End Tidal CO2

Note: I'd love to take credit for this lovely article on End Tidal CO2 - but I can't. I copied it off of some manufacturers website in the mid 90's for my own use and, a while later when I went to look for it again, it was gone. I don't know who produced it, but it's a great article. So, I decided I'd risk the horrible lawsuit and post it anyway. If anyone knows who the original authors are I would *love* to give them credit. It just seemed such a shame to not have these great graphics on the internet anymore. Please don't write to me and ask me if you can use this information. I stole it. If you want to steal it too, go ahead, but don't tell anyone I gave you permission to. [Marc]

Introduction

ETCO2 is the partial pressure or maximal concentration of carbon dioxide (CO2) at the end of an exhaled breath, which is expressed as a percentage of CO2 or mmHg. The normal values are 5% to 6% CO2, which is equivalent to 35-45 mmHg. CO2 reflects cardiac output (CO) and pulmonary blood flow as the gas is transported by the venous system to the right side of the heart and then pumped to the lungs by the right ventricles. When CO2 diffuses out of the lungs into the exhaled air, a device called capnometer measures the partial pressure or maximal concentration of CO2 at the end of exhalation. During CPR, the amount of CO2 excreted by the lungs is proportional to the amount of pulmonary blood flow.

Discussion of Theory/Pathophysiology

To understand the significant value of ETCO2, one needs to be familiar with the following:

(1) normal physiology of CO2,
(2) principle determinants of ETCO2,
(3) CO2 gradient with normal VQ relationship,
(4) ETCO2 analyzer (capnometer), and
(5 limitations of ETCO2 measurements.

ETCO2 represents the partial pressure or maximal concentration of CO2 at the end of exhalation. CO2 reflects cellular metabolism. There are four main stages of normal physiology of CO2:

(1) production,
(2) transport,
(3) buffering and
(4) elimination.

Production:
CO2 is a metabolic byproduct of aerobic cell metabolism. As the intracellular CO2 increases, CO2 diffuses out into the tissue capillaries and is carried by the venous circulation to the lungs, where it diffuses from pulmonary capillaries into the alveoli. The partial pressure of CO2 (PaCO2) of venous blood entering pulmonary capillaries is normally 45 mmHg; the partial alveolar pressure of CO2 (PACO2) is normally 40 mmHg. The pressure difference of 5 mmHg will cause all the required CO2 to diffuse out of pulmonary capillaries into the alveoli.

Transport:
The second stage is CO2 transport, which is a way of maintaining the CO2 tension of arterial blood at approximately 35-45 mmHg despite high CO2 production.

Buffering:

The third stage is where the buffer action of hemoglobin and pulmonary blood flow maintain the normal level of CO_2 tension by eliminating the excess CO_2. CO_2 can either be carried, dissolved or combined with water (H20) to form carbonic acid (H_2CO_3), which can dissipate to hydrogen ions (H^+) and bicarbonate ions (HCO_3^-): ($CO_2 + H20 <=> H_2CO_3 <=> H^+ + HCO_3^-$). The hydrogen ions are buffered by hemoglobin, and the bicarbonate ions are transported into the blood. This mechanism accounts for 90% of CO_2 transport.

Elimination:

The fourth stage involves CO_2 elimination by alveolar ventilation under the control of the respiratory center. This process allows the diffusion of CO_2 from blood to the alveoli where the partial alveolar pressure of CO_2 is lower than the tissue pressure.

During normal circulatory condition with equal VQ relationship, $PACO_2$ is closely comparable to $PaCO_2$ and $ETCO_2$; therefore, $PaCO_2$ is equivalent to $ETCO_2$. The difference between $PaCO_2$ and $ETCO_2$ is known as the CO_2 gradient. The normal $ETCO_2$ is about 38 mmHg at 760 mmHg of atmosphere with less then 6 mmHg gradient between $PaCO_2$ and $ETCO_2$.

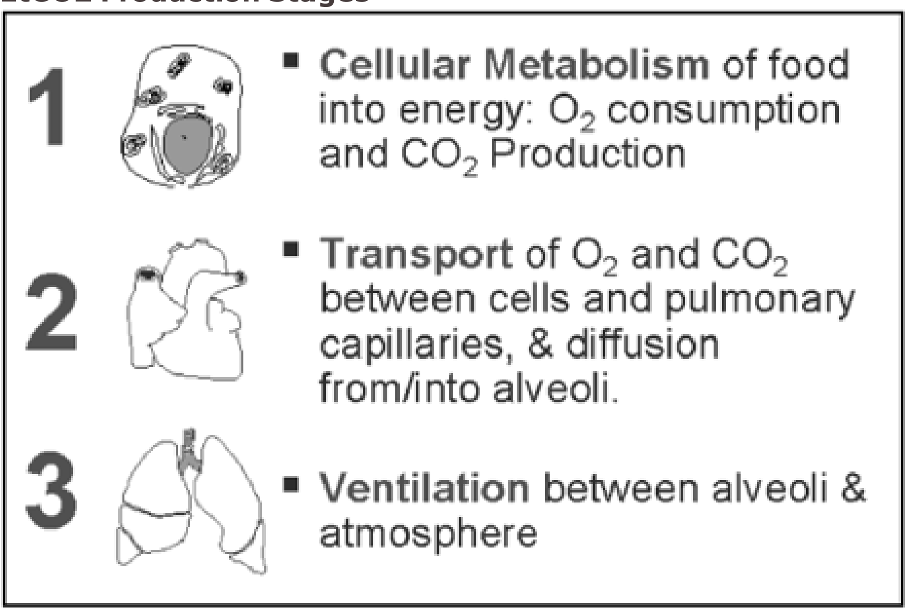
The principle determinants of $ETCO_2$ are:
(1) alveolar ventilation,
(2) pulmonary perfusion (cardiac output) and
(3) CO_2 production.

During acutely low cardiac output state as in cardiac arrest, decreased pulmonary blood flow becomes the primary determinant resulting in abrupt decrease of $ETCO_2$. Changes in alveolar ventilation can also influence $ETCO_2$ as $PACO_2$ closely approximates $PaCO_2$ and $ETCO_2$. If ventilation and chest compressions are constant with the assumption that CO_2 production is uniform, then the change in $ETCO_2$ reflects the changes in systemic and pulmonary blood flow. Ultimately, $ETCO_2$ can be used as a quantitative index of evaluating adequacy of ventilation and pulmonary blood flow during CPR.

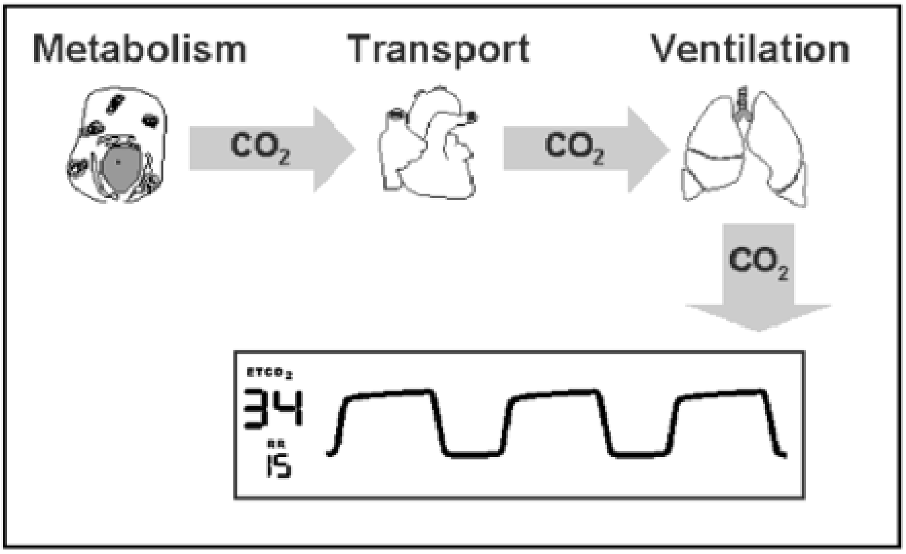
ETCO2 Monitoring Technologies

One way of measuring $ETCO_2$ is with the infrared capnometer, which contains a source of infrared radiation, a chamber containing the gas sample, and a photodetector. When the expired CO_2 passes between the beam of infrared light and photodetector, the absorbence is proportional to the concentration of CO_2 in the gas sample. The gas samples can be analyzed by the mainstream (in-line) or sidestream (diverting) techniques.

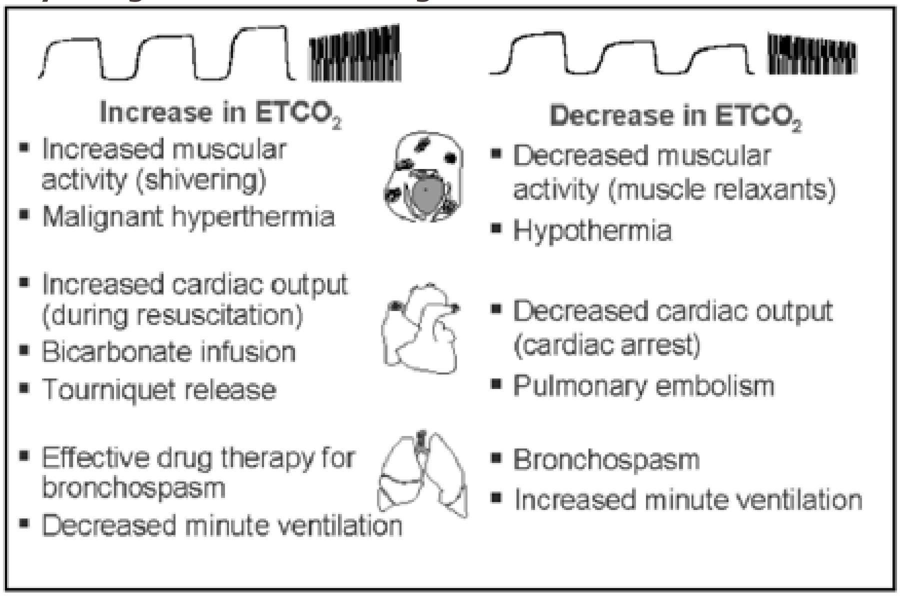
EtCO2 Production Stages



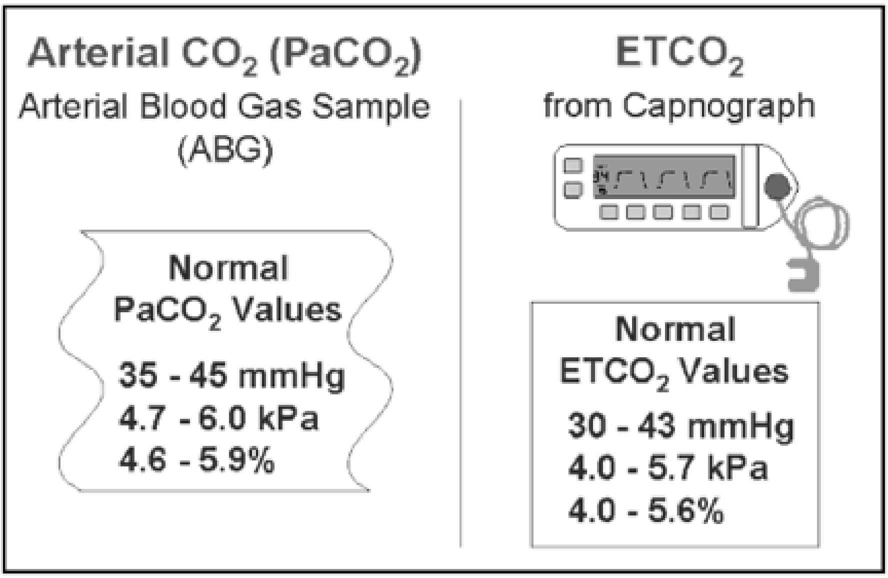
Capnography Depicts Respiration



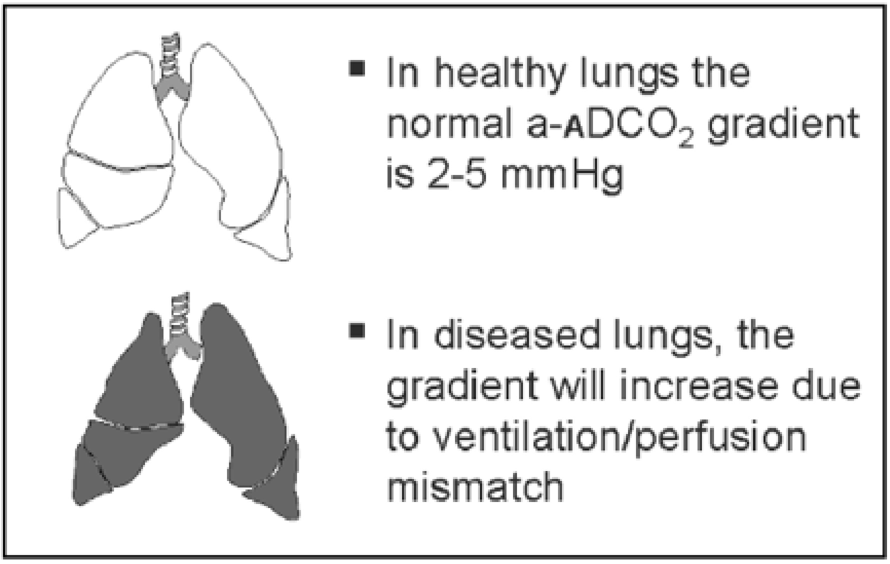
Physiologic Factors Affecting EtCO2 Levels



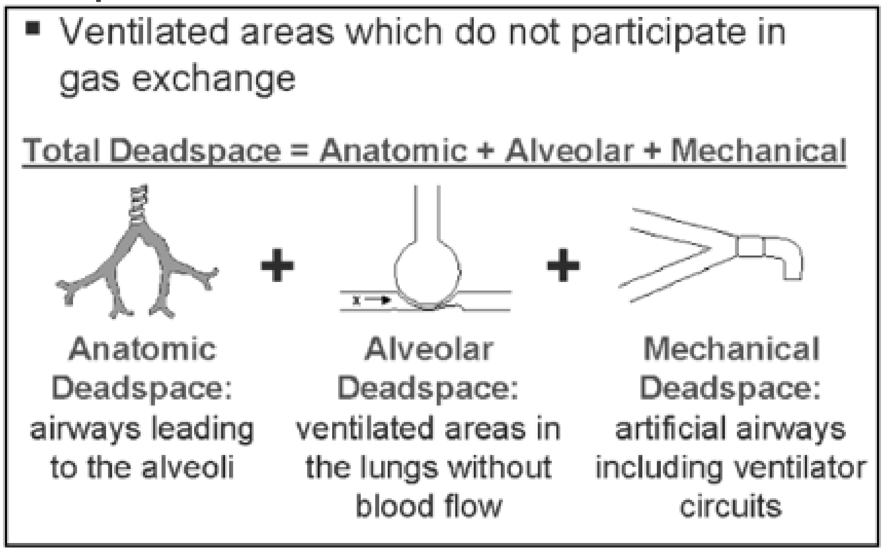
Normal Values



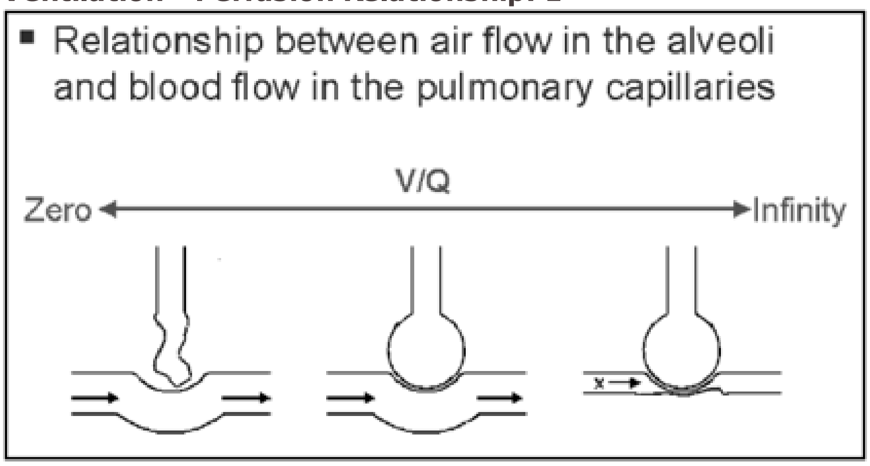
Arterial to End Tidal CO2 Gradient



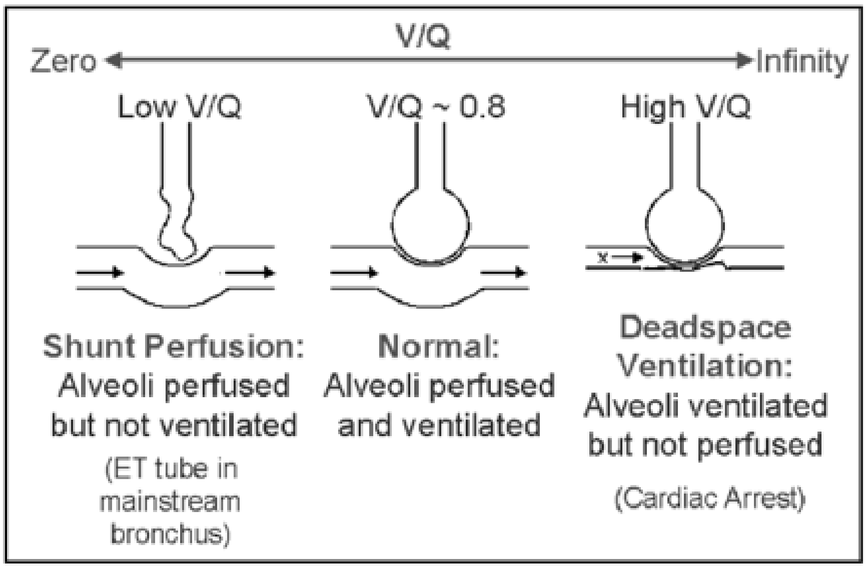
Deadspace



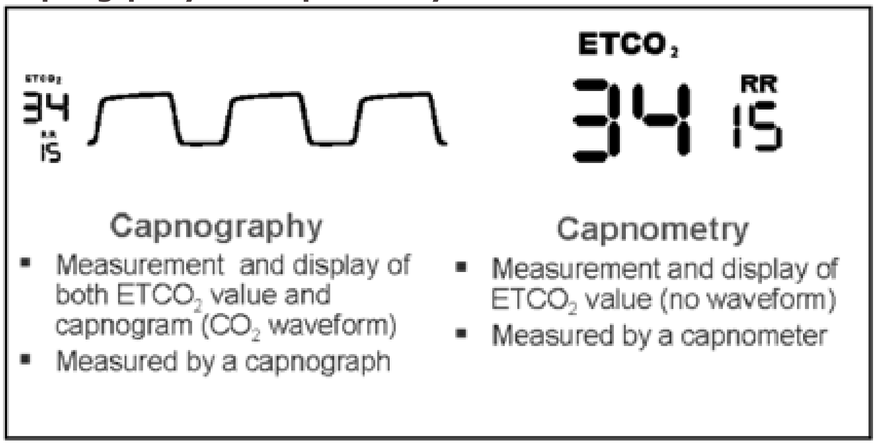
Ventilation - Perfusion Relationship: 1



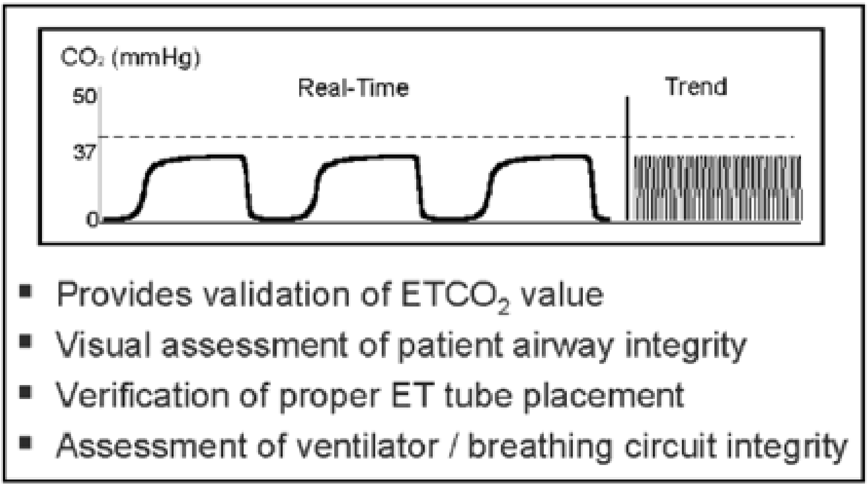
Ventilation - Perfusion Relationship: 2



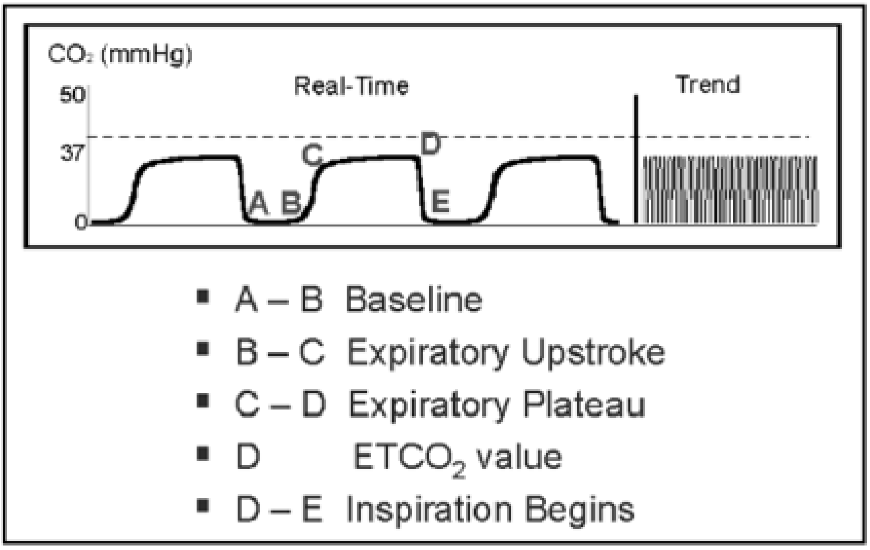
Capnogrpahy vs. Capnometry



The Capnogram

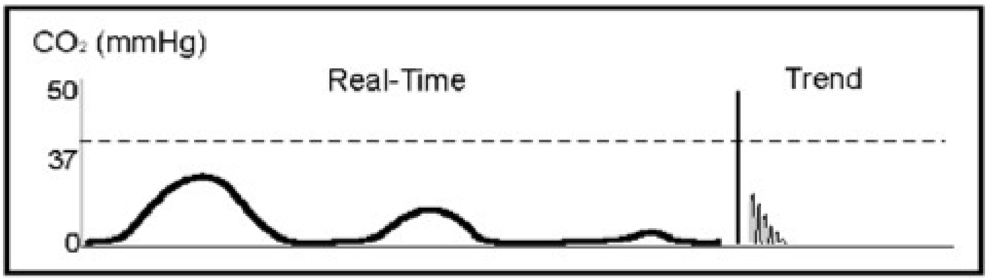


The Normal Capnogram Waveform

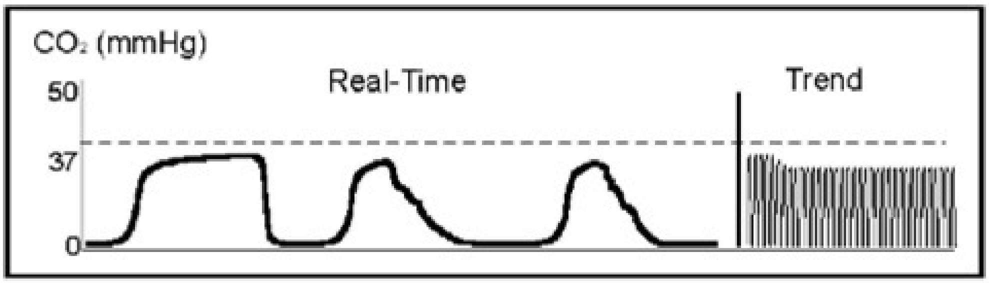


Test Yourself

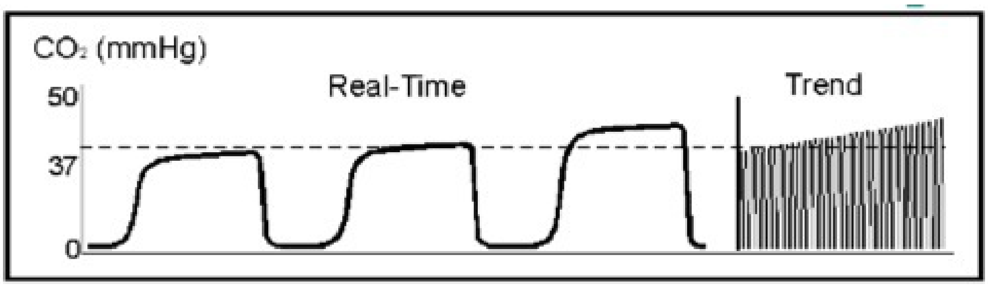
A.



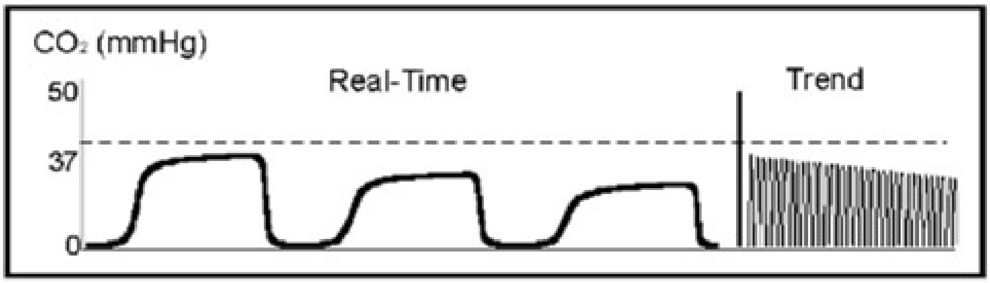
B.



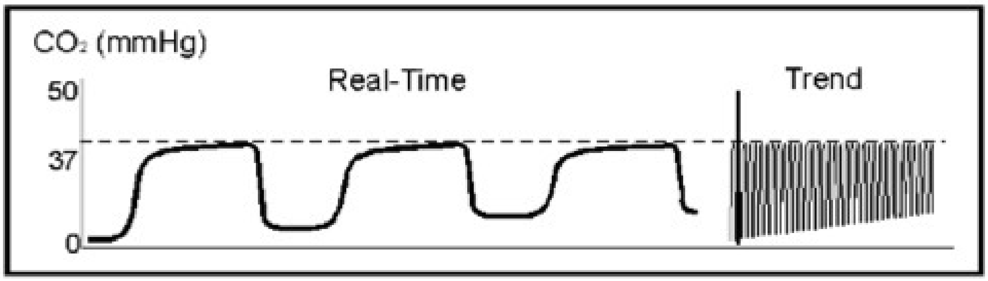
C.



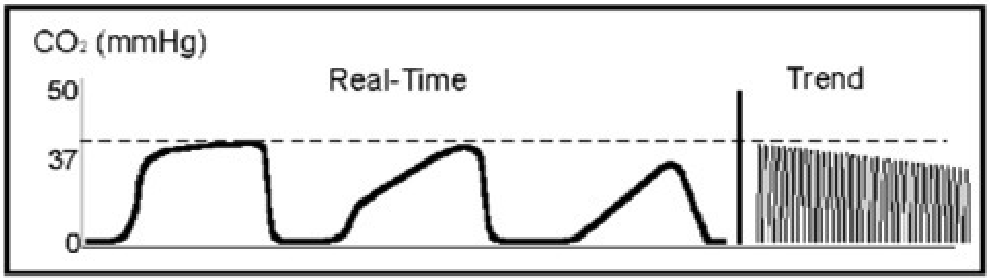
D.



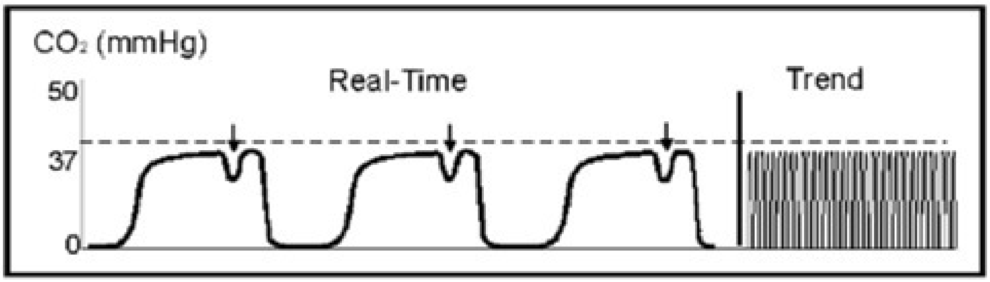
E.



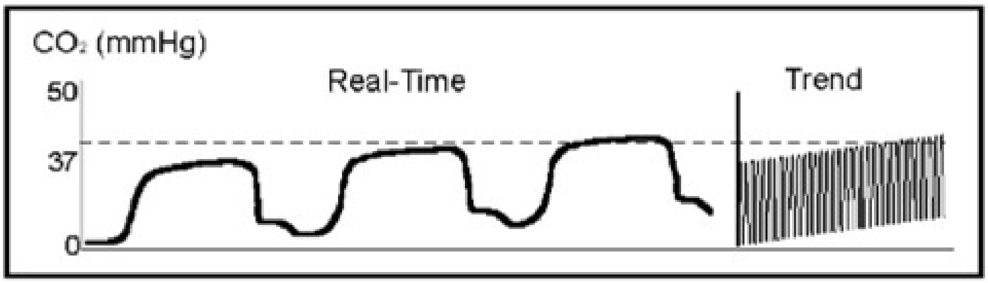
F.



G.



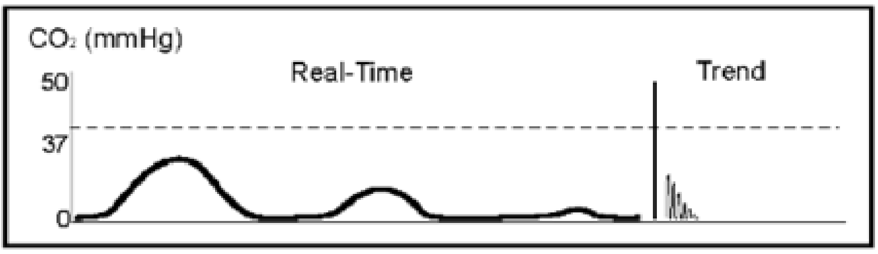
H.



Answers

A.

Endotracheal Tube in Esophagus

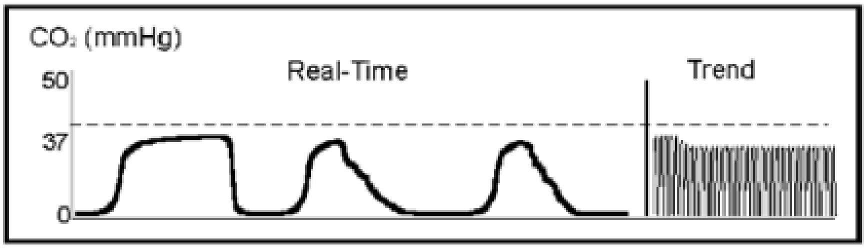


Possible Causes:

- Missed intubation
- A normal capnogram is the best evidence that the ET tube is correctly positioned
- With ET tube in the esophagus, little or no CO_2 is present

B.

Inadequate Seal Around ET Tube

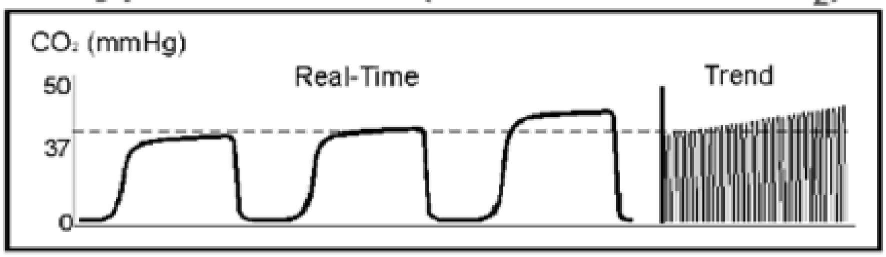


Possible Causes:

- Leaky or deflated endotracheal or tracheostomy cuff
- Artificial airway is too small for the patient

C.

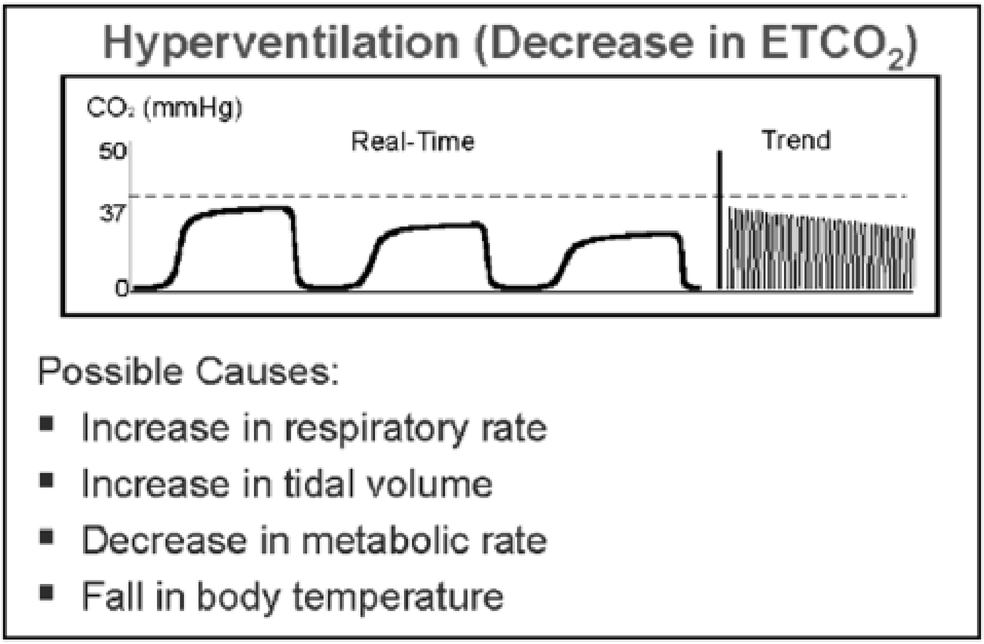
Hypoventilation (Increase in $ETCO_2$)



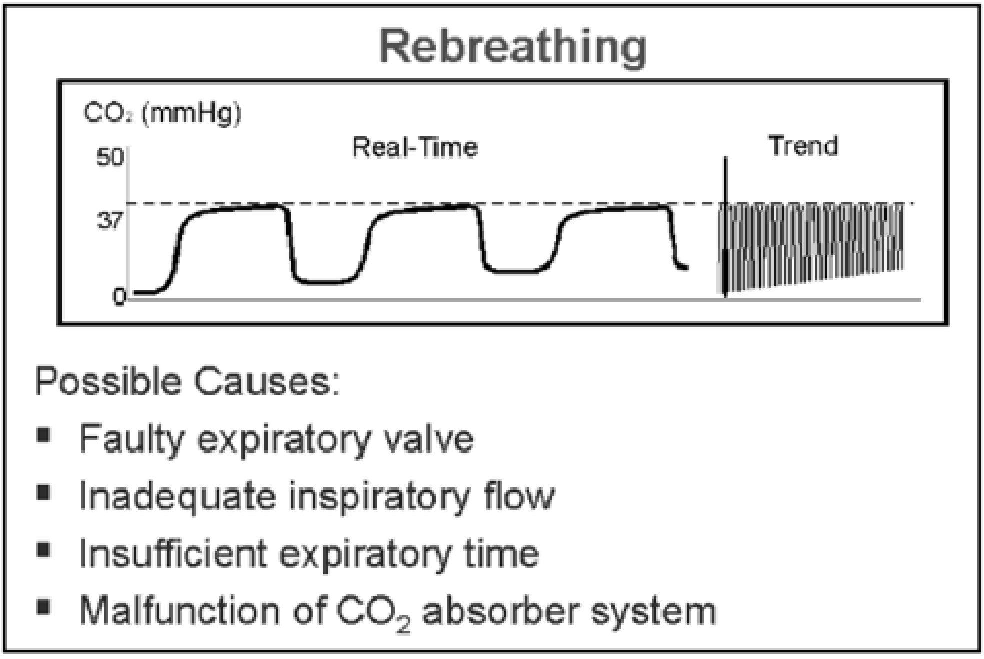
Possible Causes:

- Decrease in respiratory rate
- Decrease in tidal volume
- Increase in metabolic rate
- Rapid rise in body temperature (hyperthermia)

D.


Hyperventilation (Decrease in ETCO2)
CO2 (mmHg)
Real-Time
Trend
50
37
0
Possible Causes:
Increase in respiratory rate
Increase in tidal volume
Decrease in metabolic rate
Fall in body temperature

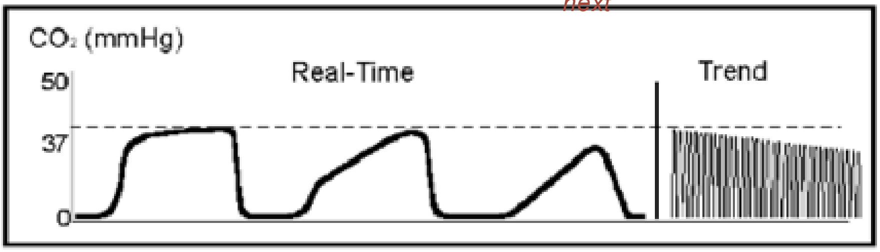
E.


Rebreathing
CO2 (mmHg)
Real-Time
Trend
50
37
0
Possible Causes:
Faulty expiratory valve
Inadequate inspiratory flow
Insufficient expiratory time
Malfunction of CO2 absorber system

F.

Obstruction in Airway or Breathing Circuit

next

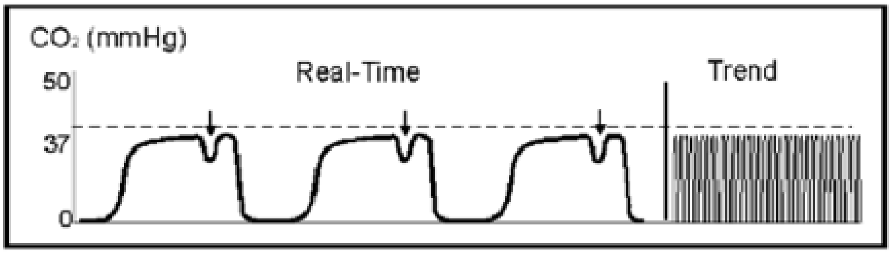


Possible Causes:

- Partially kinked or occluded artificial airway
- Presence of foreign body in the airway
- Obstruction in expiratory limb of breathing circuit
- Bronchospasm

G.

Muscle Relaxants (curare cleft)



- Appear when muscle relaxants begin to subside
- Depth of cleft is inversely proportional to degree of drug activity

H.

Faulty Ventilator Circuit Valve