I. Carbon dioxide analysis—a key area
1.

1. Capnography

<table>
<thead>
<tr>
<th>Written Board Beach Pillbox</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Capnography relies upon infrared absorption and represents an evaluation of the carbon dioxide waveform. Infrared absorption is a function of molecular weight. To review, the MW of He=2, N2O= (7 X 2) + 8= 22, CO2=6 + (8 X 2)= 22, Halothane is 197. Notice the MW of carbon dioxide at 22 is closest to nitrous oxide. Therefore, the amount of N2O would be artificially higher in the presence CO2.</td>
</tr>
<tr>
<td>b. Carbon dioxide contains two dissimilar atoms and absorbs infrared radiation in a spectral range that is convenient to measure.</td>
</tr>
<tr>
<td>c. Capnography is virtually infallible in detecting esophageal intubation and is better than a pulse oximeter (because it is quicker) in detecting esophageal intubation or disconnect. Capnography is not as useful in detecting endobronchial intubation because of the diffusibility of CO2 (see &quot;Ranger Lock and Load Series&quot; below).</td>
</tr>
</tbody>
</table>

THE NORMAL CAPNOGRAPH

![Capnography Diagram]

2. Components of a normal capnograph:
   a. Segment AB represents the beginning of exhalation when tracheal dead space empties of its CO2 free gas.
   b. Segment BC represents the period of continued exhalation when increasing amounts of CO2 rich respiratory gas mixes with dead space gas and results in an increasing CO2 concentration.
   c. Segment CD represents the near-end of exhalation, the so called "alveolar plateau" representing nearly constant CO2 rich alveolar gas.
   d. Point D is the highest value of CO2 concentration at the end of normal exhalation and is the PETCO2.

3. Causes of variation in the normal CO2 waveform.
   a. A Sudden drop to zero indicates a technical defect, such as a disconnect.
   b. A Sudden drop but not to zero indicates leakage or partial obstruction of the airway.
   c. An Exponential decrease should immediately raise suspicion about increased alveolar dead space, such as occurs with pulmonary embolus or cardiac arrest.
   d. A Sudden increase can be caused by the release of a tourniquet.
   e. A Gradual increase may indicate one of several things, including decreased minute ventilation and prolapse of the expiratory valve.

---

II. The carbon dioxide response curve: Crucial for Written Boards. Let's review in depth.

**CO₂ RESPONSE CURVE**

1. Ventilatory control is complex but in general peripheral chemoreceptors such as the aortic and carotid bodies modulate the ventilatory response to arterial hypoxemia and central receptors in the brainstem modulate the response to pH and PaCO₂.

2. The peripheral response:
   a. Carotid and aortic bodies respond primarily to hypoxemia. The carotid bodies are located at the bifurcation of the common carotid arteries and they modulate ventilation. The aortic bodies are located in the aortic arch. Activation of the aortic bodies result primarily in circulatory changes. The afferent limb is the glossopharyngeal nerve and the efferent limb is the vagus nerve. (Remember this!)
   b. The carotid and aortic bodies respond to PaO₂ levels below 60 mm Hg. They do not respond to low oxygen content.

3. The central response:
   a. The chemosensitive areas in the brainstem which control ventilation are located in the medulla, in close proximity to nerves IX (glossopharyngeal) and X (vagus).
   b. Increased PaCO₂ is a very potent stimulus for ventilation. Carbon dioxide, not H⁺, passes through the blood brain barrier and blood-CSF barrier. Once CO₂ crosses into the CSF, H⁺ is formed. The buffering capacity in CSF is small so CSF acidosis occurs.
   c. The ventilatory response to increased CO₂ is increased minute ventilation (both increased rate and tidal volume). It responds rapidly, within only about one minute after the change in PaCO₂.
   d. During apnea, CO₂ rises 6 mmHg the first minute and 3 mmHg for every minute thereafter. This is important and served as the basis for several old Board questions.
   e. Hypoxic ventilatory drive is primarily regulated by the carotid bodies, at the bifurcation of the common carotid artery.
   f. Apneic threshold: This is the highest arterial or alveolar CO₂ at which a subject will remain apneic. The apneic threshold in humans is 5 mmHg below the resting PaCO₂.
4. The carbon dioxide response curve:
   a. The slope of the line is about 0.5-0.7 liters/min/mm Hg. (When PaCO2 reaches 100, the slope is approximately 2 liters/min/mm Hg.)
   b. Above a PaCO2 of 100 mm Hg, carbon dioxide becomes a ventilatory depressant.
   c. Left shifts of the CO2 response curve (increased sensitivity to CO2) result from:
      1) Arterial hypoxemia
      2) Metabolic acidemia
      3) Central causes (Increased ICP, anxiety, fear, cirrhosis)
      4) Drugs: Doxapram, strychnine, picrotoxin (analectics)
   d. Right shifts of the CO2 response curve (decreased sensitivity to CO2) result from:
      1) Aminophylline
      2) Salicylates
      3) Catecholamines
      4) Opioids
      5) Physiologic changes
         a) Metabolic alkalemia
         b) Denervation of peripheral chemoreceptors
         c) Normal sleep
         d) Drugs
         e) Hypothermia
   e. Down and to the right
      1) High doses of opioids
      2) Potent anesthetics: With increasing dose, the curve becomes horizontal with no response to CO2. (The greatest to least ventilatory depression is produced by enflurane, halothane, and isoflurane.)
      3) Neuromuscular blockade
   f. No effect upon the CO2 response: droperidol

Rangers Lock and Load!

From The Joint Council on In-Training Examinations
American Board of Anesthesiology-American Society of Anesthesiologists

"The following responses on the ITE suggested misconceptions in specific areas of the knowledge domain of Anesthesiology...based upon the performance of CA-3 residents taking the examination for ABA credit."

With respect to this 2005 topic, in a memo to residency program directors our ASA president wrote: "As in previous years, the results of some questions on the examination revealed gaps in knowledge that we may find surprising. In the hope that it may assist you in developing your didactic programs, I have listed topics below where fewer than 50% of CA3 residents correctly answered the question. As you know, keywords are included in feedback reports to residents only if 50% or more of the CA3 residents answered the question correctly, so the resident's individual reports will not include these topics. I'm also providing more information than keywords typically reveal. Questions testing these concepts were reviewed by a panel of question content experts, and no flaws in question construction or content were found."

(I regard these "misconceptions" to be important because in a competitive situation questions related to them would stratify and discriminate within a group, thus meeting their goal. Also, critically, since less than 50% of CA-3 residents scored correctly on these topics they are not included in keyword reports. Go over these several times. Be careful! Expect these concepts to be disguised with distracters and land mines all around the target area. Warrior attack!)

According to the Board: More than 50% of CA-3 residents did not know that 0.1 MAC of a volatile anesthetic has a greater effect on the ventilatory response to hypoxemia than to hypercapnia.

Coach's comment: More than 50% miss these topics because most often they are very esoteric and very hard to textbook verify. One can spend more than an hour trying to document and support these statements and cannot clearly do so. Memorize as fact.
III. Physiologic effects of carbon dioxide (memorize): Very important!
1. Hypoventilation-hypercarbia (mnemonic: A RIPE): This physiology is important and has formed the basis for several questions in past years. Memorize!
   a. Acidosis, Arrhythmias
   b. Right shift of the oxy-hemoglobin dissociation curve
   c. Intracerebral steal
   d. PA pressure increase
   e. Epi-norepi release (cutaneous vasodilatation, splanchnic vasoconstriction)

2. Physiologic effects of hyperventilation-hypocarbia (mnemonic: AVCO). Again, memorize!
   a. Apnea, alkalosis, airway constriction
   b. V/Q mismatch
   c. Decreased cardiac output, cerebral blood flow, coronary blood flow, calcium
   d. Oxy-Hb dissociation curve shifted to the left

IV. Carbon dioxide transport:


2. Carbon dioxide is extremely diffusible, more than twenty times greater than oxygen. The importance of this is clear as we "Lock and Load".

---

Rangers Lock and Load!
From The Joint Council on In-Training Examinations
American Board of Anesthesiology-American Society of Anesthesiologists
“The following responses on the ITE suggested misconceptions in specific areas of the knowledge domain of Anesthesiology... based upon the performance of CA-3 residents taking the examination for ABA credit.”

(I regard these “misconceptions” to be important because in a competitive situation questions related to them would serve to stratify and discriminate within a group, thus meeting their goal. Go over these
several times. Be careful! Expect these concepts to be disguised with distracters and landmines all around the target area. Ranger now attack!

According to the Board:
More than 1/3 of CA-3 residents included the detection of endobronchial intubation as one of the uses of capnography intraoperatively.

1. Shunt areas are perfused but not ventilated. The prototype lesion is a pneumothorax. Endobronchial intubation also creates shunting.

2. Consider the impact of these and of shunt upon both CO₂ and O₂ tension.

\[ \begin{array}{c|c}
\% \text{ Shunt} & \text{PaO}_2 & \text{PaCO}_2 \\
\hline
0 & 100 & 40 \\
20 & 80 & 60 \\
40 & 60 & 80 \\
60 & 40 & 100 \\
\end{array} \]

a. The normal shunt is <10%; therefore, over 90% of cardiac output participates in gas exchange.

b. As the shunt fraction increases, PaO₂ progressively falls. This is shown in the above graph. Importantly, PaCO₂ does not increase until Qs/Qt exceeds 50%. (Paul Marino, The ICU Book)

3. Carbon dioxide is highly diffusible, over 20 times more so than oxygen. Endobronchial intubation is not detected by capnography.

A Question From Dr. Jensen's Written Course

K type
End-tidal PCO₂ is a valid measure of PaCO₂ in the presence of
1) Pulmonary emphysema
2) Pulmonary embolism
3) Deliberate hypotension
4) Exhausted carbon dioxide absorber

1. Be careful. You have to have the patience to apply what you know.
2. Pulmonary embolism and severe hypotension represent dead space problems. Pulmonary emphysema can be both a dead space and a shunt problem. In these situations, PaCO₂ may not correlate with PETCO₂.
3. In the case of an exhausted carbon dioxide absorber, there will be no uptake and no excretion so alveolar concentration will equilibrate with arterial and venous concentrations.

Esophageal intubation resulting few abnormal capnograms with relatively normal initial CO₂ numerical values.
Recent keywords of importance—most all of which are covered in Big Blue and/or at the Course:

**Equipment**
1. Carbon dioxide absorption (see physics)
2. Inspired CO2: causes
3. High ETCO2: equipment causes
4. CO2 Absorption in Circle System

**Capnography**
1. DDX: abnormal capnography
2. Capnography and airway obstruction
3. Causes: PaCO2-PETCO2 gradient
4. PaCO2-PETCO2 gradient: etiology
5. Increased ET-arterial CO2 gradient: causes
6. Large art PCO2-ETCO2 grad: causes
7. Cause: low ETCO2
8. Low ETCO2 in cardiac arrest; low ETCO2 (97); PETCO2 during CPR
9. Capnography: applications; Capnography: esophageal intubation
10. Factors affecting end-tidal CO2; factors affecting the capnogram
11. End tidal CO2 and cardiac output

**CO2 response curve**
1. CO2 response curve: drug effects (94); Vent resp to CO2: drug effects
2. CO2 response curve: anesthetic implications

**Hypercarbia**
1. Effects: hypoventilation
2. Effects: intraoperative hypercarbia
3. Apneic oxygenation

**Hypocarbia**
1. Acute hypocarbia and arrhythmias
2. Hypocarbia: physiologic effects; Systemic effects

**Laparoscopy**
1. Responses: CO2 insufflation/laparoscopy
2. Laparoscopy and gas embolism
3. Laparoscopy: EtCO2 and CO2

Other important related keywords from past years are found below. The reference is first, the keyword is underlined.

Capnography

Capnography: applications
Cause: low ETCO2
DDX: abnormal capnography
1. Capnometry and capnography provide important information in assessing the adequacy of ventilation. Capnometry is the measurement of CO2 at the patient’s airway during the ventilatory cycle. A graphic display of the CO2 waveform is capnography.
2. Carbon dioxide analysis usually relies upon infrared spectrometry and takes advantage of the fact that carbon dioxide contains two dissimilar atoms and absorbs infrared radiation in a spectral range that is convenient to measure.

3. Components of a normal waveform. (see drawing in chapter)
   a. Segment AB represents the beginning of exhalation when tracheal dead space empties of its CO2 free gas.
   b. Segment BC represents the period of continued exhalation when increasing amounts of CO2 rich respiratory gas mixes with dead space gas and results in an increasing CO2 concentration.
   c. Segment CD represents the near-end of exhalation, the so called "alveolar plateau" representing nearly constant CO2 rich alveolar gas.
   d. Point D is the highest value of CO2 concentration at the end of normal exhalation and is the PETCO2.

   a. A Sudden drop to zero indicates a technical defect, such as a disconnect.
   b. A Sudden drop but not to zero indicates leakage or partial obstruction of the airway.
   c. An Exponential decrease should immediately raise suspicions about increased alveolar dead space, such as occurs with PE or shock.
   d. A Sudden increase can be caused by the release of a tourniquet or following a sodium bicarbonate injection.
   e. A Gradual increase may indicate one of several things, including decreased minute ventilation and prolapse of the expiratory valve.

CO2 Response Curve

Respiratory
CO2 response curve
Drugs decreasing CO2 response curve
CO2 response curve: Drug effects
CO2 response curve: Anesthetic implications
1. This curve measures minute ventilation (VE in liters/min, y-axis) in response to varying levels of CO2 (PaCO2 torr, x-axis). A picture of this curve can be found in the CO2 chapter and should be reviewed.
   a. The ventilatory response to CO2 is reduced by: sleep; increasing age; genetic, racial, personality factors; in trained athletes and divers; in cases where there is increased work of breathing.
   b. Most of CO2 response is modulated by the medulla (85%). Carotid body chemoreceptors are also important. These are obviously ablated in carotid endarterectomy, decreasing to some extent the ventilatory response to carbon dioxide.

2. The CO2 response curve can be decreased (shifted either down, to the right, or down and to the right) by disease or drugs.
   a. **1 MAC halothane is a respiratory depressant.** The slope returns toward normal after several hours but the curve is right shifted, ventilatory depression continues.
   b. 50% nitrous oxide does not depress the CO2 response curve.
   c. Combined doses of halothane and nitrous oxide depress the CO2 response less than halothane alone.
   d. The respiratory depressant effects of opioids are increased or prolonged with potent anesthetics, alcohol, barbiturates, and benzodiazepines.
   e. Droperidol has no effect on the CO2 response curve.
   f. Benzodiazepines shift the curve down but not to the right

3. Hypoxia: Hypoxia normally shifts the CO2 response curve up and to the left, in other words stimulating ventilation. However, volatile anesthetics (even at subanesthetic concentrations) are known to attenuate this response, too.
   a. Hypoxic ventilatory drive normally becomes strong when the PO2 is reduced to below about 50 torr.

Dead space

**Respiratory**

**Mech:** Increased Intraop A-a CO2  
**Causes:** PACO2-PETCO2 gradient  
**PaCO2-PETCO2 gradient:** Etiology  
**Increased ET-arterial CO2 gradient:** Causes  
**Cause:** Low FTCO2  
**Low FTCO2 in cardiac arrest**

1. Normally the gradient between PaCO2 and PACO2 is less than 3 mm in healthy individuals. Factors which can increase this gradient include:
   a. V/Q mismatching due to the supine position, atelectasis, pulmonary embolism  
   b. Increased dead space ventilation  
   c. Rapid respiratory rates with inadequate time for expiration (there must be a plateau on the capnometry curve for it to be accurate)  
   d. High sampling rate of side stream monitor, in the face of an elevated fresh gas flow rate  
   e. Excessive PEEP, causing overdistension of alveoli and impaired perfusion  
   f. Decreased pulmonary blood flow caused by circulatory arrest, pulmonary embolism, sudden hypotension  
   g. Other: Kinked endotracheal tube, partial disconnect, defective carbon dioxide analyzer

---

**Hypercarbia**


**Effects:** Hypoventilation

**Effects:** Intraoperative hypercarbia

**Physiologic effects of carbon dioxide (memorize)**

1. Hypoventilation-hypercarbia (A RIPE))
   a. Acidosis, Arrhythmias  
   b. Right shift of the oxy-hemoglobin dissociation curve  
   c. Intracerebral steal  
   d. PA pressure increase  
   e. Epi-norepi release  

2. Physiologic effects of hyperventilation-hypocarbia (AVCO)
   a. apnea, alkalosis, airway constriction  
   b. V/Q mismatch  
   c. decreased cardiac output, cerebral blood flow, coronary blood flow, calcium  
   d. oxy-Hb dissociation curve to the left

---

**Laparoscopy**


**Increasing End Tidal CO2 and Laparoscopy**

**Responses:** CO2 insufflation/laparoscopy

Let's review important issues related to laparoscopy.

1. Pneumoperitoneum can be created by either carbon dioxide or nitrous oxide. The **pressure target is about 20 mm Hg.**
   a. Carbon dioxide is usually chosen. Because of its high solubility, it would offer some protection in case of accidental intravascular injection. Associated risks are noted below.
b. Nitrous oxide is associated with less diaphragmatic or peritoneal irritation and, although it is much less soluble, offers the advantage of less shoulder pain. This is especially important in the setting of regional anesthesia. In addition, the risks of arrhythmias are less because hypercarbia from absorption does not occur.

2. There are several important risks:
   a. Gas embolism: signalled by hypotension or hypoxemia
   b. Pneumothorax or pneumomediastinum: secondary to gas travelling retroperitoneally or through congenital defects in the diaphragm
   c. Arrhythmias: secondary to hypercarbia
   d. Reflux: secondary to increased intra-abdominal pressure
   e. Cardiovascular depression: associated with pressures greater than 30 mm Hg. These lead to hypotension, tachycardia, and decreases in CVP, pulse pressure, and cardiac output.

3. The source of an increase in ETCO2 during CO2 laparoscopy may be:
   a. Absorption of CO2 from the pneumoperitoneum
   b. Inadequate spontaneous ventilation secondary to anesthetic agents or pre-meds, as well as mechanical factors, such as pneumoperitoneum, and trendelenburg position.

4. Because of the potential problems with spontaneous ventilation and the many factors favoring retention of carbon dioxide, mechanical ventilation to 1.0-1.5 times the normal requirement is recommended.