Ventilation

"…it was concluded from an ASA closed claim analysis study (Tinker *et al*. Anesthesiology 1989;71:541-6) that the application of capnography and pulse oximetry together could have helped in the prevention of 93% of avoidable anesthesia mishaps"

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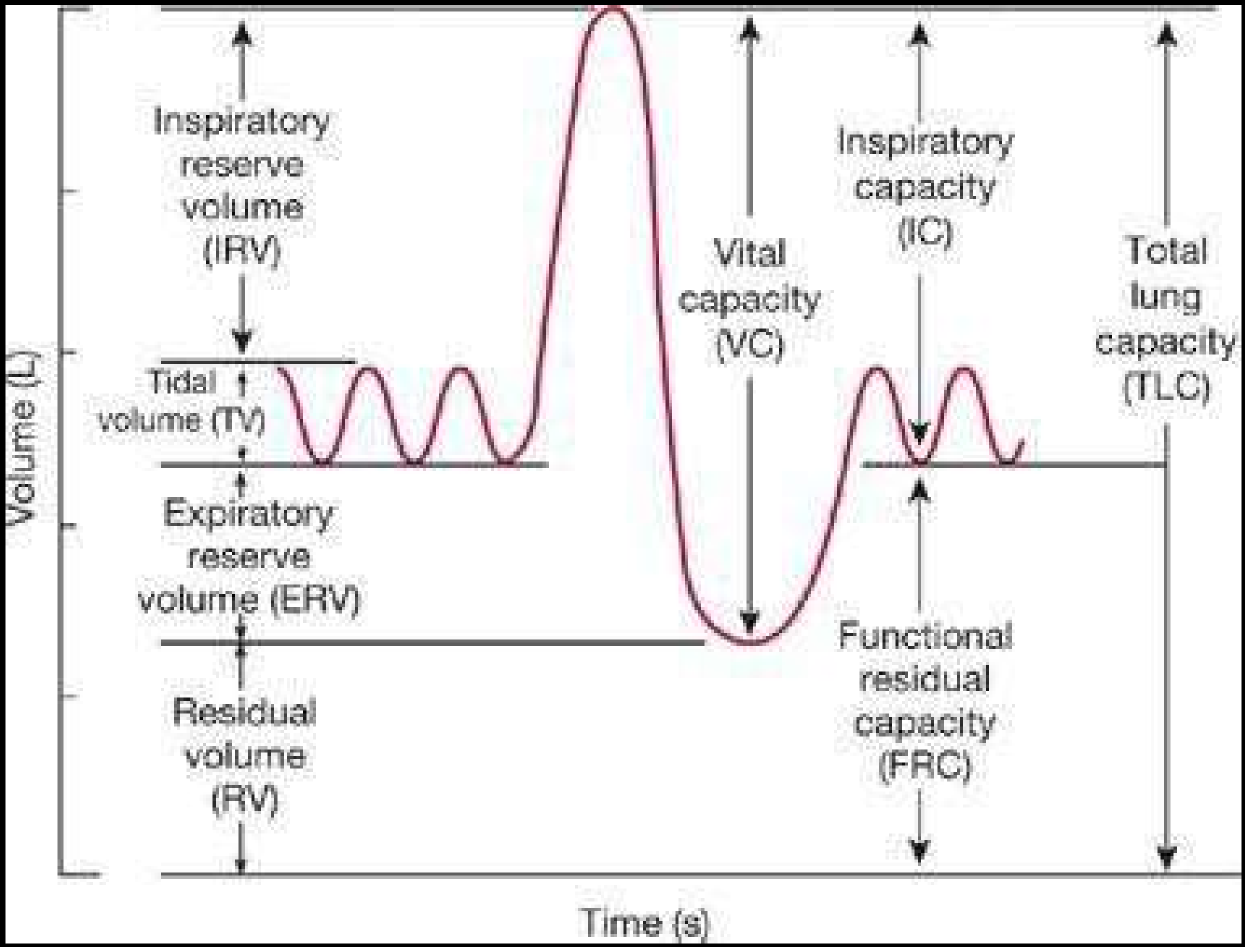
Objective

Understand physiology of ventilation, monitoring it, and intervening as needed

Ventilation

- **A mechanical process -** Accomplished by the work of respiratory muscles
- Movement of gas between the atmosphere and respiratory system, to include the conducting airways and alveoli
- Ventilation maintains homeostatic levels of blood oxygen, carbon dioxide, and pH to facilitate normal cellular function

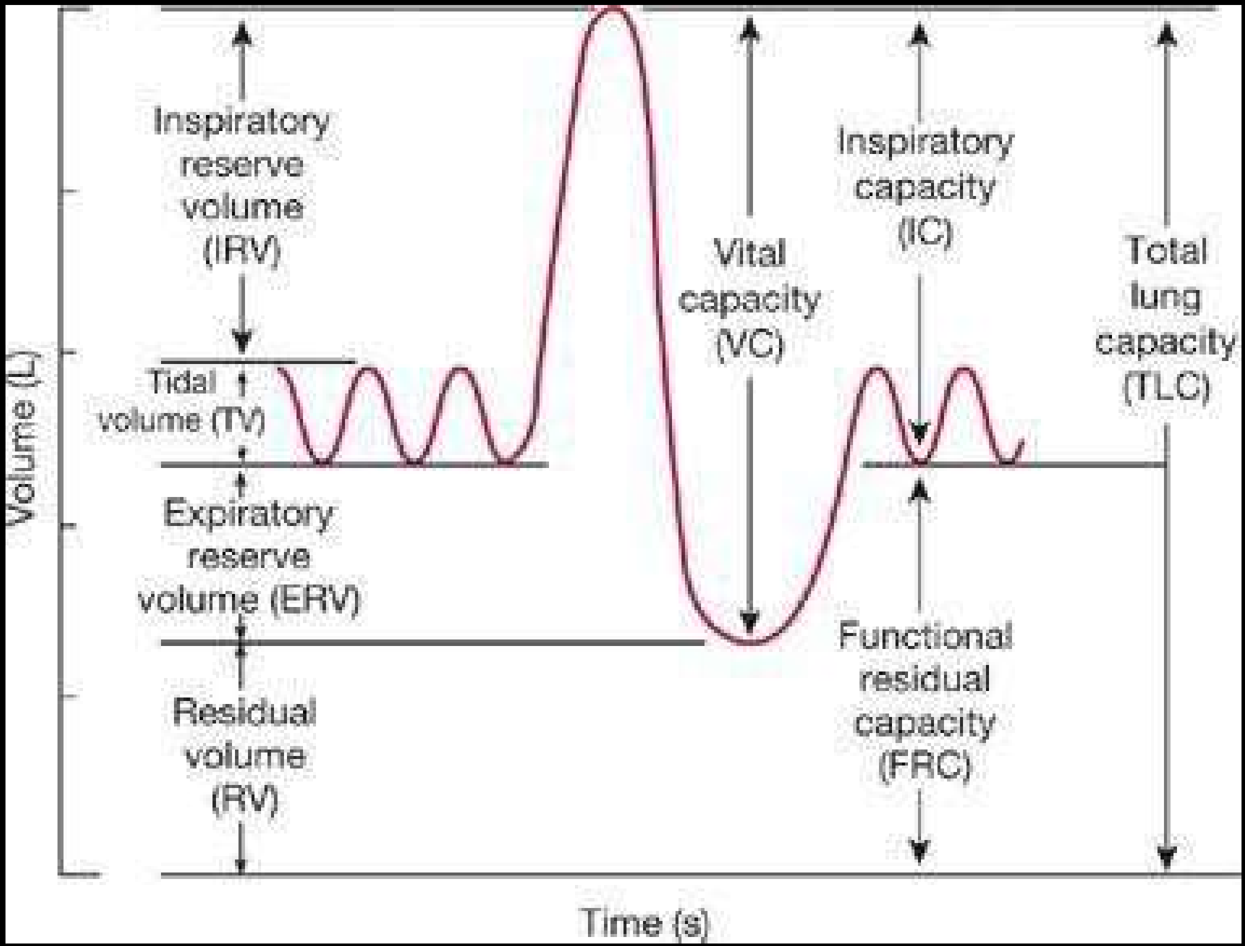


Ventilation

- Ventilation is the mechanical *effector* by which the brainstem maintains homeostasis.
- A measurable process

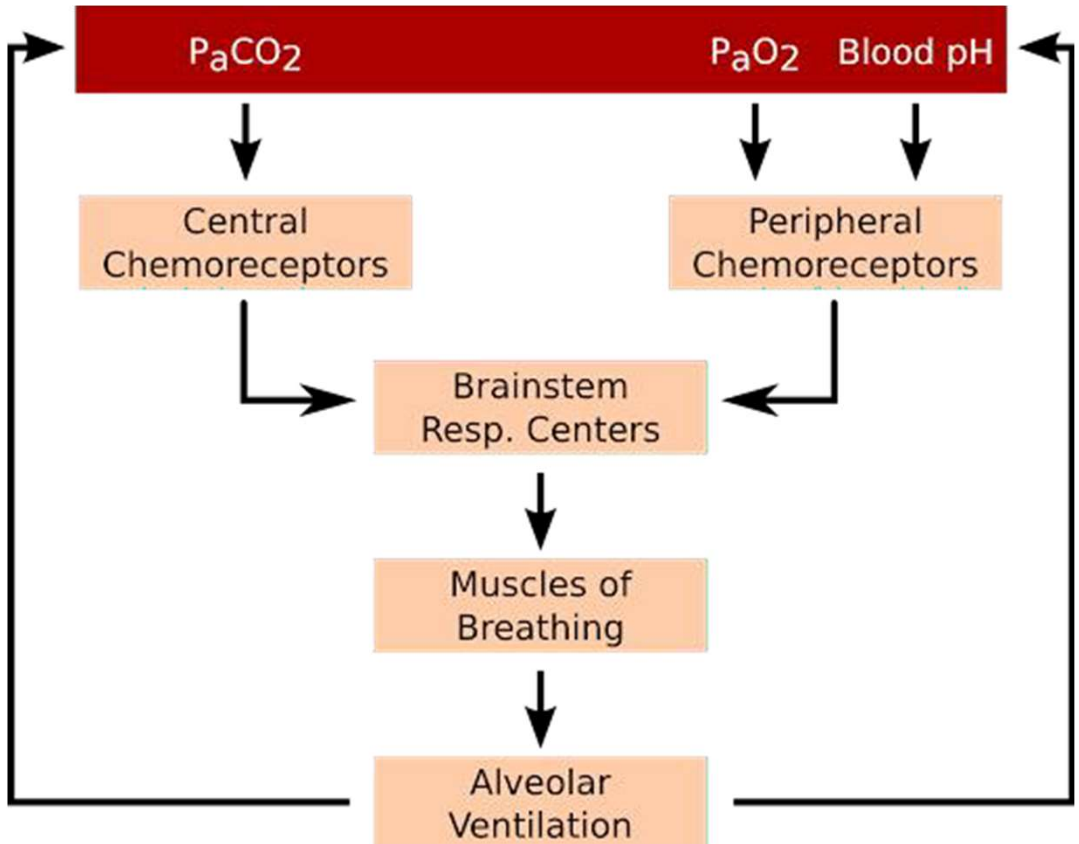
Minute ventilation (V_E):

- Amount of gas moved in/out of respiratory system in one minute
- **V_E = Tidal volume* (V_T) x respiratory rate (RR)**



Ventilation
…mechanical process:

- **Accomplished** through the work of respiratory muscles
- **Controlled** by the respiratory center in the medulla which controls:
  - Respiratory rate
  - Ventilatory rhythm
  - Breath size (tidal volume)
- **Influenced** by acid-base status (blood pH) and partial pressure of oxygen and carbon dioxide*

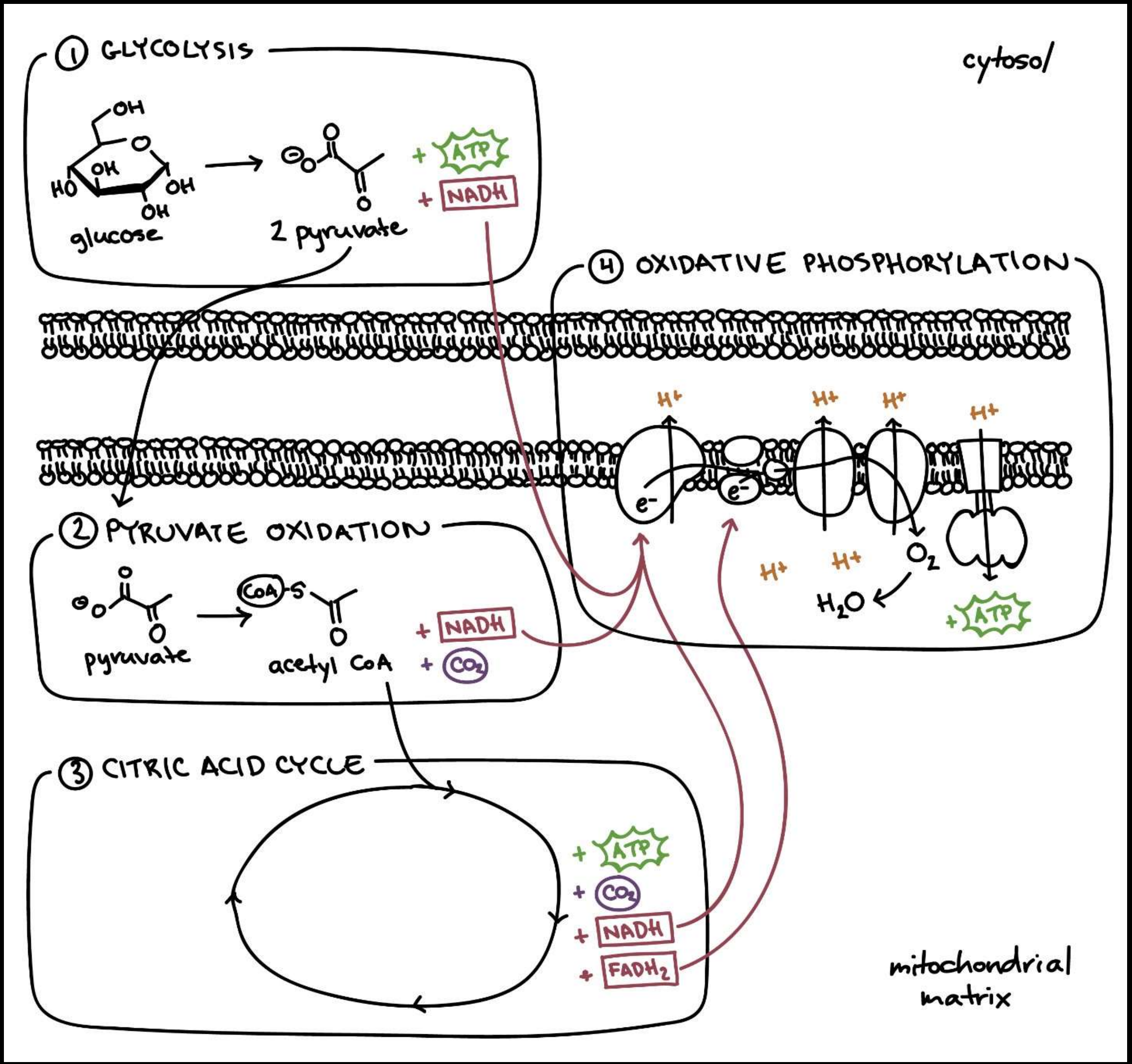


*Carbon dioxide most influential factor on ventilation in health

Carbon dioxide (CO_2)

- **Significant influence on V_E**

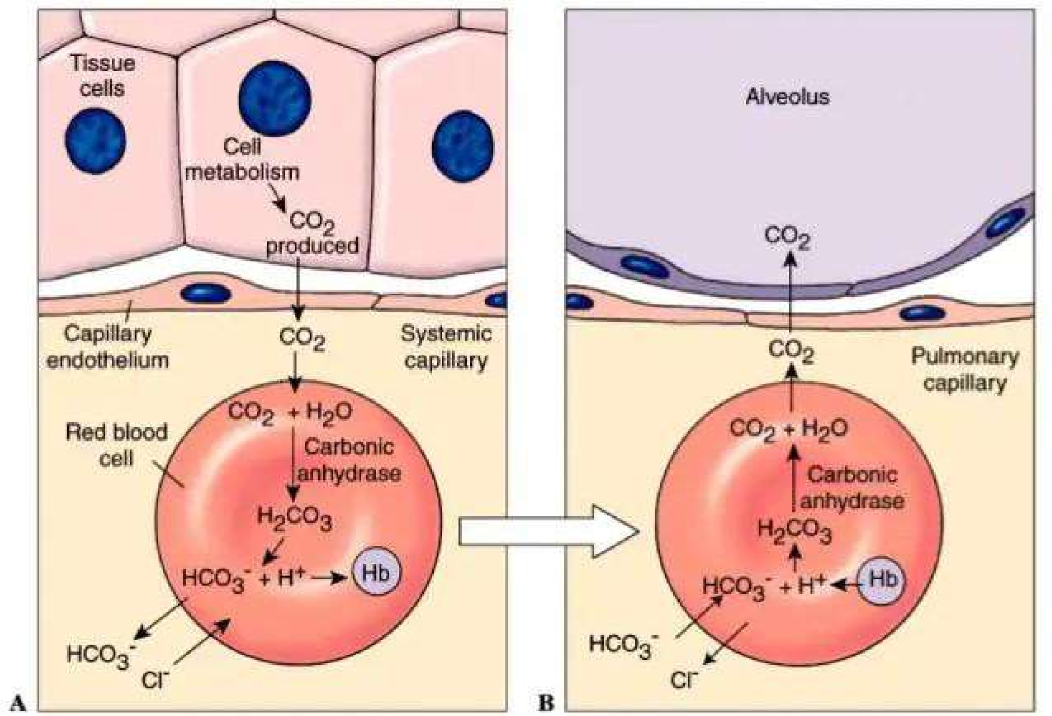
- Sources of CO_2
  - Aerobic respiration
  - Anaerobic respiration



Carbon dioxide (CO_2)

- Bicarbonate buffer system* primary means of CO_2 transport in blood

- CO_2 is ±20x more diffusible than O_2
  - = $PaCO_2$ directly proportional to V_E
  - = If there is effective ventilation, then CO2 will be eliminated



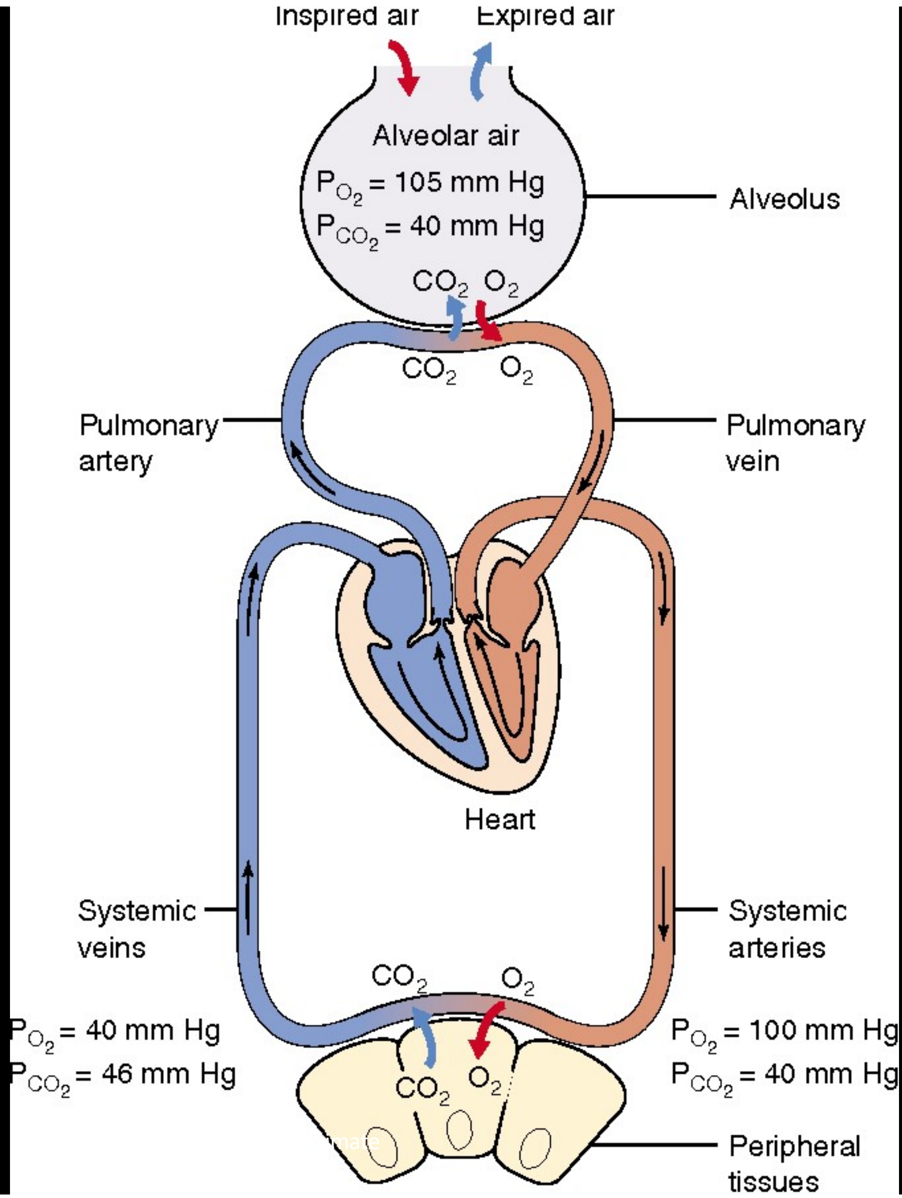
*Bicarbonate buffer system facilitates CO_2 carriage and delivery to alveolus for elimination

**must also be effective cardiac output and delivery of CO2 to the lungs for elimination

Carbon dioxide (CO_2)

- Significant influence on V_E
- Partial pressure reflects effective ventilation
  - How well CO2 is being eliminated (mechanical process)
  - Ventilatory drive from brainstem (stimulatory process)

- Hypoventilation = elevated PCO_2
  - Reduced V_T, RR, or both
- Hyperventilation = reduced PCO_2

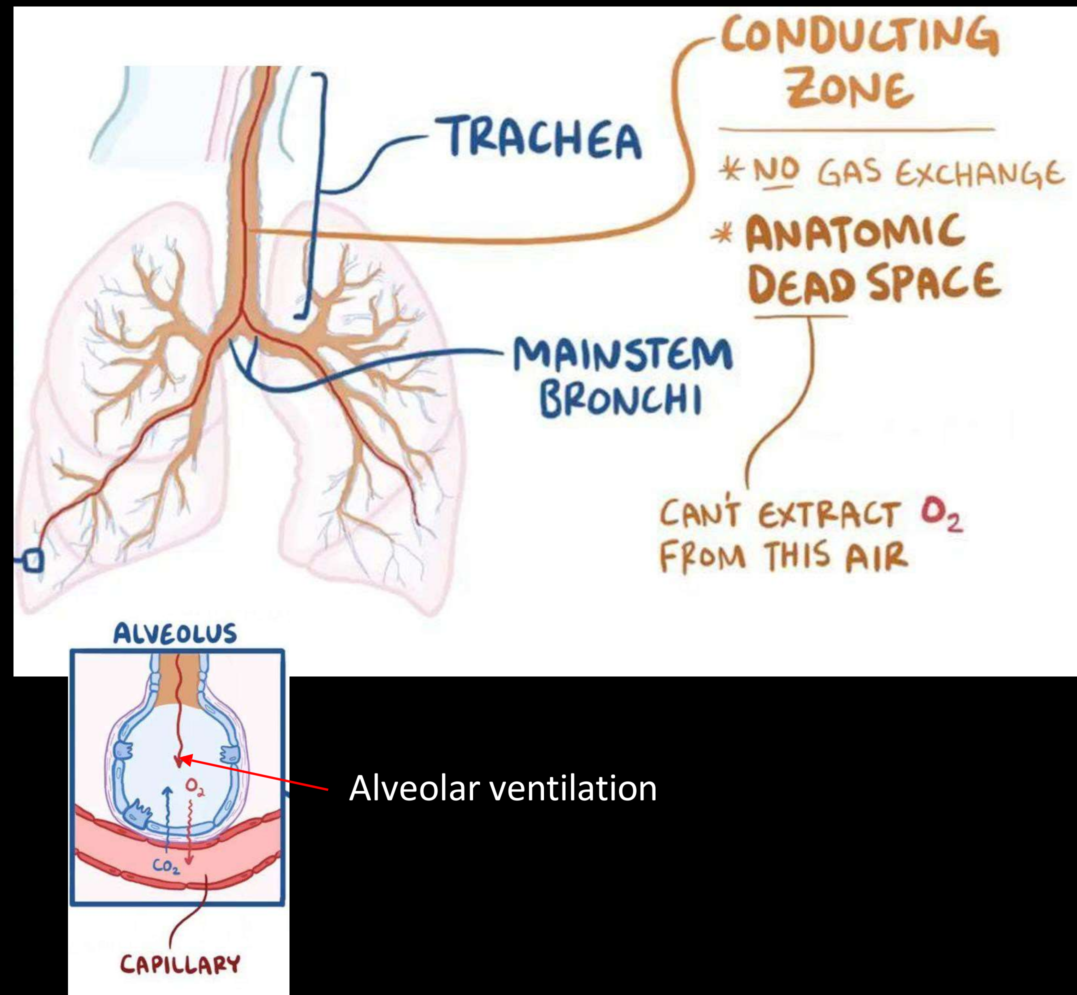


Anatomy of a breath

Not all ventilated gas is the same

V_D:V_T ratio – Proportion of each breath *not* participating in gas exchange

- Dogs, cats, humans: ~ 30-35%
- Horses, ruminants: ~40-50%



Alveolar ventilation

Anatomy of a breath

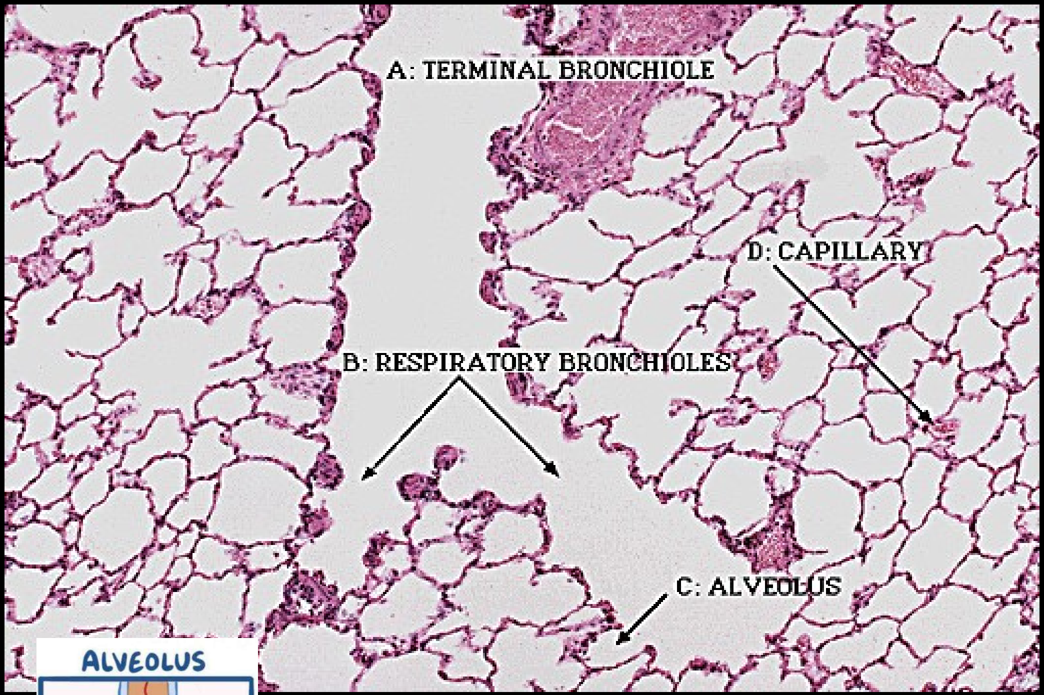
Not all ventilated gas is the same

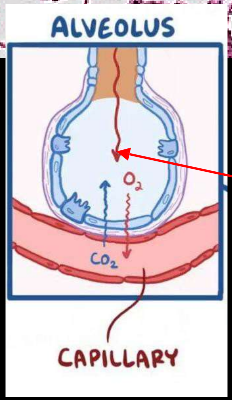
- **Alveolar ventilation (V_A)** – gas that enters the respiratory system and does participate in gas exchange (*effective ventilation*)
  - Respiratory/alveolar bronchioles
  - Alveoli

$$V_A = V_T - V_D$$

Alveolar *minute* ventilation

$$\text{Alveolar}V_E = (V_T - V_D) \times RR$$

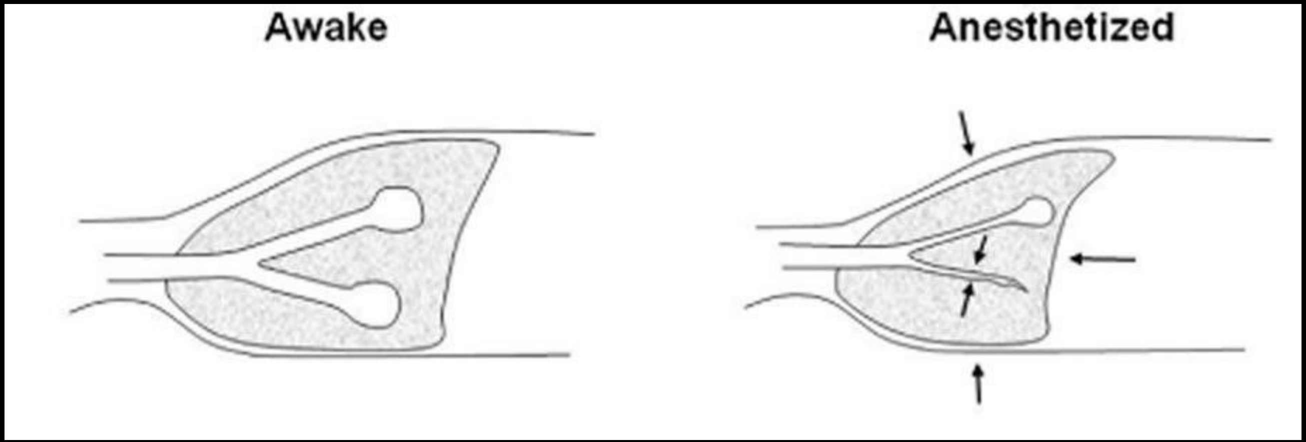




Alveolar ventilation

*Amount of gas moved in/out of respiratory system in one minute, that is effective and involved in gas exchange

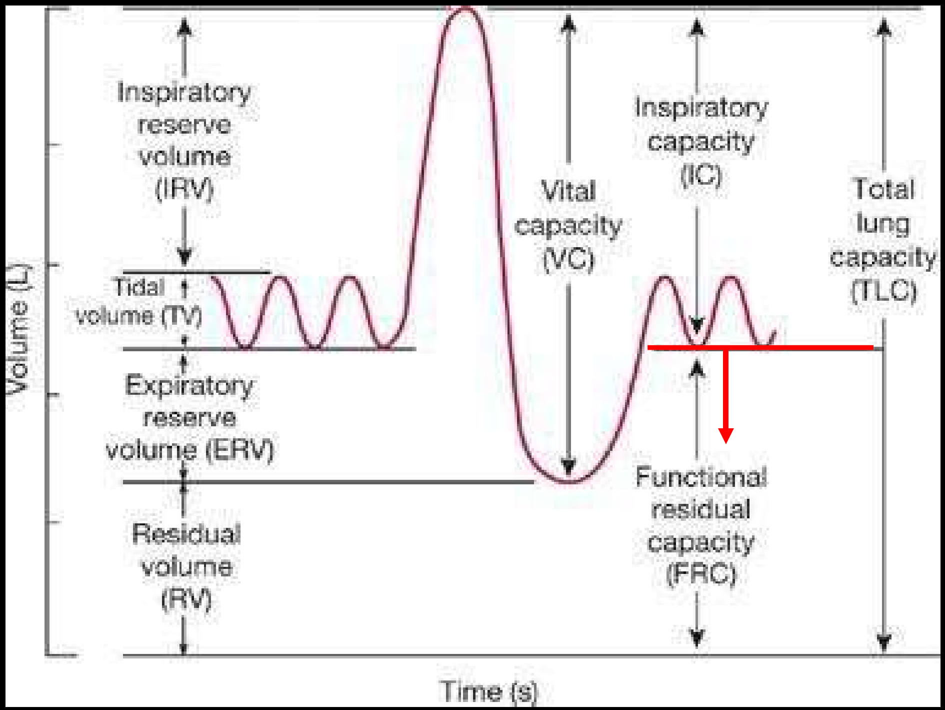
Major effects of anesthetics on ventilation

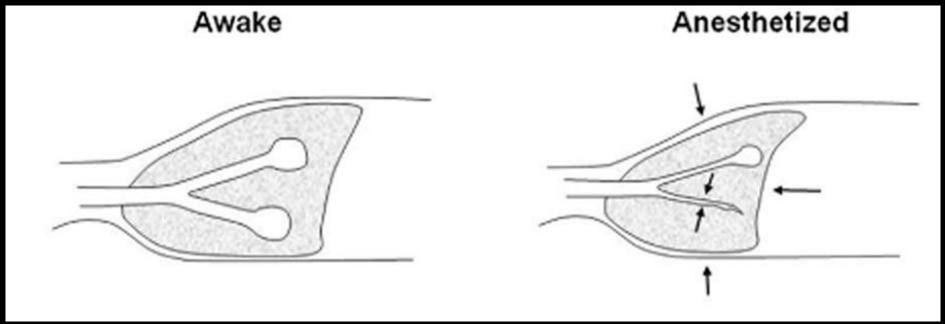


1. Altered static lung volumes
2. Respiratory depression

Effects on static lung volumes

- Muscle relaxation upon induction of anesthesia
  - Progressive reduction in primary & accessory respiratory muscle (e.g., intercostals) function with increased depth, additional drugs producing respiratory depression
  - Tidal volumes reduced in size
- Reduced functional residual capacity (FRC) – less volume in lungs following normal exhalation
  - Reduced gas exchange efficiency
  - Reduced time to desaturation

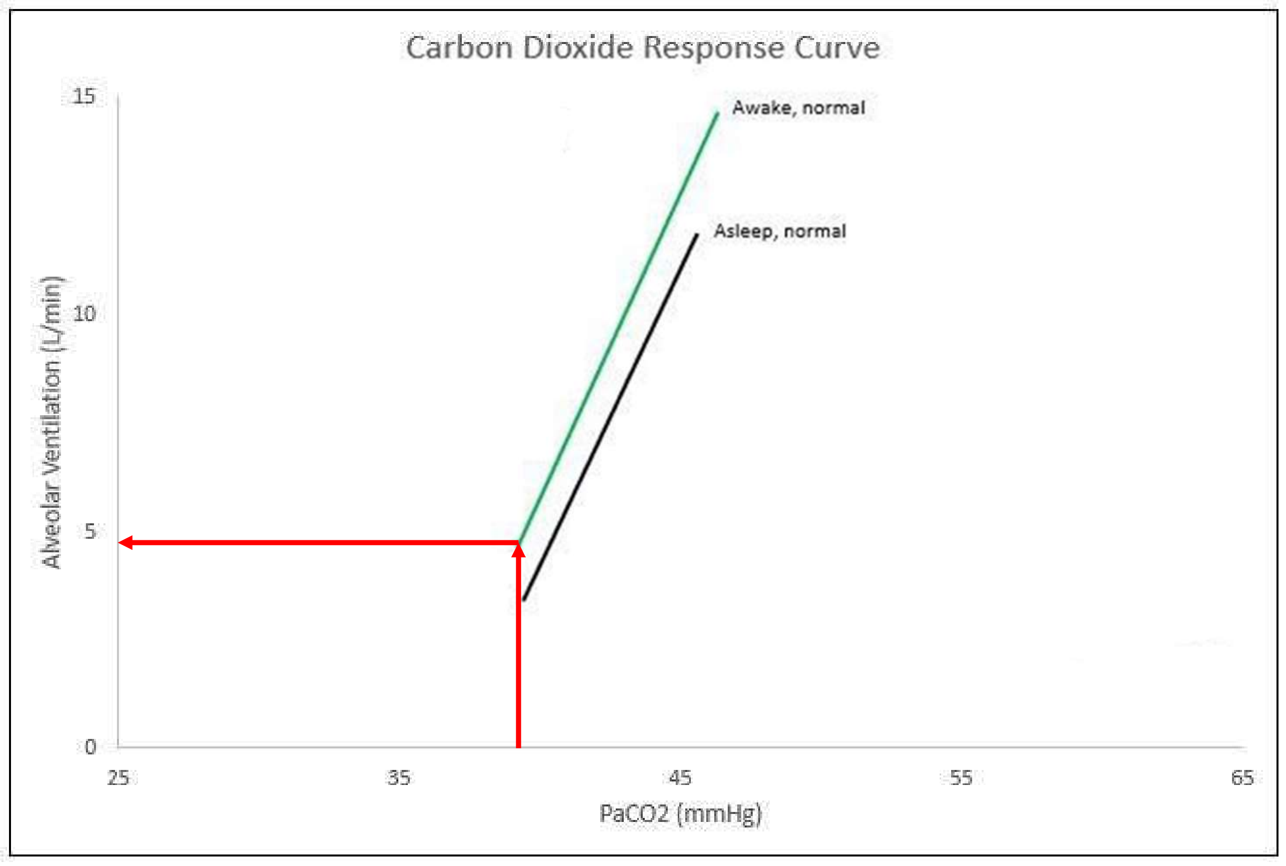




Respiratory depression

ventilatory (CO_2) response curve

A representation of the physiologic effect* $PaCO_2$ has on alveolar minute ventilation.

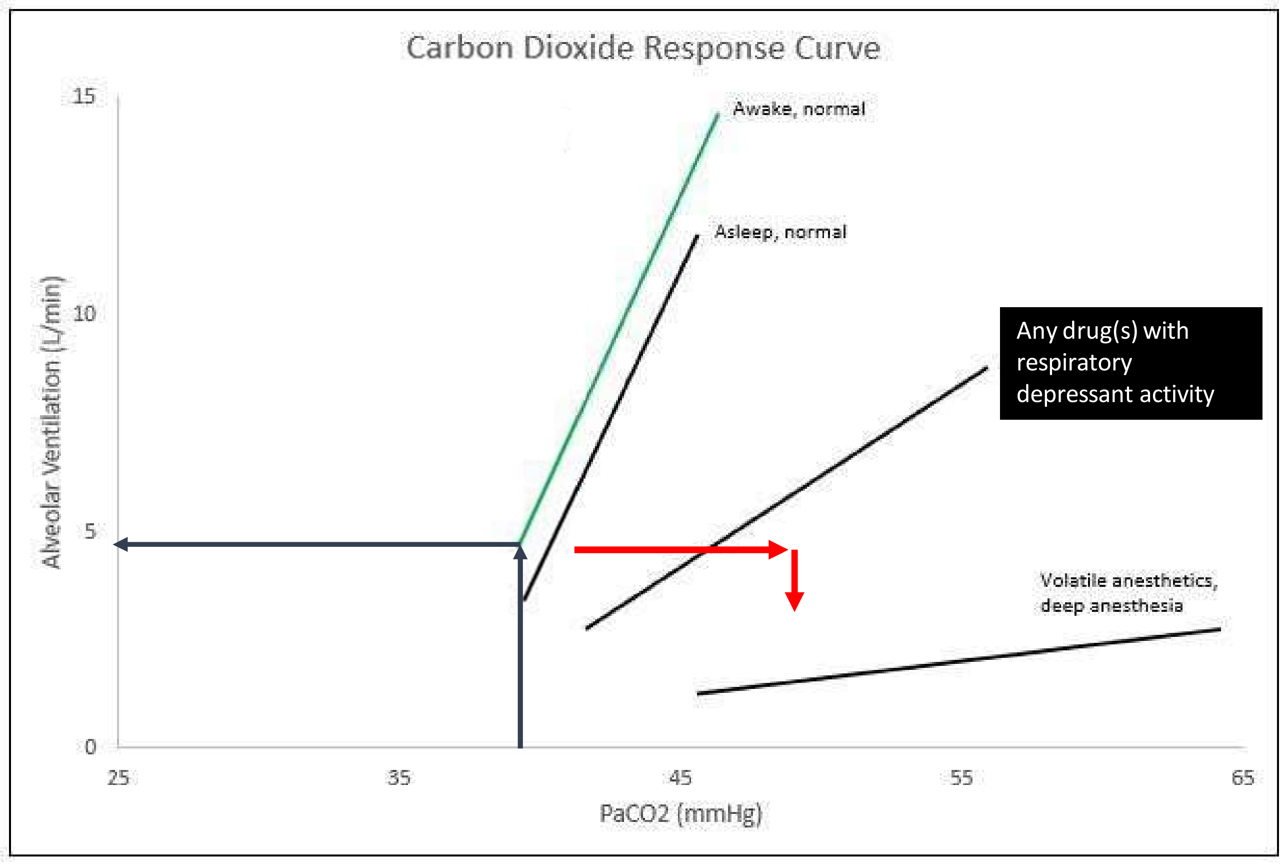


*via the central chemoreceptors, stimulating the respiratory system, which then augments tidal volume and respiratory rate in order to maintain PaCO2 in a normal range

Respiratory depression

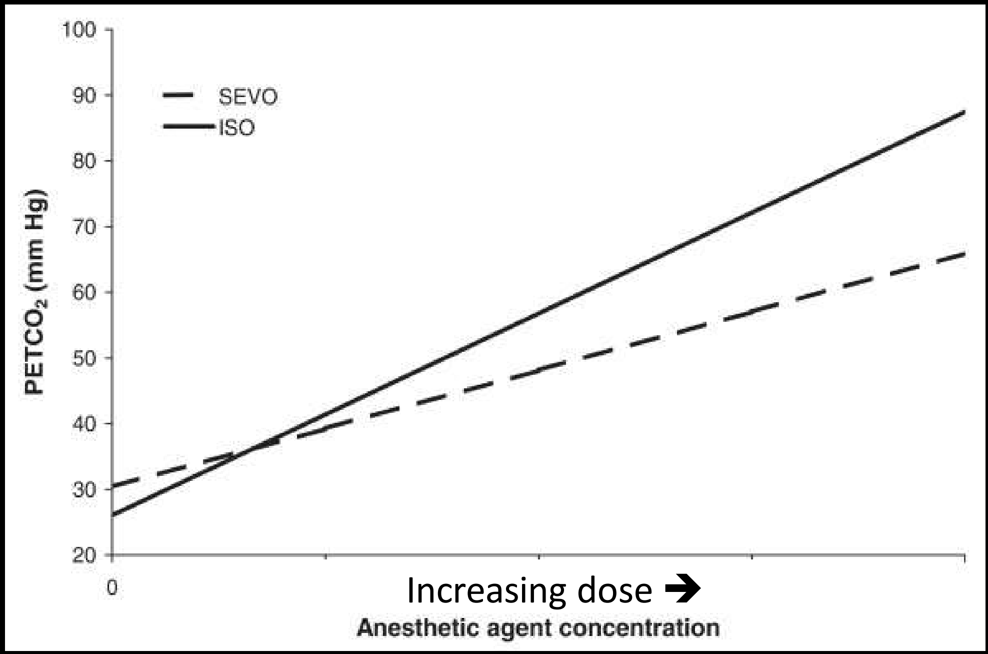
Reduced central sensitivity to CO_2 whereby a higher $PaCO_2$ is required to produce the same relative alveolar minute ventilation

Caused by numerous drugs with respiratory depressant effects



Respiratory depressants

- Reduce the sensitivity of central chemoreceptors to CO_2.
- Results in the ventilatory response relationship shifting right and down*
  - Reduced RR, V_T, or both
  - Increased $PaCO_2$, $PvCO_2$
- Most analgesics/anesthetics
  - Opioids
  - Ketamine
  - Propofol, alfaxalone
  - Volatile anesthetics
- Additive effects



Parameter	Awake	Light plane of anesthesia	Deep(er) plane of Anesthesia
PaCO2 (mmHg)	±30	±40-45	60-70+
PvCO2 (mmHg)	33-35	±45	65-75+
pH	~7.4	~7.32	7.18-7.24

*meaning baseline PaCO2 increases and when PaCO2 increases further, there is a less substantial ventilatory response

If you do not monitor it, is there a problem?

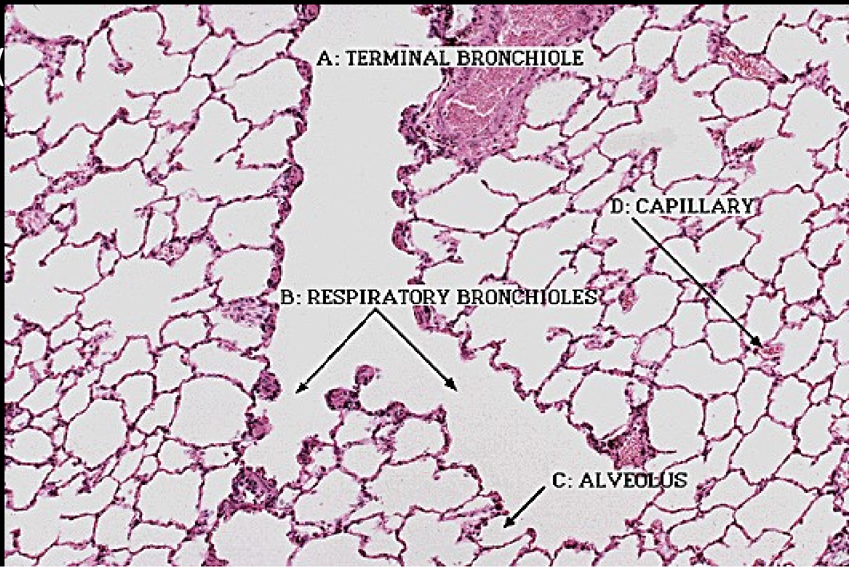
Why we care about ventilation/CO2

…systemic effects of hypoventilation

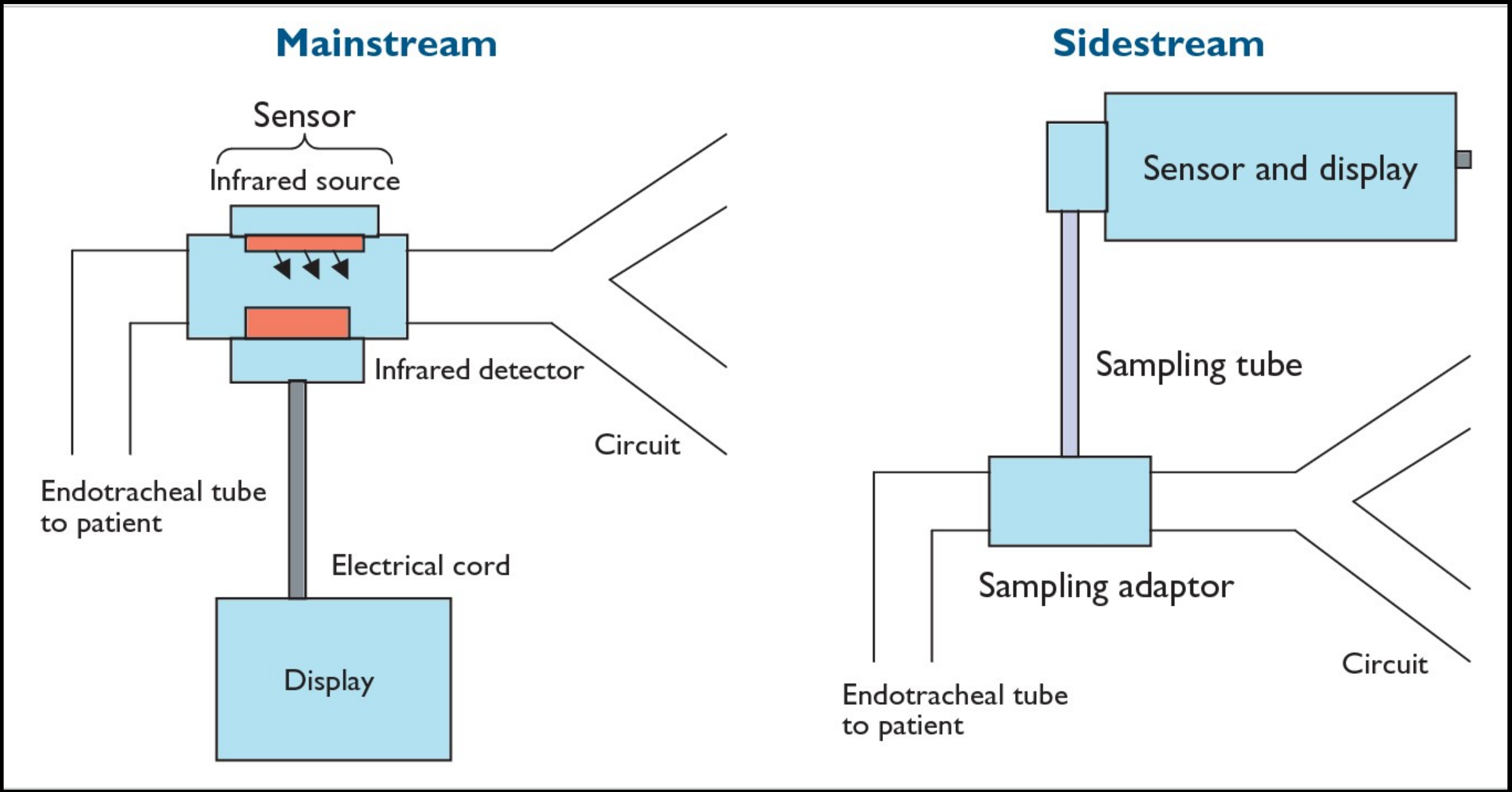
- Acid-base/electrolytes
  - Proportional respiratory acidosis with increased PCO_2
  - Increased plasma [K] over time
  - Exacerbates concurrent metabolic acidosis causing disease process
- Cardiovascular
  - Peripheral vasodilation, negative inotropy
  - Sympathetic nervous system stimulation
  - Increased circulating catecholamines – risk for arrythmias
- CNS
  - Depression at higher PCO_2 (>80-90 mmHg)
  - Cerebral blood flow increases (4% increase/mmHg increase)
  - Prolonged elevation (>80-90 mmHg), cerebral edema
- Respiratory
  - Pulmonary vascular constriction (increased right heart work)

Capnography

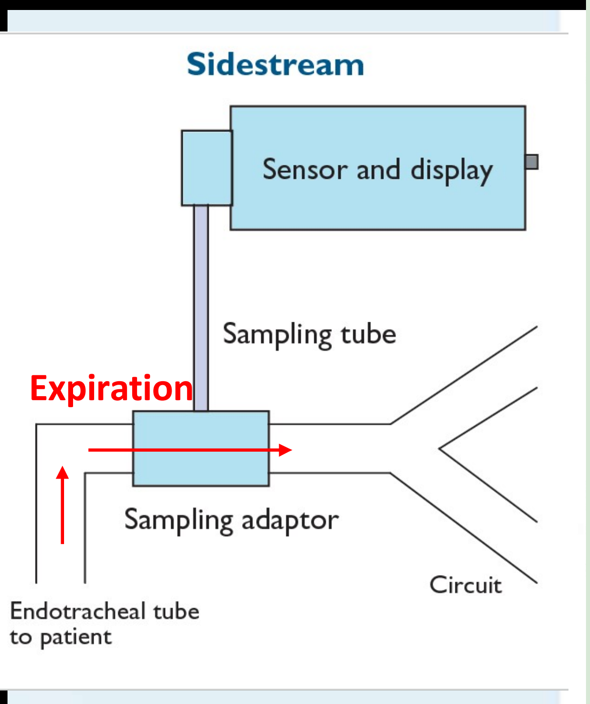
- Measures CO2 from expired breaths
- End-Tidal gas sampled at the end of expiration
  - Reflects alveolar PCO2, which reflects arterial PCO2
  - EtCO2 always 3-5 mmHg < arterial PCO2
- Continuously measures EtCO2 after each breath
  - Assess ventilation status of patient (hypo, hypervent. etc.)
  - Assess cardiovascular system – must pump blood (CO2) to the lungs for elimination
  - Assess patency of airway (endotracheal tube)



Capnographs – two types

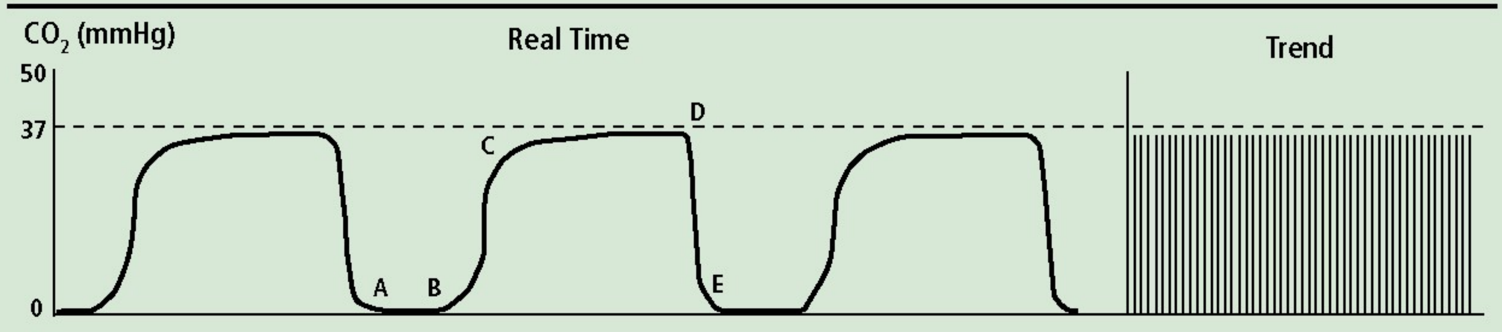


Capnography



Normal capnogram

Normal $EtCO_2$: 35–45 mmHg



The 'normal' capnogram is a waveform which represents the varying CO_2 level throughout the breath cycle.

Waveform Characteristics

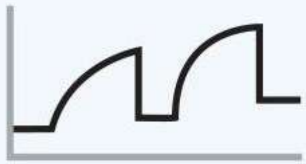
A-B	Baseline
B-C	Expiratory upstroke
C-D	Expiratory plateau
D	End-Tidal concentration
D-E	Inspiration

https://www.capnography.com/equipment-malfunction/

Capnography

BRONCHOSPASM AND REBREATHING/AIR TRAPPING

- Increase or loss of α-angle (aka "shark fin")
- Dead space has not finished emptying before next inspiration
- Increasing level of baseline P_ECO_2 due to air trapping

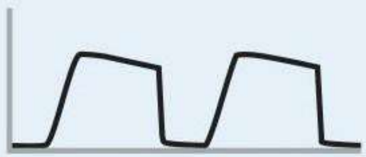


MECHANICAL AIRWAY OBSTRUCTION

- Fixed mechanical obstruction affects both inspiration (phase IV/0) & expiration (phase II)
- α-angle and β-angle both >90°

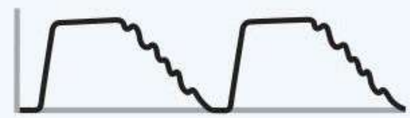
EMPHYSEMA

- Arterial CO_2 represented by early peak, not end-tidal, due to hypercompliance and poor gas exchange surface
- Pattern can also be seen with pneumothorax with air leak



CARDIOGENIC OSCILLATIONS

- Pulsation transmitted from the heart to the lung parenchyma produces small volume changes that manifest as oscillations
- Sign of cardiomegaly

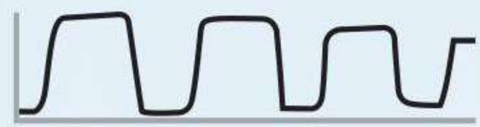


SUDDEN LOSS OF WAVEFORM

- Critical event needing emergency intervention
- ET tube disconnected, dislodged, kinked, or obstructed

DOWNTRENDING $ETCO_2$

- Decreasing waveform size can indicate:
  - Shock/low cardiac output state
  - Pulmonary embolism
  - Hyperventilation



Assessing hypoventilation under GA?

Parameter	Awake	Light plane of anesthesia	Deep plane of anesthesia
PaCO2 (mmHg)	±30	±40-45	60-70+
PvCO2 (mmHg)	33-35	±45	65-75+
pH	~7.4	~7.32	7.18-7.24

- Healthy patients:
  - $EtCO_2 \leq$ 55-60 mmHg is tolerated safely
  - Exceptions exist*, more when on clinics
- Patient hypoventilating?
  - Ensure adequate plane of anesthesia (last lecture)
  - Hypoventilation worsens with deeper planes of anesthesia (previous slide)
- We monitor and record EtCO2 measurements, just like SpO2, HR, RR, BP etc. q5 min

*pulmonary hypertension, some right heart disease intracranial disease, concurrent metabolic acidosis, hyperkalemia

Intermittent positive pressure ventilation (IPPV)

'giving breaths'

- Technique by which short term/intermittent mechanical ventilation may be supplied to a patient, augmenting delivery of oxygen/anesthetic gases and removal of CO_2.
- Not every patient requires it, but many anesthetized patients do!
- Requires endotracheal intubation

- Indications*:
  - Hypoventilation (above cut-off previously discussed) / apnea
  - Management of anesthetic depth
  - Disrupted thoracic wall/diaphragm (e.g., thoracotomy), loss of pleural pressure
  - Neuromuscular blocking agents

*Not exhaustive list

Administering manual IPPV

- Close pop-off valve
- Squeeze re-breathing bag over 1-1.5 seconds while:
  - Looking at patient (undraped patient) – breath should appear as normal breath
  - Pressure manometer (draped patient) – administer to 10-12 mmHg peak inspiratory pressure (PIP)
- ‘Release’, *open pop-off valve*

Repeat as needed to maintain appropriate EtCO2/depth of anesthesia

Giving an ‘extra’ breath once a minute does not help

(approximate) Appropriate respiratory variables for IPPV in the anesthetized patient

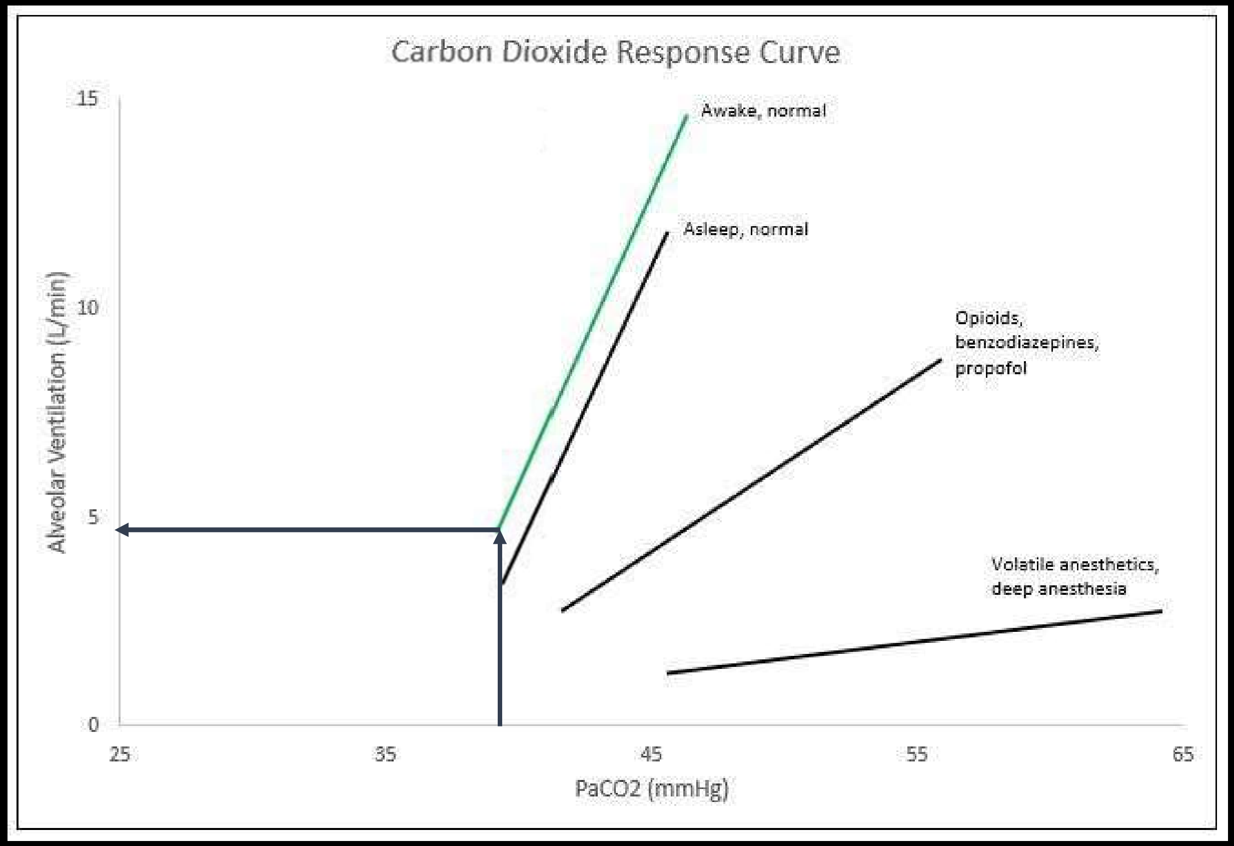
Parameter	Dog	Cat
VT (ml/kg)	8-12	
RR (bpm)	8-20	10-20
Inspiratory time (s)	1-1.5	
Peak inspiratory pressure (cmH2O)	8-12	5-8
EtCO2 (mmHg)	45-55	

Not truly taking over ventilation when/if needed –

‘the dog’s CO_2 was getting high so I have been giving an extra 2 breaths a minutes’

‘the dog’s respiratory rate was low so I gave…’

Not truly taking over ventilation when/if needed –

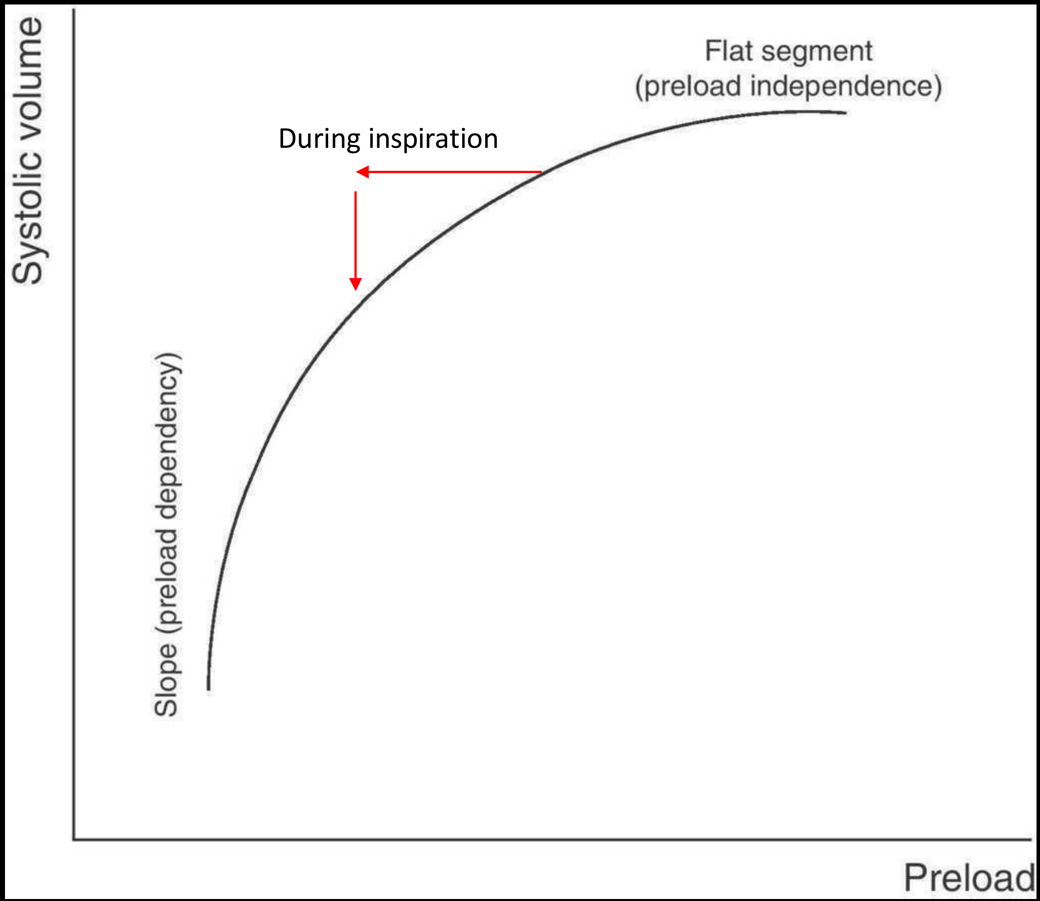


Implications of IPPV

- Not benign – completely altered mechanism by which gas enters respiratory system

- Positive intrathoracic pressure
  - External compression of low-pressure venous vessels
  - Reduced venous return (preload) during inspiration
  - Reduced cardiac output and subsequent blood pressure

- *Barotrauma easily incurred



Questions for consideration

1. Assuming V_D is fixed, using a 10 kg beagle with V_D:V_T of 0.35 and a resting (normal) V_T of 8 ml/kg as an example.
   - What is this dogs deadspace in ml/kg?
   - Calculate this dog's alveolar minute ventilation if the V_T is reduced to 4 ml/kg, assuming a RR of 10 breaths per minute?
   - If this patient continues to have tidal volumes of 4 ml/kg, will this dogs $PaCO_2$ increase, decrease, or stay the same – and why?
2. Assume the dog in question 1 has a respiratory compliance of 13 ml/cmH2O; what would the peak inspiratory pressure have to be in order to deliver a 12 ml/kg V_T?
3. A 5 kg cat has a respiratory compliance of 10 ml/cmH2O, what would the peak inspiratory pressure have to be in order to deliver a 48 ml V_T?

Automatic mechanical ventilators

- A ventilator is an automatic device which is designed to provide or augment patient ventilation and take over the function of manually administering IPPV
  - Can set patient-specific respiratory rate, inspiratory time, and V_T.
  - Stabilizes each breath delivered to the patient
  - Improves anesthesia provider attention to patient

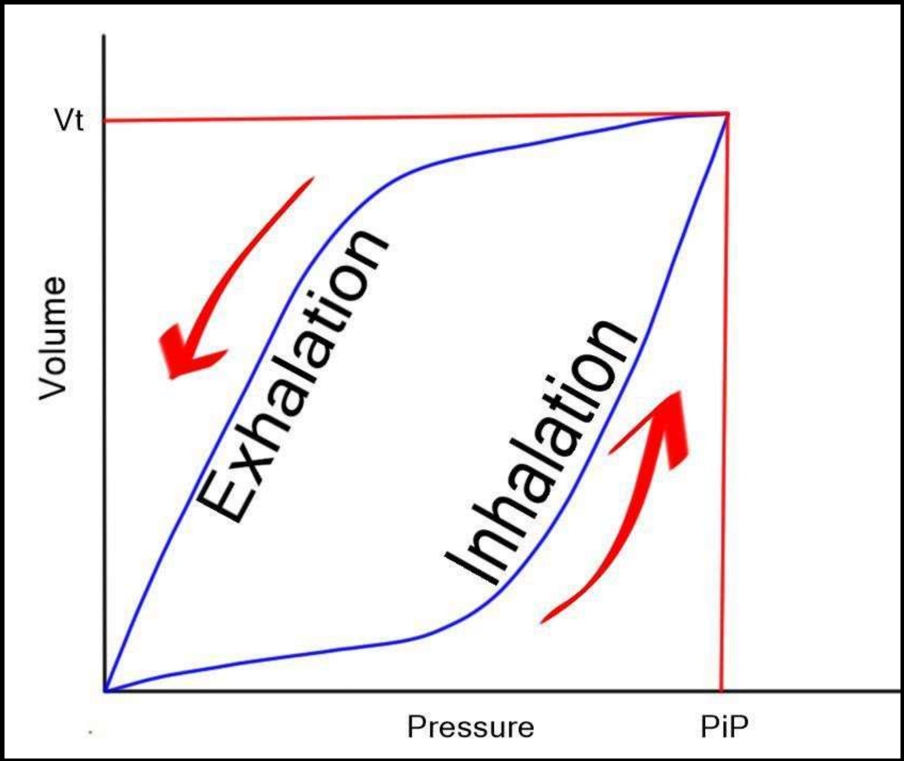
Basics of mechanical ventilator function

- Hallowell ventilators
  - Commonly used in veterinary medicine
  - Electronically powered
  - Pneumatically driven
  - Pressure limited
  - Different bellows sizes: 0.3 L, 1.6L, 3 L
- Numerous ventilators available
- *Not* cost-prohibitive

Questions?

Peak inspiratory pressure (PIP)

- The maximum pressure achieved within the anesthetic circuit while administering a positive pressure breath.
  - Estimates the pressure within the thoracic cavity
  - Important to monitor when administering IPPV
- Appropriate PIP when delivering IPPV
  - Average adult dog: 10-12 cmH2O
  - Average adult cat: 5-8 cmH2O
  - Puppy/kitten: 5-8 cmH2O
  - Adult horse: 20-30 cmH2O
  - Adult human: 16-20 cmH2O



Respiratory compliance – determines PIP

The relative distensibility of the lungs for a given change in inspiratory pressure

Compliance = ΔVolume (ml) / ΔPressure (cmH2O)