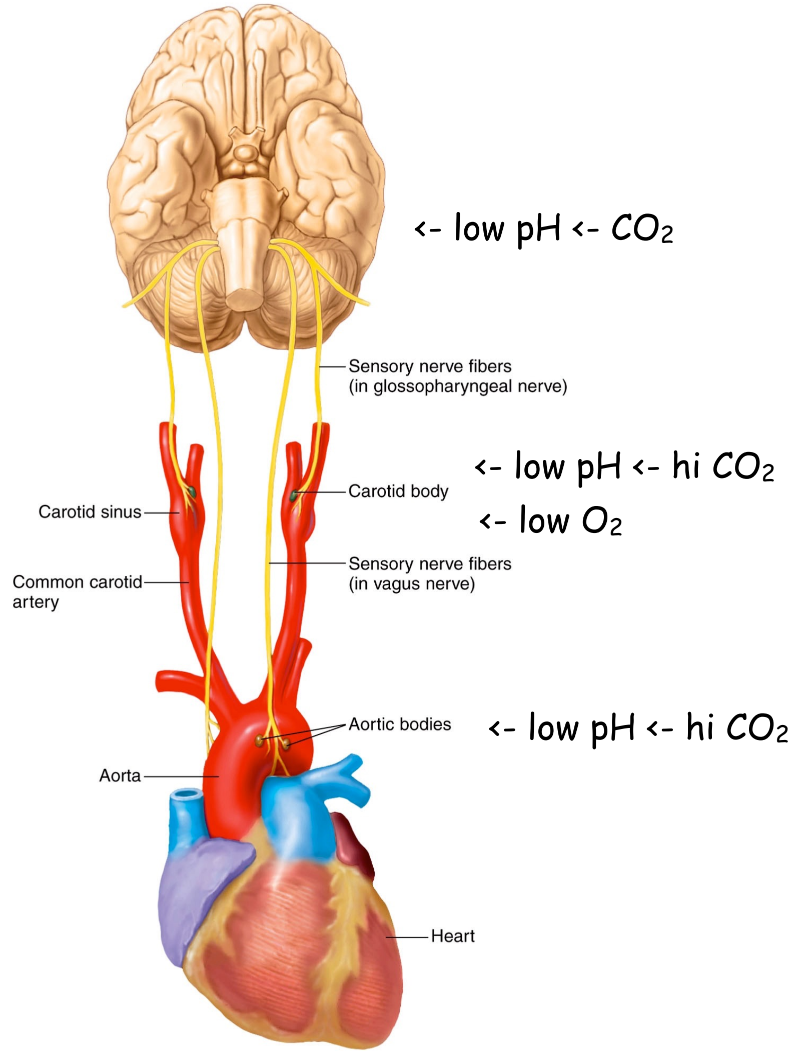
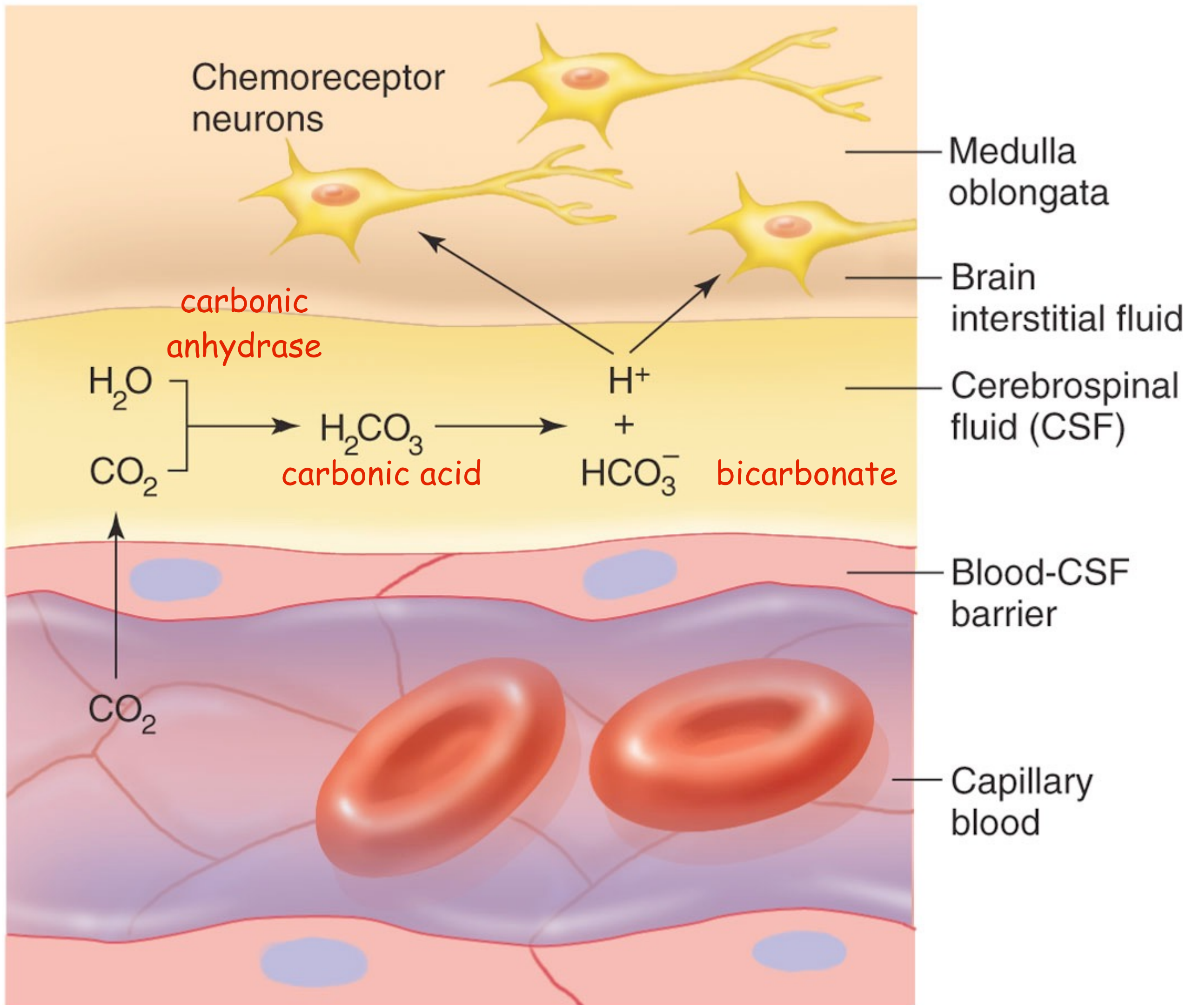
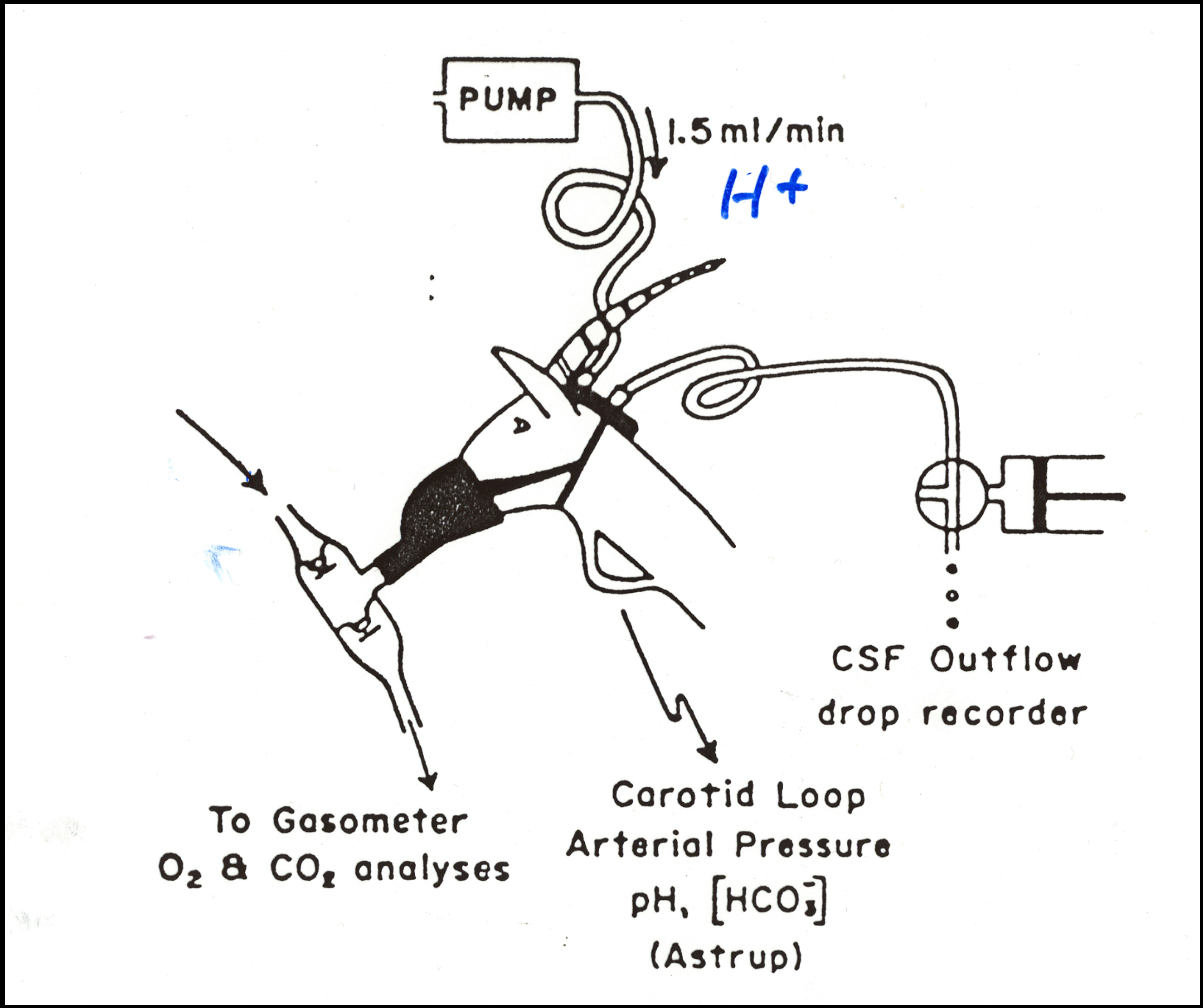


Figure 16.25

Figure 16.29


Chemoreceptor neurons
Medulla oblongata
Brain interstitial fluid
carbonic anhydrase
H_2O
CO_2
H_2CO_3
carbonic acid
H^+
+
HCO_3^-
bicarbonate
Cerebrospinal fluid (CSF)
Blood-CSF barrier
CO_2
Capillary blood


PUMP
1.5 ml/min
H+
CSF Outflow
drop recorder
To Gasometer
O_2 & CO_2 analyses
Carotid Loop
Arterial Pressure
pH, $[HCO_3^-]$
(Astrup)

Brain pH → BREATHING RATE IN GOATS

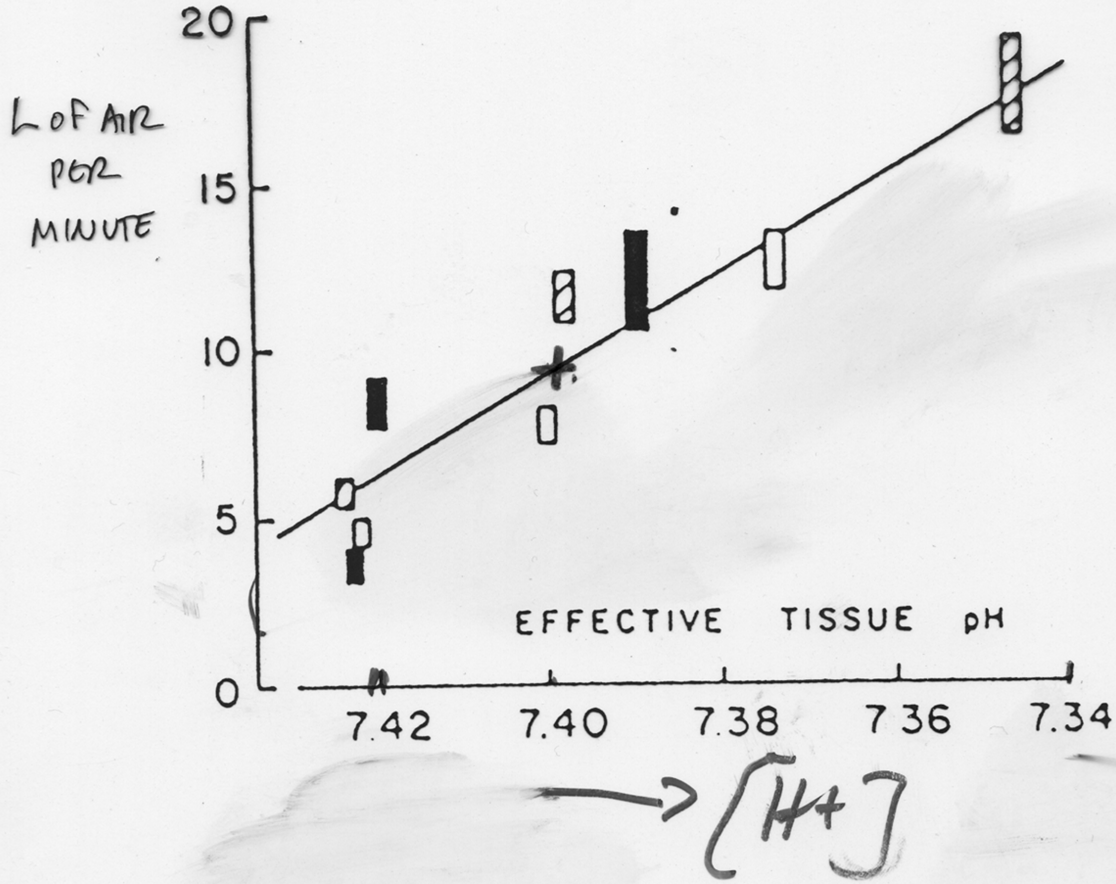
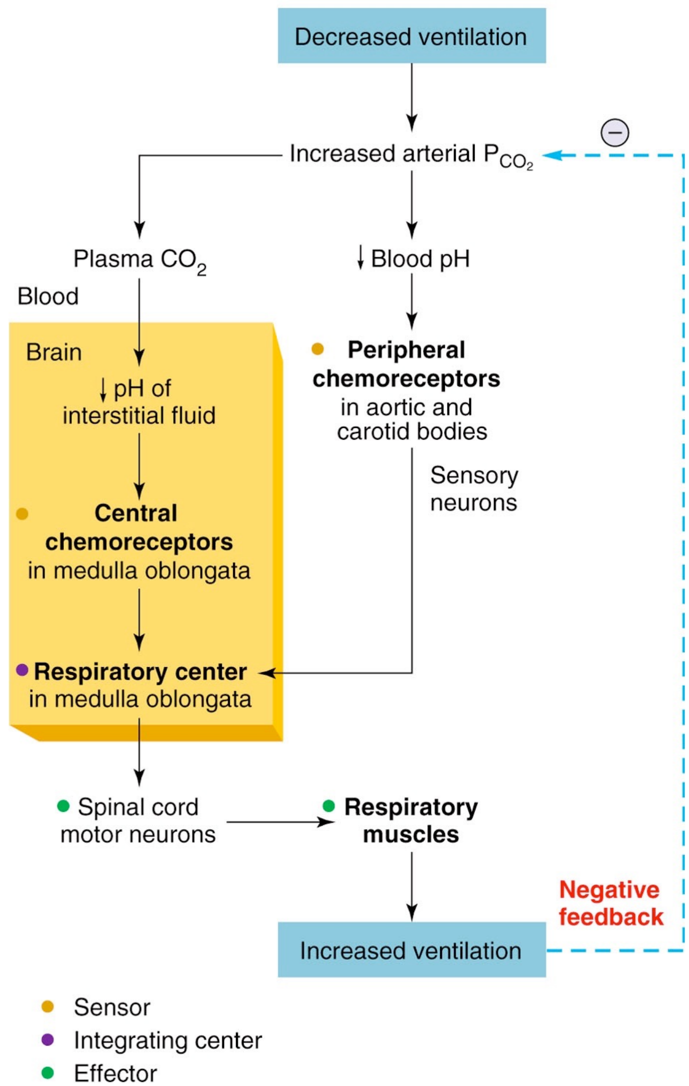


Table 16.6

Table 16.6 | Sensitivity of Chemoreceptors to Changes in Blood Gases and pH

Stimulus	Chemoreceptor	Comments
↑P_{CO_2}	Medullary chemoreceptors; aortic and carotid bodies	Medullary chemoreceptors are sensitive to the pH of cerebrospinal fluid (CSF). Diffusion of CO_2 from the blood into the CSF lowers the pH of CSF by forming carbonic acid. Similarly, the aortic and carotid bodies are stimulated by a fall in blood pH induced by increases in blood CO_2.
↓pH	Aortic and carotid bodies	Peripheral chemoreceptors are stimulated by decreased blood pH independent of the effect of blood CO_2. Chemoreceptors in the medulla are not affected by changes in blood pH because H^+ cannot cross the blood-brain barrier.
↓P_{O_2}	Carotid bodies	Low blood P_{O_2} (hypoxemia) augments the chemoreceptor response to increases in blood P_{CO_2} and can stimulate ventilation directly when the P_{O_2} falls below 50 mmHg.



Decreased ventilation
Increased arterial P_{CO_2}
Plasma CO_2
↓ Blood pH
Blood
Brain
↓ pH of interstitial fluid
Peripheral chemoreceptors
in aortic and carotid bodies
Sensory neurons
Central chemoreceptors
in medulla oblongata
Respiratory center
in medulla oblongata
Spinal cord motor neurons
Respiratory muscles
Negative feedback
Increased ventilation
Sensor
Integrating center
Effector

Figure 16.28