BREATHING QUANTIFIED

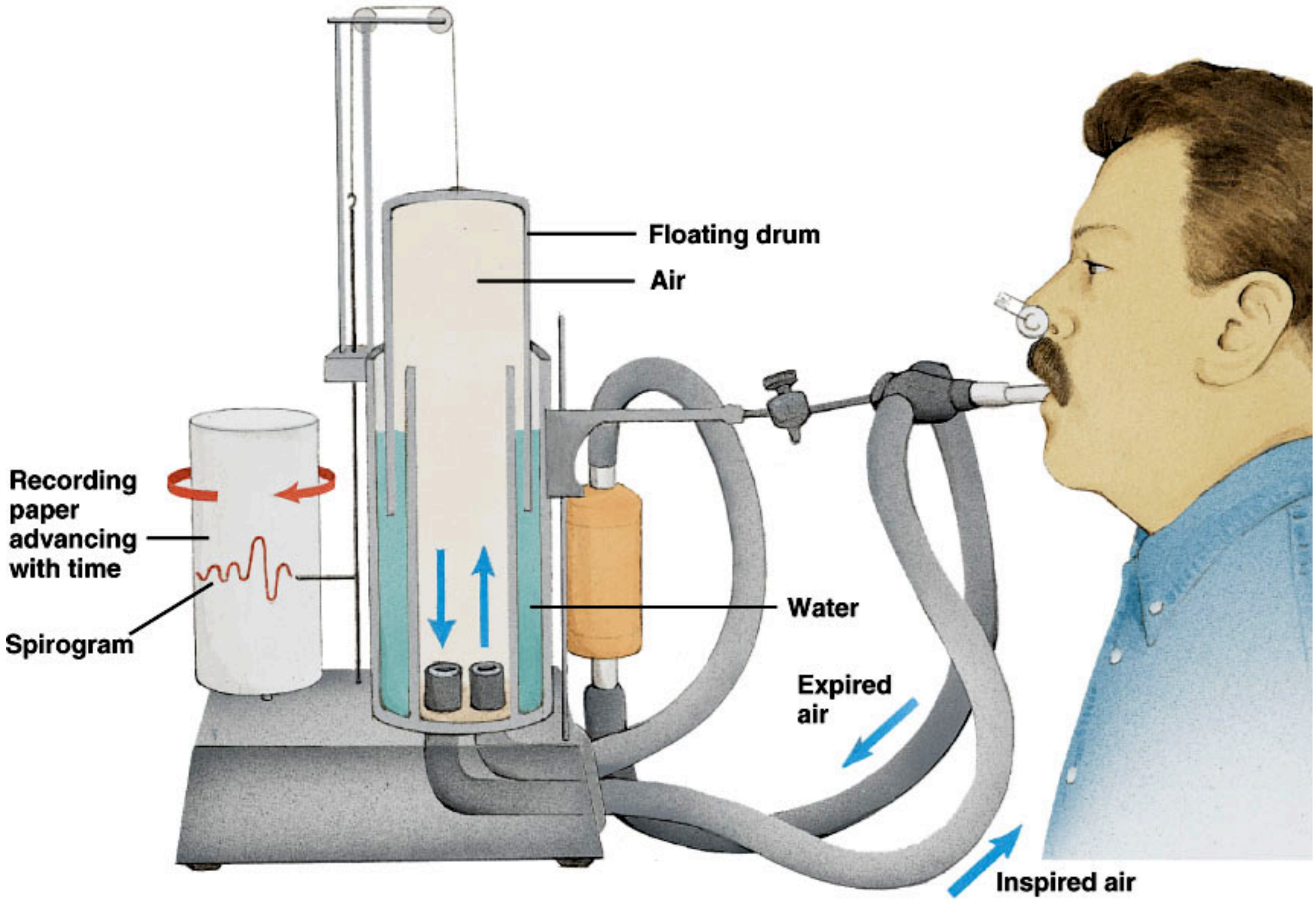
STATICS & DYNAMICS

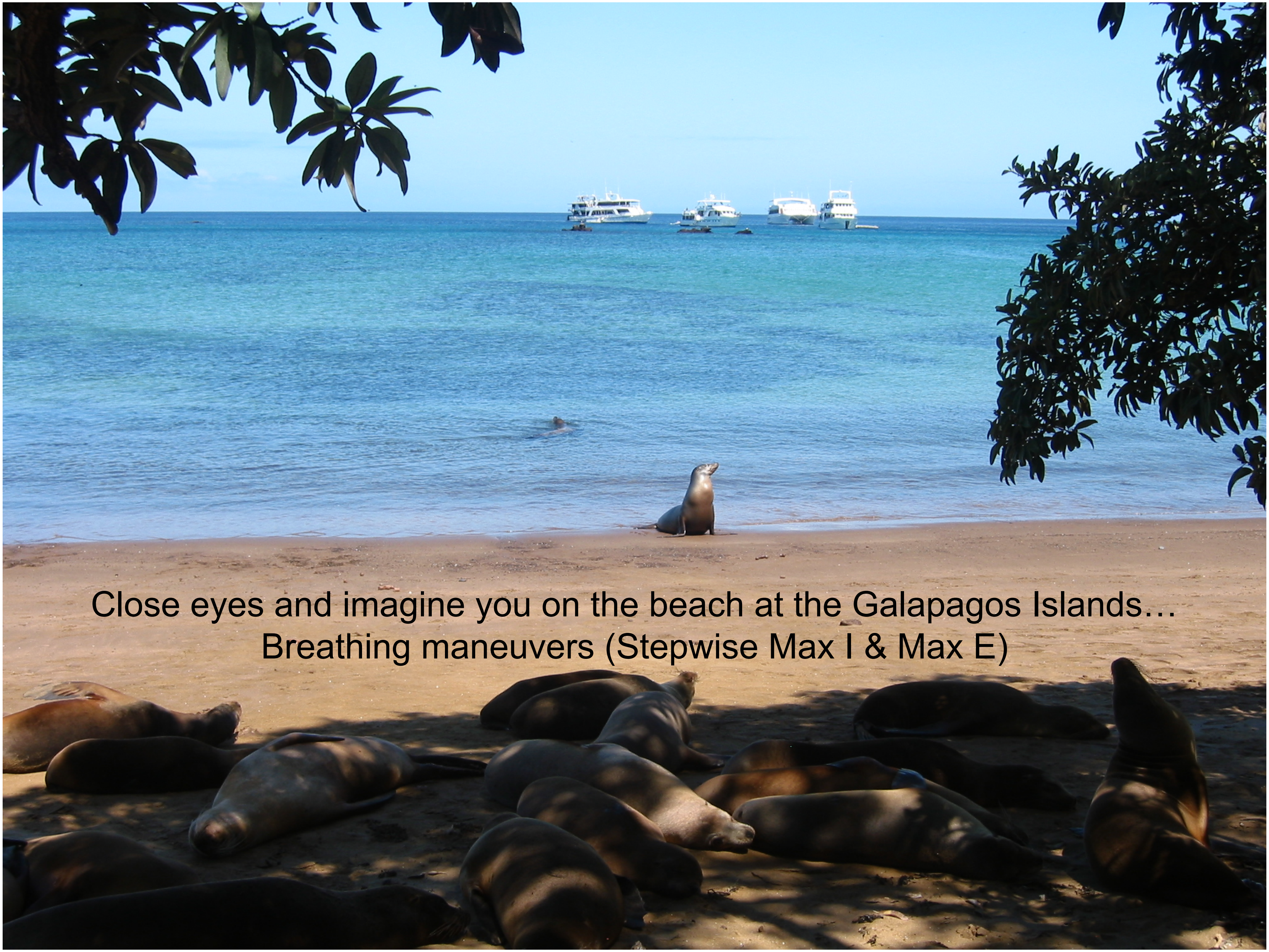
Objectives

1. Identify static lung volume and capacities on a spirogram. Given lung volumes be able to calculate lung capacities.
2. Describe three factors that affect lung volumes in a healthy person.
3. Compare the effect of obstructive and restrictive respiratory diseases on static lung volumes and the ratio of forced expired volume in one second to forced vital capacity.
4. Distinguish between minute, alveolar and dead space ventilation and be able to calculate them given tidal volume, breathing frequency and the anatomic dead space. Identify how pattern of breathing affects alveolar ventilation.
5. Compare the partial pressure of gases in an ideal alveolus to those in extremes of ventilation/perfusion mismatch. Describe the homeostatic mechanisms in place for matching of ventilation and perfusion at the alveolar level.

SPIROMETRY

WATER-SEALED SPIROMETER USED IN CAPS 303 LAB




Close eyes and imagine you on the beach at the Galapagos Islands…
Breathing maneuvers (Stepwise Max I & Max E)

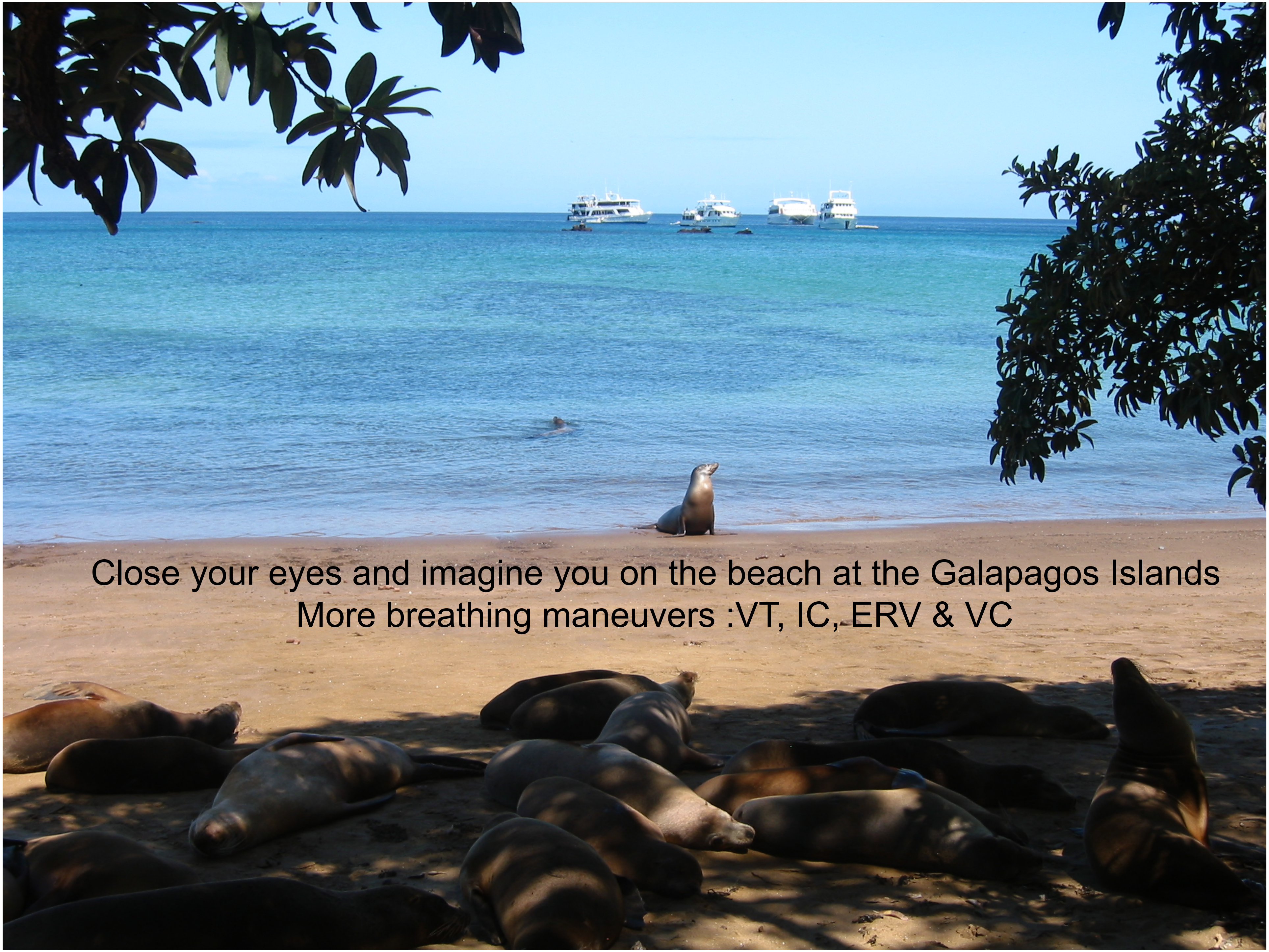
A SPIROMETER CAN NOT MEASURE ALL STATIC LUNG VOLUMES

Maximum Inspiration

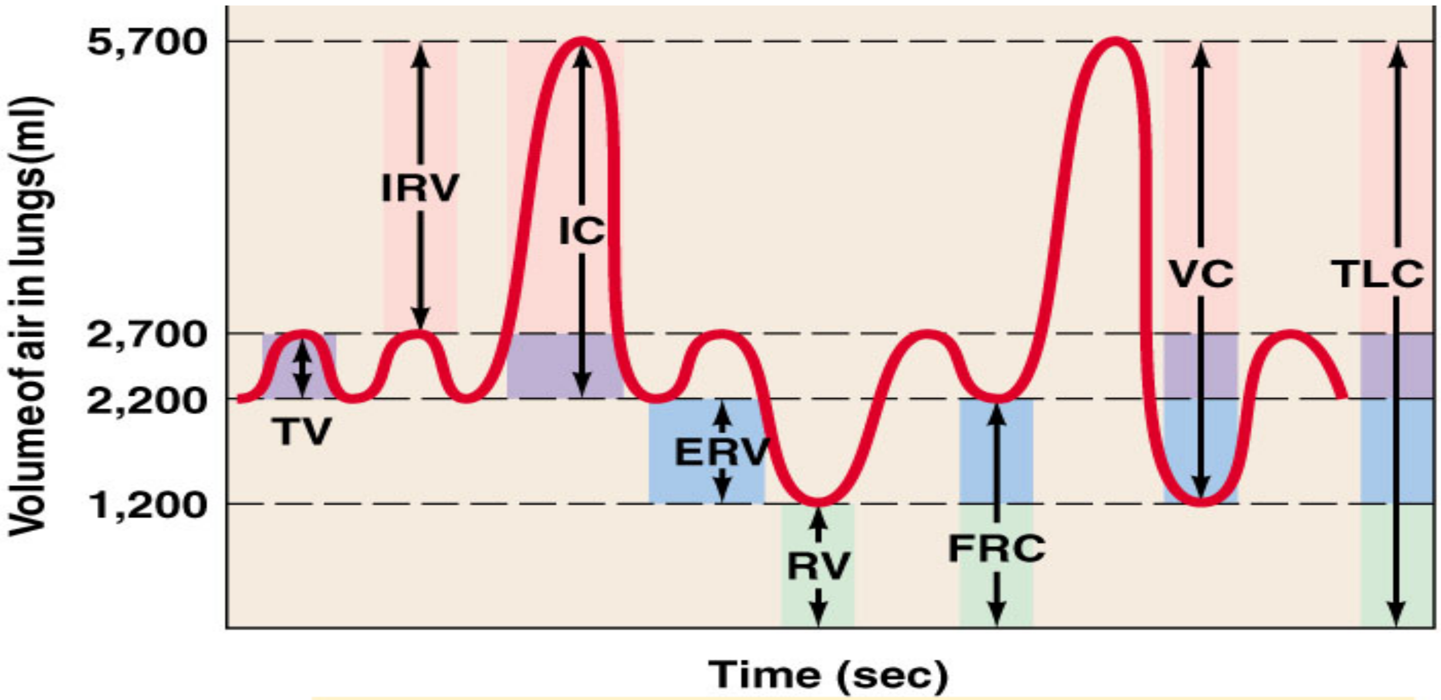
Total Lung Capacity (TLC)

Maximum Expiration

Residual Volume (RV)


Close your eyes and imagine you on the beach at the Galapagos Islands
More breathing maneuvers :VT, IC, ERV & VC

STATIC LUNG VOLUMES



- **TV = Tidal volume (500 ml)**
- **IRV = Inspiratory reserve volume (3,000 ml)**
- **IC = Inspiratory capacity (3,500 ml)**
- **ERV = Expiratory reserve volume (1,000 ml)**
- **RV = Residual volume (1,200 ml)**
- **FRC = Functional residual capacity (2,200 ml)**
- **VC = Vital capacity (4,500 ml)**
- **TLC = Total lung capacity (5,700 ml)**

FACTORS DETERMINING STATIC LUNG VOLUMES

HEIGHT taller individuals have larger lung volumes

GENDER males have larger lung volumes than females

AGE in children lung volumes increase with development until the ages 20-25 when lung volumes remains constant. In old age, RV & FRC increase, VC decreases resulting in gas trapping These changes are considered to be due to stiffening of the chest wall.

ETHNICITY differences are considered to be in part due to body build (e.g. relative length or width of chest wall) consider Asian, Black & Inuit populations.

PERFORMING THE FORCED VITAL CAPACITY MANEUVER
FIRST STEP IN DIAGNOSING RESPIRATORY DISEASES

Two main categories of ventilatory defects—

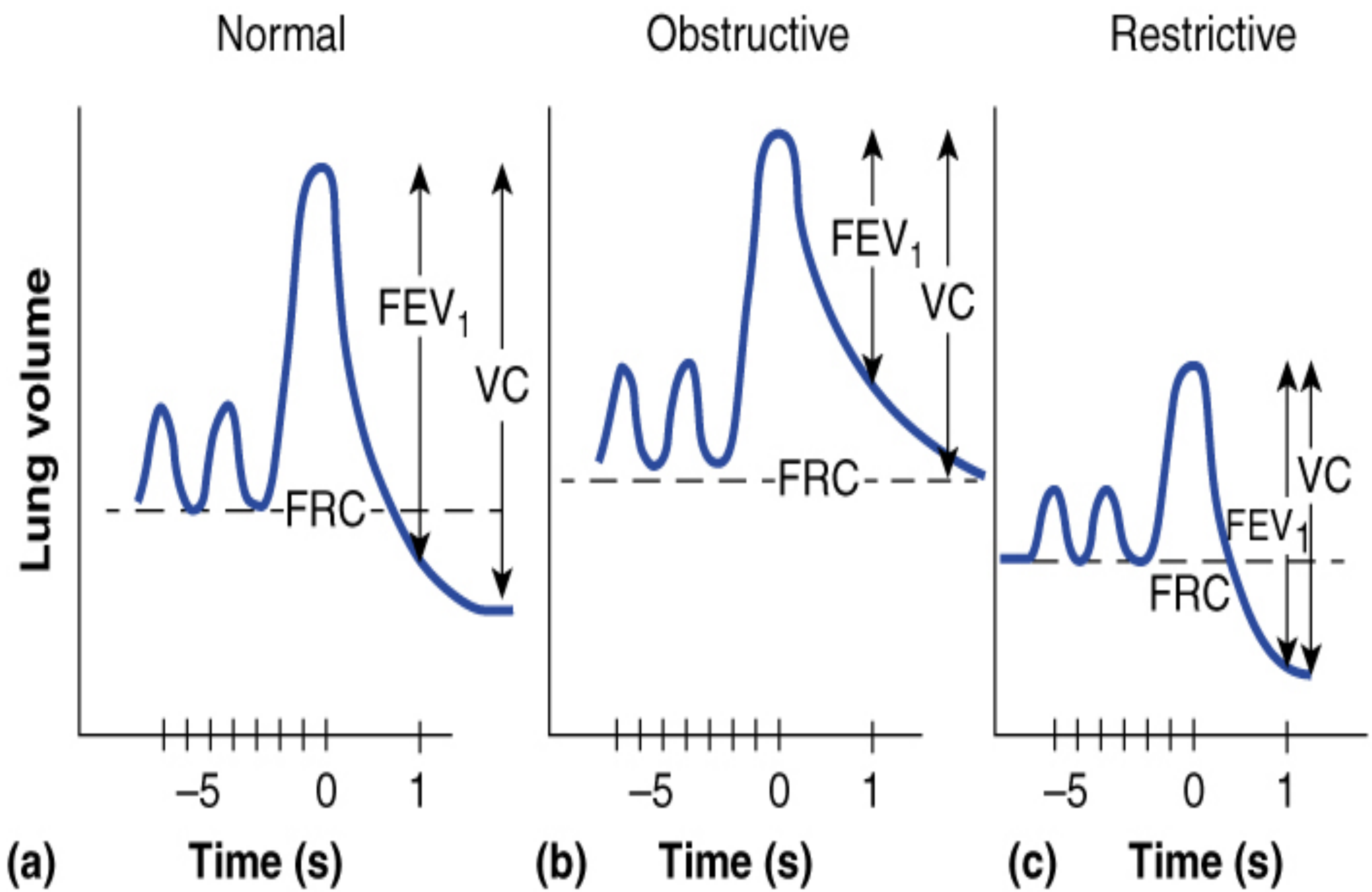
1. Restrictive diseases make it more difficult to get air into the lungs they "restrict" inspiration.

- Pulmonary Fibrosis-140 known causes- the most common form of lung fibrosis is of unknown etiology [idiopathic], others include occupational diseases: asbestosis, silicosis, farmer's lung (moldy hay)- bird breeder's lung (excreta)

- Stiff Chest Wall   e.g. ankylosing spondylitis, kyphoscoliosis

2. Obstructive diseases make it more difficult to get air out of the lungs they obstruct and "limit air flow" during expiration.

- Chronic Obstructive Pulmonary disease (COPD)
- Asthma
- Chronic Bronchitis
- Emphysema



Normal
Obstructive
Restrictive
Lung volume
FEV1
VC
FRC
FEV1
VC
FRC
VC
FEV1
FRC
–5
0
1
–5
0
1
–5
0
1
(a)
Time (s)
(b)
Time (s)
(c)
Time (s)

FIGURE 11-20

In disease, airway radius ↓s due to several mechanisms leading to EXPIRATORY AIRFLOW LIMITATION

Bronchoconstriction asthma/COPD

Inflammation asthma/chronic bronchitis/COPD/bronchiolitis

Excess Mucus Production asthma/chronic bronchitis/cystic fibrosis

Reduced Alveolar Elastic Recoil emphysema

reduced recoil means less tethering on neighboring airway which in turn will be reduced in airway size-see next slide]

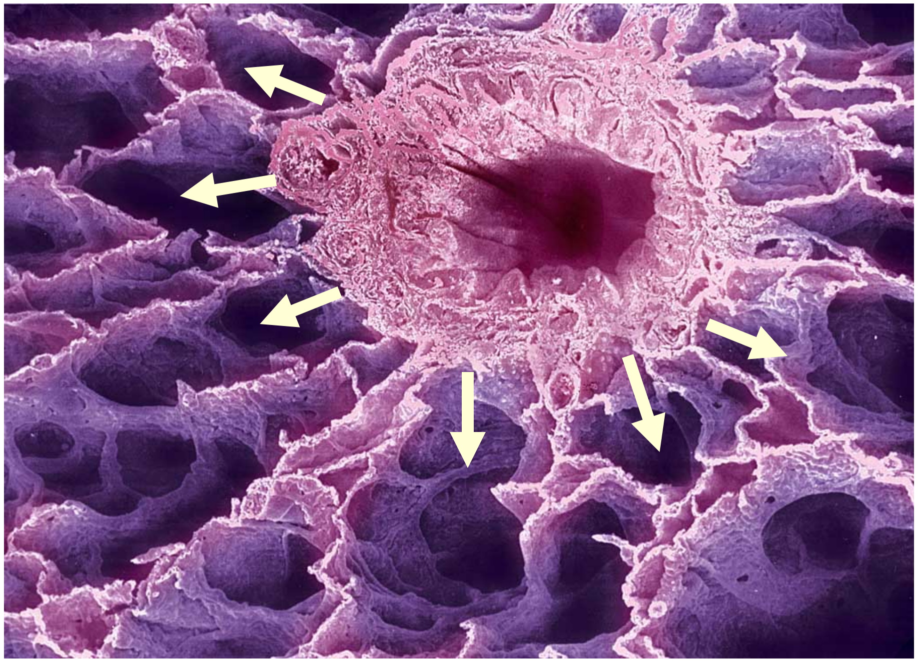
In all cases, airway resistance ↑ & maximal expiratory flow ↓. These individuals have a hard time breathing out.

How come breathing in is not affected during inspiration?

The inward elastic recoil of airways contributes to:

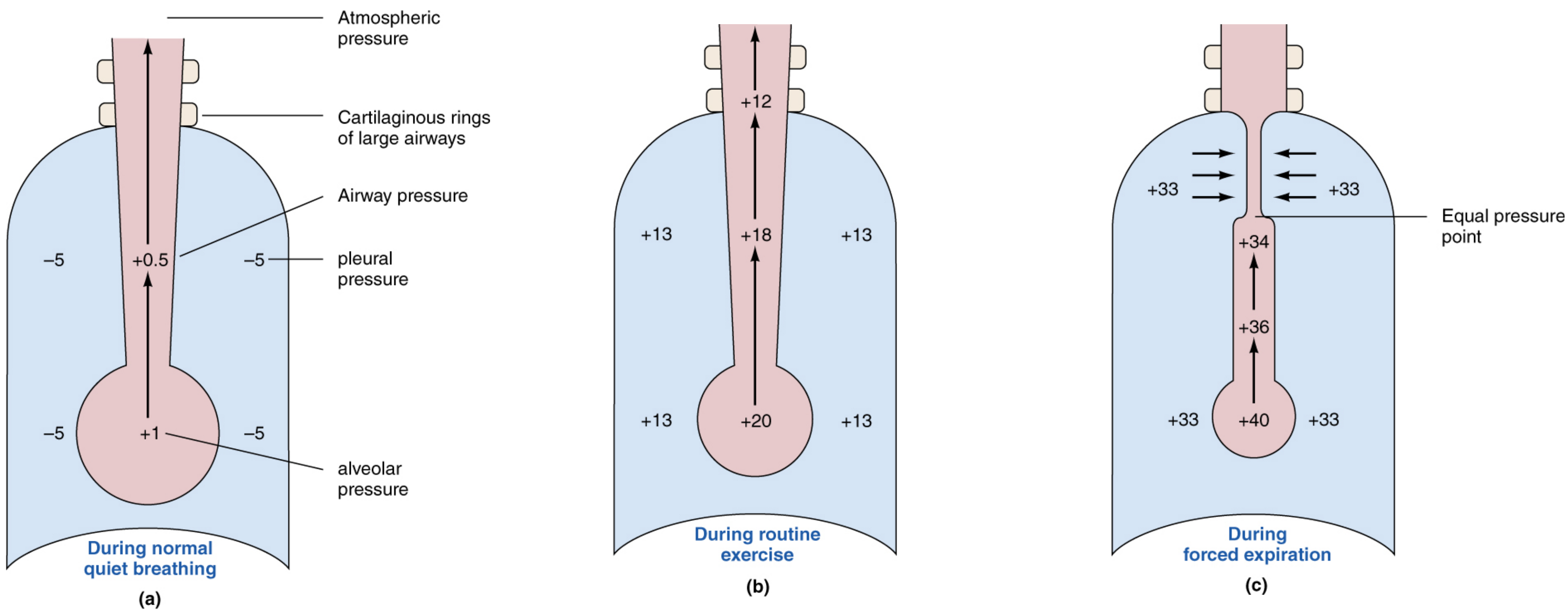
1) Driving pressure for expiration
2) Patency of neighboring airways

Alveolar inward recoil creating radial traction on neighboring airways--keeping them open



EXPIRATORY AIRFLOW LIMITATION

NON-CARTILAGINOUS AIRWAY PATENCY DEPENDS ON AIRWAYS TRANSMURAL PRESSURE

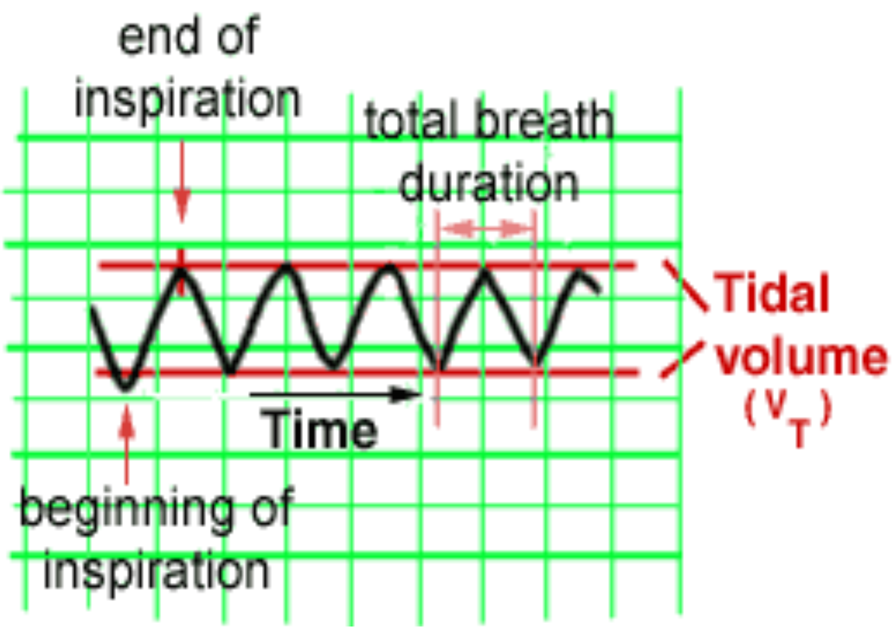


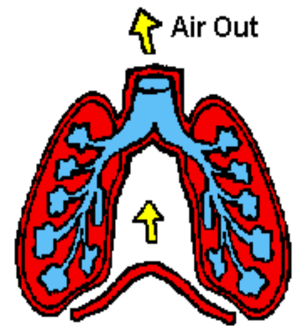
BREATHING QUANTIFIED

DYNAMIC VOLUMES

MINUTE VENTILATION ($\dot{V}_E$)

$\dot{V}_E$ = VT X breathing frequency





= 500 ml X12 b/min
= 6000 ml/min
= 6 L/min

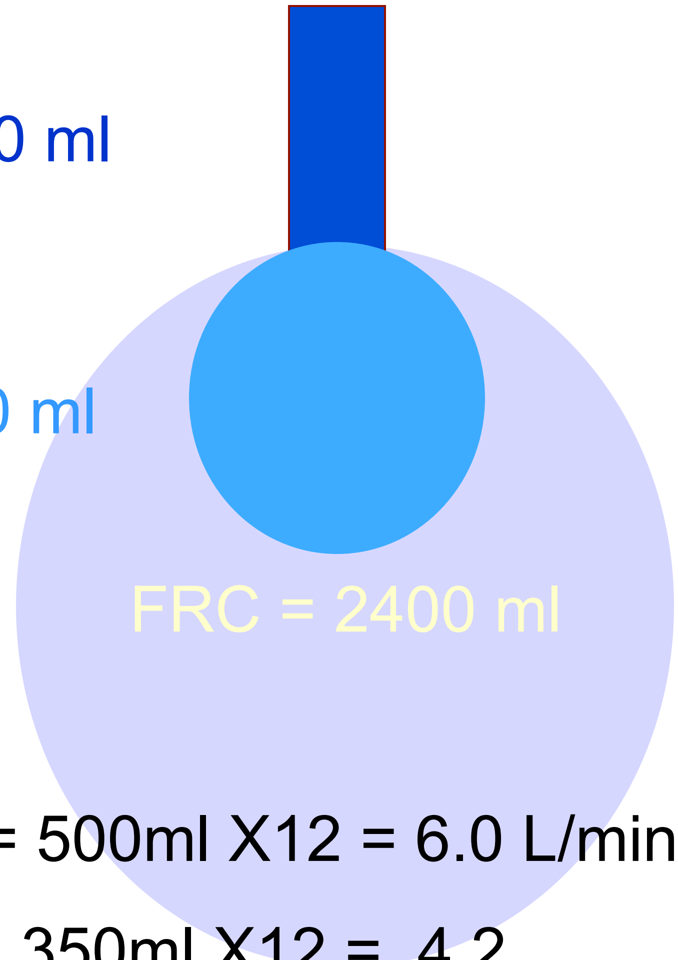
VENTILATION

MINUTE, ALVEOLAR & DEAD SPACE

$\dot{V}E$
500 ml

V_{DS}=150 ml

V_A=350 ml



$\dot{V}E$ = V_T X breathing frequency = 500ml X12 = 6.0 L/min

$\dot{V}A$ = V_A X breathing frequency = 350ml X12 = 4.2

$\dot{V}DS$ = V_{DS} X breathing frequency=150mlX12 = 1.8

PATTERN OF BREATHING AFFECTS

ALVEOLAR VENTILATION & GAS EXCHANGE

Breathing Pattern	Tidal Volume (ml)	Breathing Frequency (breaths/min)	Minute Ventilation (ml/min)	Dead Space Ventilation (ml/min)	Alveolar Ventilation (ml/min)
normal quiet breathing	500	12	6000	150X12=1800	4200
shallow & fast	150	40	6000	150X40=6000	0
deep & slow	1000	6	6000	150X6=900	5100

Is panting effective in improving gas exchange?
What breathing pattern is generated when you exercise?

MECHANICAL VENTILATION

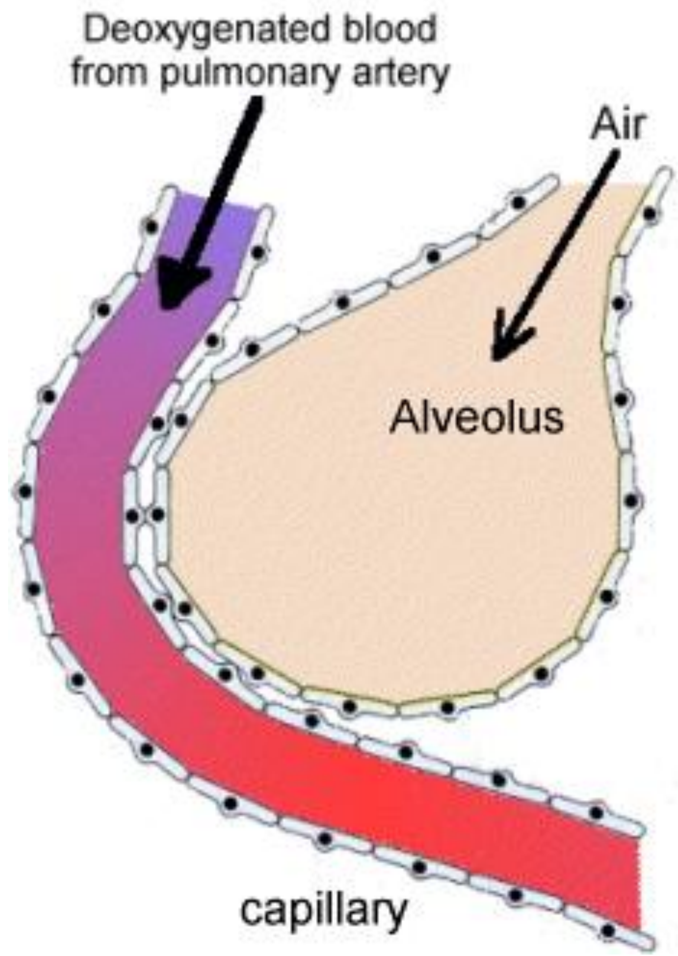
In the figures below, a mechanical ventilator is assisting the patient to breathe. What is the dead space volume?

ALVEOLAR VENTILATION

A KEY PLAYER IN GAS EXCHANGE

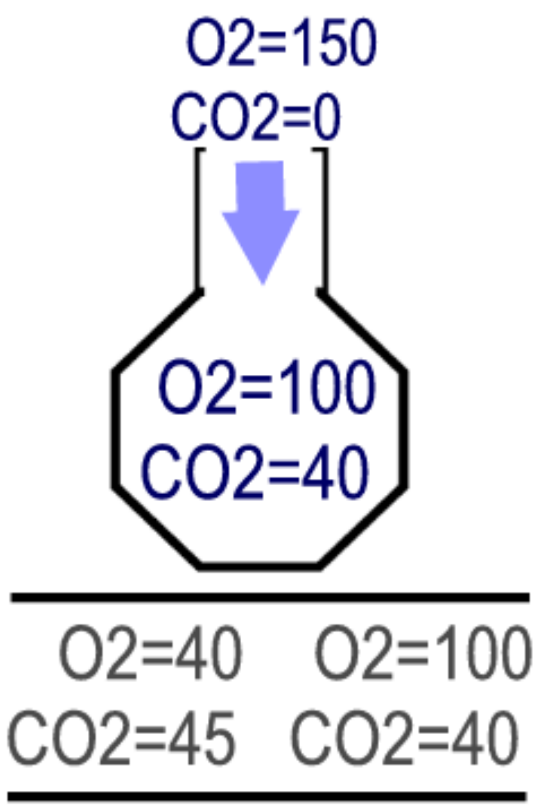
Alveolar ventilation is the portion of breathing that reaches the alveoli & participates in gas exchange

Two key players in gas exchange are alveolar ventilation & alveolar perfusion



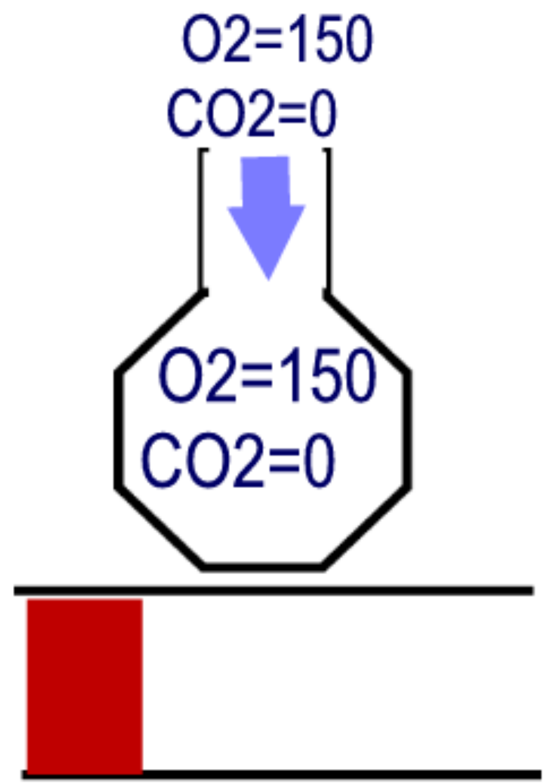
MATCHING OF VENTILATION & PERFUSION AFFECTS THE PARTIAL PRESSURE OF RESPIRATORY GASES

IN THE ALVEOLUS & THE PULMONARY CAPILLARY



ideal unit= $\dot{V}/\dot{Q}=1$

What if blood flow is obstructed only partially?



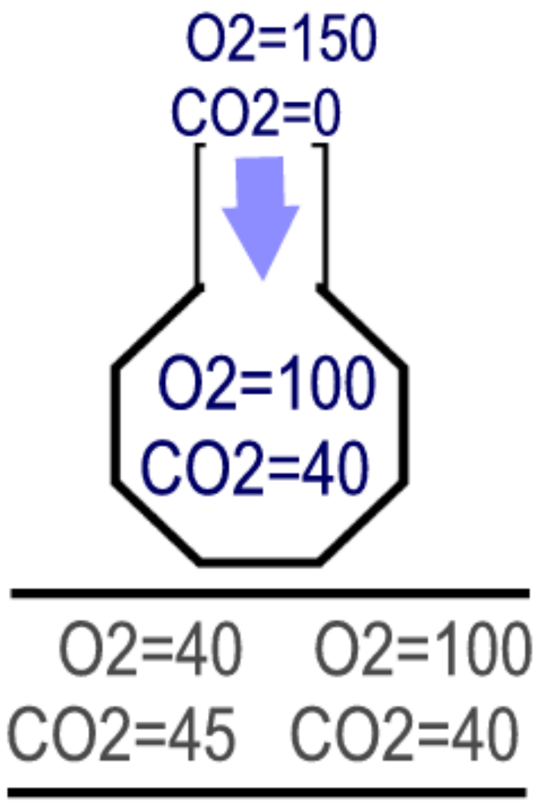
alveolar dead space= $\dot{V}/\dot{Q}=1/0=\infty$

ALVEOLAR DEAD SPACE

- Is the portion of breathing that reaches the alveoli
- Does not participates in gas exchange because of inadequate perfusion to the alveolus
- Insignificant in healthy lungs
- Important in disease states [e.g. pulmonary embolus]

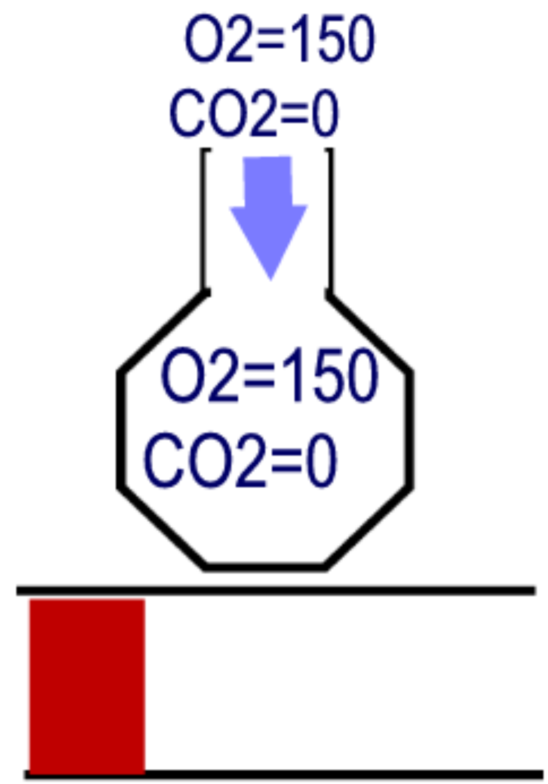
PARTIAL PRESSURE OF RESPIRATORY GASES

IN THE ALVEOLUS & PULMONARY CAPILLARY



ideal unit= $\dot{V}/\dot{Q}=1$

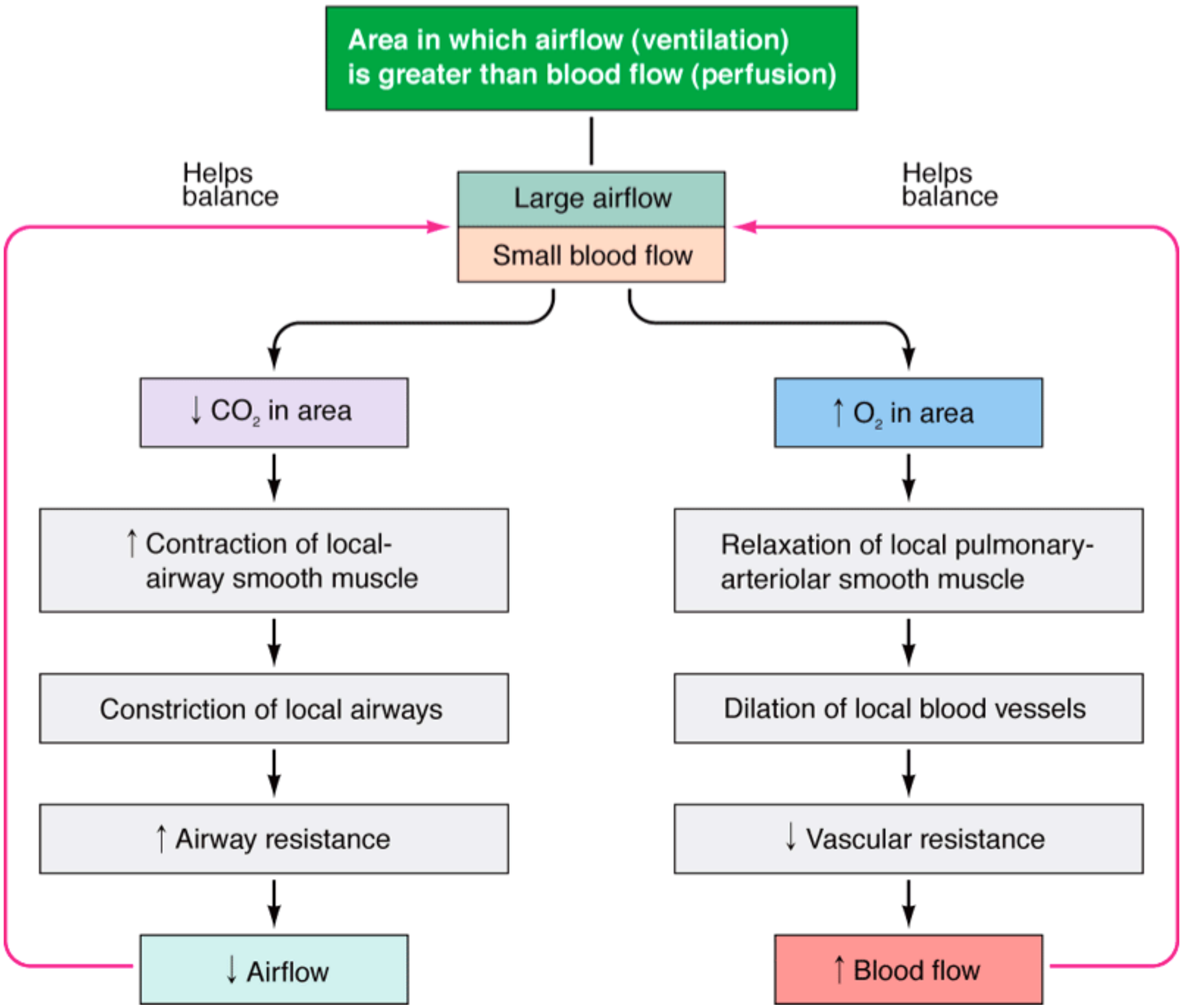
What if blood flow is obstructed only partially?

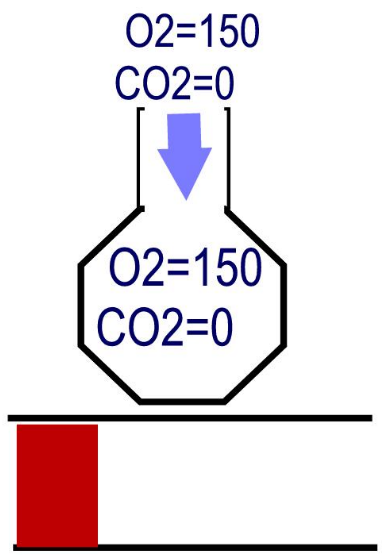


alveolar dead space= $\dot{V}/\dot{Q}=1/0=\infty$

HOMEOSTASIS--VENTILATION & PERFUSION EQUALITY

O2 & CO2 LEVELS AFFECT AIRWAY & BLOOD VESSEL SIZE

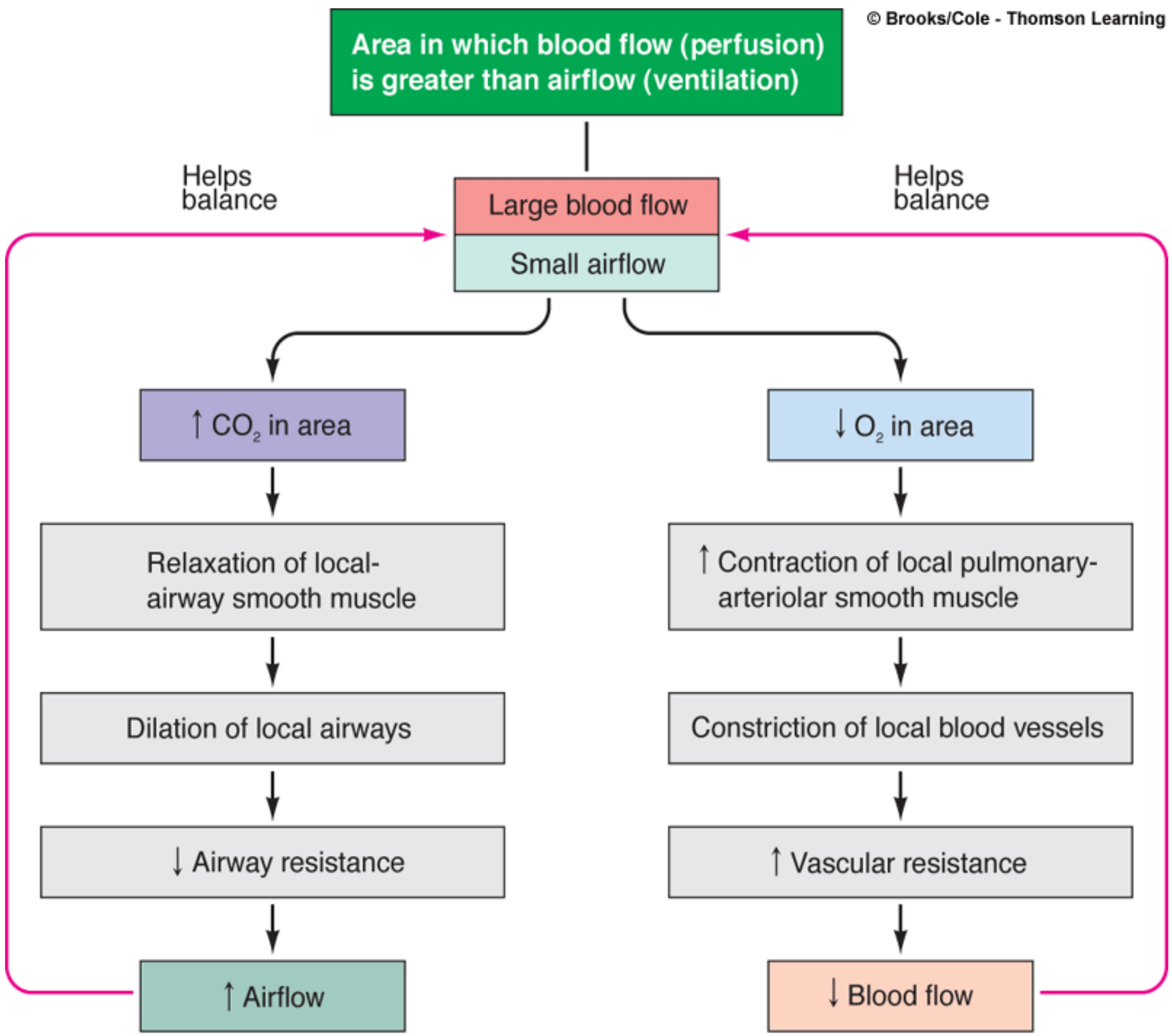


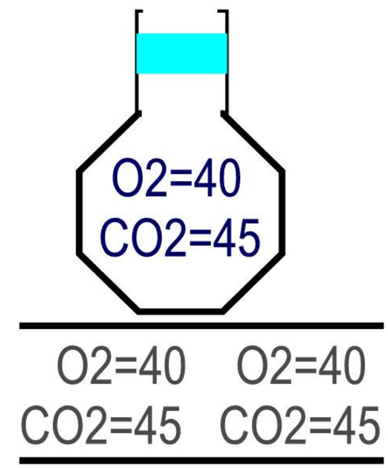


Dead Space
V/Q=infinity

HOMEOSTASIS-VENTILATION & PERFUSION EQUALITY

O2 & CO2 LEVELS AFFECT AIRWAY & BLOOD VESSEL SIZE





"Shunt Like"
V/Q=0

SUMMARY OF CORE CONCEPTS

1. Most static lung volumes can be measured with spirometer, a device that allows plotting lung volume against time. There are four static lung volumes and the combination of these give rise to four lung capacities.

2. In restrictive lung disease, the volume of air entering the lungs is diminished. In obstructive lung disease, airflow out of the lungs during maximal expiratory maneuvers is diminished.

3. Breathing pattern determines alveolar ventilation and the amount of air available for gas exchange.

4. Matching of ventilation and perfusion ensures proper gas exchange. In health, the effect of O_2 and CO_2 on local smooth muscle of airways and pulmonary vessels minimizes ventilation perfusion mismatch.