

## **Clinical Arterial Blood Gas Analysis<sup>1, 2, 3</sup>**

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### **"Let's Go for a Ranger Run!"**

Some of us would prefer an essay test to the difficult K type format. After reading some of these however, I'll take the K type.

#### Questions For the Final Exam

Prof. Louis Phillips

Humanities 101

Please write all your answers legibly in your blue books. If you want to make notes, additional blue books are available. Do not make notes on this question sheet. You have two hours to complete the exam. Good luck!

1. Compare and contrast the Density of Matter with the Age of Enlightenment.
2. Does a minor chord hibernate? If so, for how long?
3. If Mount Everest could speak to those who have conquered it, what would it say? What language would it speak?
4. How long is a question if it cannot be answered?
5. If human beings were born with seven (7) fingers on each hand, how much of the universe would have to be redesigned?
6. If the number 2 could leave footprints in the sands of time, what shape would those footprints take?
7. List five possible causes for the War Between the States of Mind found in Finnegan's Wake and the Declaration of Independence.
8. The logic of waterfalls. Discuss.
9. Using only a compass and a rule, trisect history.
10. In Shakespeare's The Tempest, Antonio asks, "Who's the next heir of Naples?" The answer, of course, is Claribel. Is the entire history of modern television implied?

1. Acid-base balance is the maintenance within a relatively narrow range of the H<sup>+</sup> concentration in the extracellular fluid. This is both a formidable and a critical physiologic function--formidable because the body must deal with and defend itself against about 15,000 meq of organic acid each day and critical because the H<sup>+</sup> concentration in the extracellular fluid compatible with life covers a relatively narrow five-fold range, from about a pH of 7.0 to about 7.7. This is a broad topic, requiring an understanding of many aspects of physiology and medicine. It is best approached in parts.

- a. The basics are key including a review of ventilation and oxygenation. In terms of ventilation, one must review PaCO<sub>2</sub> and in terms of oxygenation one must review oxygen content, mixed venous oxygenation, and several parameters which are related to lung function and oxygenation: V/Q mismatch, shunt, and the A-a gradient.
- b. Other important areas: basic guidelines and pitfalls in obtaining arterial blood gas samples, essential physics and chemistry of the blood gas measurement, and, finally, an approach to the interpretation of blood gas measurements.

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<sup>1</sup>Smith, H, and Lumb, PD. Acid Base Balance. Clinical Anesthesia, Barash, PG, Cullen, BF, and Stoelting, RK (eds.), Lippincott.

<sup>2</sup>Moon, RE, and Camporesi, EM. Respiratory Monitoring. Anesthesia. Miller, RD (ed.), Churchill Livingstone.

<sup>3</sup>Stoelting, RK, Miller, RD. Acid-Base Balance and Blood Gas Analysis. Basics of Anesthesia, Stoelting, RK, Miller, RD (eds.), Churchill Livingstone.

2. Assessing the adequacy of ventilation is straightforward.
- a. Ventilation is described by PaCO<sub>2</sub>. Recall that PaCO<sub>2</sub> is approximately equal to the production of carbon dioxide divided by CO<sub>2</sub> elimination via the lung, that is,

alveolar ventilation. Since alveolar ventilation (V<sub>CO<sub>2</sub></sub>) is equal to total minute ventilation (VE) minus alveolar dead space (VD) the equation can be written as following:

$$PaCO_2 = \frac{VCO_2}{(VE-VD)}$$

- b. Therefore, the three main determinants of PaCO<sub>2</sub> and of the adequacy of ventilation are carbon dioxide production, minute ventilation, and dead space fraction. Let's consider each of these parameters individually, and review their determinants.

- 1) Looking first at CO<sub>2</sub> production, there are many causes of both high and low CO<sub>2</sub> production.

#### CO<sub>2</sub> PRODUCTION

##### High VCO<sub>2</sub>

Fever  
Thyrototoxicosis  
CNS trauma  
Overfeeding (TPN)

##### Low VCO<sub>2</sub>

Hypothermia  
Hypothyroidism  
Drugs (barbs)

- 2) In terms of minute ventilation, increased minute ventilation can be caused by a number of factors. Decreased minute ventilation can also be caused by a number of factors.

#### MINUTE VENTILATION

##### Decreased MV - High PaCO<sub>2</sub>

Drugs  
CNS disease (CVA)  
Metabolic alkalosis  
Muscle weakness  
Sleep apnea  
Hypothyroidism  
COPD

##### Increased MV - Low PaCO<sub>2</sub>

Anxiety  
Head trauma  
Metabolic acidosis  
Pregnancy  
Asthma  
CHF

- 3) With respect to dead space, recall that there are several types: anatomic, alveolar, physiologic, and apparatus. Dead space is the part of the tidal volume which does not participate in gas exchange. Dead space areas are ventilated but not perfused.

- a) We can visualize this better by considering the zones of the lung, the so-called West zones. Alveolar dead space is represented by zone 1, where there is ventilation but no perfusion.
- b) What are the causes of increased alveolar dead space:

**Pulmonary vascular disease**  
**PE**  
**Vasculitis**  
**COPD**  
**ARDS**  
**Pulmonary fibrosis**  
**Shock**

- c) Anatomic dead space is the volume of air in conducting airways.

d) Physiologic dead space is the sum of anatomic and alveolar dead space. The dead space to tidal volume fraction,  $V_D/V_T$ , is the most common way of quantitating physiologic dead space. It's equal to:

$$\frac{V_D}{V_T} = \frac{PaCO_2 - PeCO_2}{PaCO_2}$$

$$\text{nl } \frac{V_D}{V_T} = 0.3$$

Note that the  $P$  expired  $CO_2$  ( $PeCO_2$ ) is the mixed expired carbon dioxide tension collected from a Douglas Bag, not the  $P$ end tidal  $CO_2$ .

3. There are many important issues related to oxygenation, including oxygen content, oxygen delivery, the oxy-Hb dissociation curve,  $V/Q$  mismatch, shunt, and the A-a gradient.

a. In terms of arterial oxygen content, recall that oxygen is present in the blood in two forms: First, bound to hemoglobin and second dissolved in plasma. Memorize the arterial oxygen content equation:

$$CaO_2 = (1.34 \times Hb \times Sat) + (0.003 \times PaO_2)$$

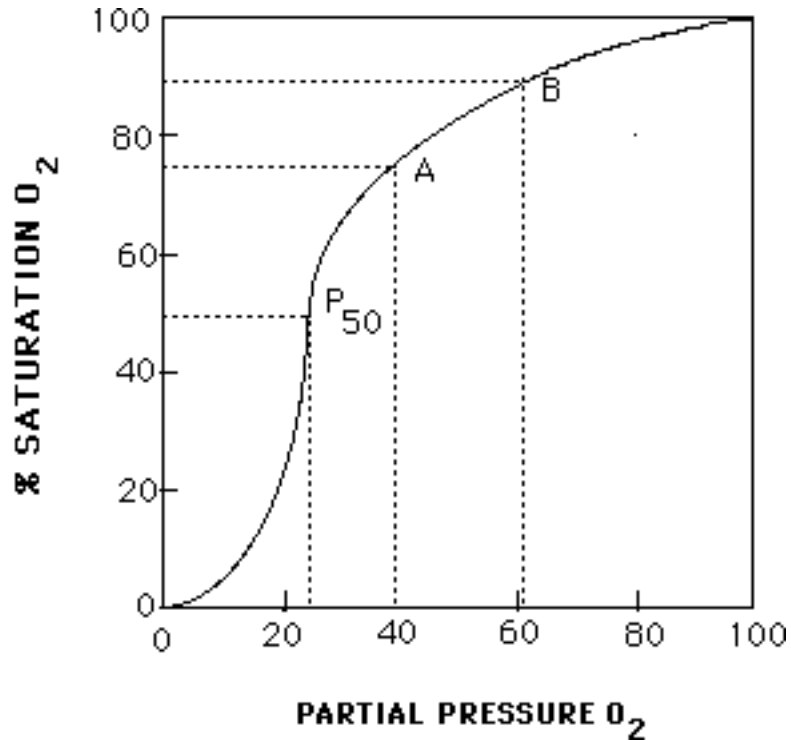
Arterial oxygen content is perhaps the most critical factor in evaluating the adequacy of oxygen delivery to tissues. Looking at this equation it is obvious that by far the greatest amount of oxygen in normal arterial blood is bound to hemoglobin. In fact, only about 1.5% of the total content of oxygen in arterial blood is dissolved in plasma. Clearly then, the most efficient way to increase oxygen content is to increase hemoglobin.

b. Two questions related to oxygen delivery appeared on the 1995 Written examination. Rangers attack! Let's review. Oxygen delivery is defined as the amount of oxygen delivered to the capillaries per minute. It is calculated as the cardiac index multiplied by the content of arterial blood. Remembering this equation will enable you to apply whatever information is given and to score:

$$DO_2 = CI \times CaO_2 \text{ (ml / min / m}^2\text{)}$$

c. We have seen how important it is that there be appropriate quantities of hemoglobin. It is also important how easily the hemoglobin releases its oxygen to the tissues. This is reflected by the oxy-hemoglobin dissociation curve (see below). The  $P_{50}$  is the partial pressure of oxygen at which hemoglobin is 50% saturated. Point A represents normal mixed venous blood and point B a 90% saturation of hemoglobin, which corresponds to a  $PaO_2$  of about 60 mm Hg.

d. Recall that the normal  $P_{50}$  in adults is about 27 mm Hg and in infants about 19 mm Hg, as a result of increased levels of fetal hemoglobin.



- 1) A right shift in this curve results in increased unloading of oxygen at the tissue level and is caused by a number of factors including increased hydrogen ion (acidosis), increased CO<sub>2</sub>, increased temperature, and increased 2,3-DPG.
  - 2) A left shift of the oxy-hemoglobin dissociation curve results in decreased unloading of oxygen at the tissue level and is caused by alkalosis, decreased temperature, and hemoglobin variants such as methemoglobin.
- e. Besides the arterial oxygen content, a second way of assessing the tissue oxygen delivery is by evaluating the mixed venous oxygen level. **This is a crucial area for the Written Boards, with several key words relating to it every year for the past 5 years.**
- 1) Mixed venous oxygen saturation provides one of the most important assessments of tissue oxygen metabolism.
  - 2) Recall that accurate sampling of true mixed venous oxygen saturation requires sampling from PA.
  - 3) (Ranger Lock 'n Load: The normal levels and factors determining PvO<sub>2</sub> are tested virtually every year.) The normal PvO<sub>2</sub> is 35-45 mmHg and the normal saturation is 65-75%. What factors determine PvO<sub>2</sub>? There are several, they can be remembered by the mnemonic COAL, and they include:
    - Cardiac output
    - Oxygen consumption
    - Amount of hemoglobin
    - Loading of hemoglobin (saturation of Hb)

4) PvO<sub>2</sub> and SvO<sub>2</sub> are derived from the following equation--which is a derivation of the Fick equation: (Memorize, to apply in the exam room.)

$$S\bar{v}O_2 = SaO_2 - \left( \frac{\dot{V}O_2}{\dot{Q} \times Hb \times 13} \right)$$

S $\bar{v}$ O<sub>2</sub>: Saturation of mixed venous O<sub>2</sub>

SaO<sub>2</sub>: Saturation of arterial O<sub>2</sub>

$\dot{V}O_2$ : Oxygen consumption

$\dot{Q}$ : Cardiac output

nl SvO<sub>2</sub> = 65 - 75%

f. In evaluating lung function as it relates to oxygenation, one must have an understanding of V/Q mismatch, shunt, and the A-a gradient.

1) Both ventilation and blood flow decrease as one goes up the lung but blood flow decreases more, causing ventilation-perfusion mismatching at the top of the lung. Virtually all PO<sub>2</sub> abnormalities are caused by V/Q abnormalities of some kind.

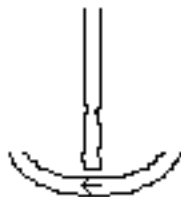
2) The normal V/Q is 1. If there is ventilation but no perfusion, the V/Q ratio is infinity and this is dead space. If the V/Q ratio is zero, perfusion but no ventilation, this is defined as absolute shunt.

### SHUNT- DEADSPACE LESIONS

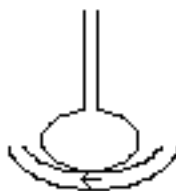
**SHUNT**

**NORMAL**

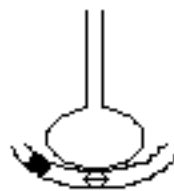
**DEADSPACE**



V/Q = 0  
Perfused But  
Not Ventilated



V/Q = 1



V/Q = ∞  
Ventilated But  
Not Perfused

a) The shunt fraction is calculated by this equation:

$$\frac{Q_s}{Q_T} = \frac{C_cO_2 - C_aO_2}{C_cO_2 - C_vO_2}$$

$$\frac{Q_s}{Q_T} = \text{shunt function}$$

$C_cO_2$  = Content pulmonary capillary blood

$C_aO_2$  = Content arterial blood

$C_vO_2$  = Content mixed venous blood

$$\text{nl } \frac{Q_s}{Q_T} = 0.1$$

The shunt fraction represents one guide as to the efficiency of the lung in facilitating the movement of oxygen molecules from the alveolar space to the capillaries.

3) Another useful parameter of this efficiency is the A-a gradient. Isolated knowledge of the  $PaO_2$  is almost meaningless without knowing the alveolar  $O_2$ . For example, a  $PaO_2$  of 75 to 100 may be normal when the alveolar  $O_2$  is 100 but grossly abnormal if it is 500. Again, the alveolar  $O_2$  is critically important and is defined by this equation:

$$PAO_2 = [(P_B - P_{H_2O}) \times FiO_2] - \left[ \frac{PaCO_2}{0.8} \right]$$

nl A - a  $PO_2$  = 10 - 20 mm Hg

a) The alveolar  $PO_2$  therefore, depends upon the fraction of inspired oxygen and therefore the altitude, as well as other factors such as age.

b) Consider altitude. The inspired  $PO_2$  diminishes as altitude increases and barometric pressure decreases. In Denver, with a barometric pressure in the low 600's the maximum inspired  $PO_2$ , the alveolar  $PO_2$ , is in the low 100's. The highest human habitation occurs at about 20,000 feet where the  $PAO_2$  is in the low 50's. At the summit of Mount Everest, where the altitude is about 29,000 feet and the barometric pressure in the low 200's, the maximum inspired  $PO_2$  is only in the low 40's..

c) Comparing the alveolar  $PO_2$  to the arterial  $PO_2$ , that is calculating the A-a gradient, provides a useful measure of lung function. An approximation is 1/4 the patient's age. Patients under anesthesia have a widened A-a gradient for a number of reasons and this approximation is not completely valid. These reasons include increased V/Q mismatching due to a number of factors, including altered lung and chest wall compliance.

d) Use of the A-a gradient and the shunt fraction can help one delineate the major physiologic causes of hypoxemia: Notice that the first two, alveolar hypoventilation and decreased pressure or fraction inspired  $PO_2$  have normal A-a gradients whereas the last three, ventilation-perfusion abnormalities, diffusion impairments, and right to left intracardiac shunts have elevated A-a gradients.

<u>Cause of Hypoxemia</u>	<u>A-a gradient</u>
<b>Hypoventilation</b>	<b>Normal</b>
<b>Low FiO<sub>2</sub></b>	<b>Normal</b>
<b>V/Q mismatch</b>	<b>increased</b>
<b>Diffusion impairment</b>	<b>increased</b>
<b>Right to Left (intracardiac) shunt</b>	<b>increased</b>

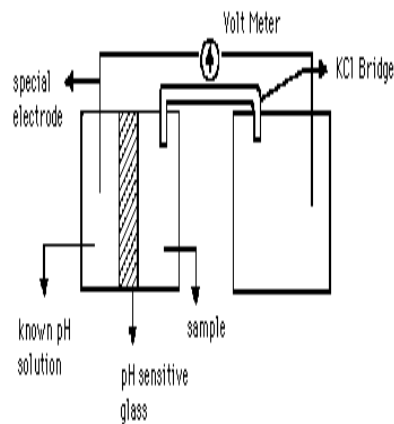
4. With this quick review of ventilation and oxygenation in mind, let's turn to some very practical aspects of blood gas analysis. (Points 4-5 have not been tested in the past few years.)

a. Blood gas samples are highly susceptible to pre-analytic error due to improper methods in obtaining and handling samples.

- 1) Glass syringes should be used if possible because CO<sub>2</sub> and O<sub>2</sub> don't dissolve into the wall of a glass syringe and the minimal friction of glass minimizes the risk of introducing air bubbles into the syringe.
- 2) Heparin is the recommended anticoagulant. EDTA, citrates, and oxalates alter the pH.
- 3) Samples must be obtained under anaerobic conditions because the introduction of air bubbles.
- 4) FiO<sub>2</sub>, Temperature, Source, Ventilator Settings are obviously necessary for interpretation.
- 5) Mixed venous samples are interpretable only when withdrawn slowly from the pulmonary artery, preventing contamination by capillary blood.
- 6) Samples must be stored in an iced slurry because blood is a living tissue and cooling it to 4 degrees centigrade will reduce the metabolism.
- 7) It is important to analyze samples as soon as possible but when properly stored, several hours will result in little change in the blood gas measurements.

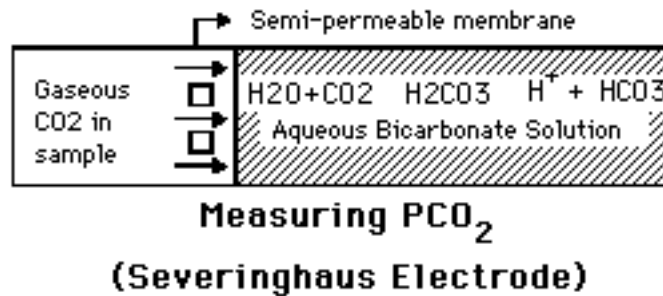
5. The essentials in terms of the physics and chemistry of blood gases can be divided into several key areas:

a. It is not important to dwell on the details at this time other than to know that the pH measurement relies upon the Sanz Electrode:

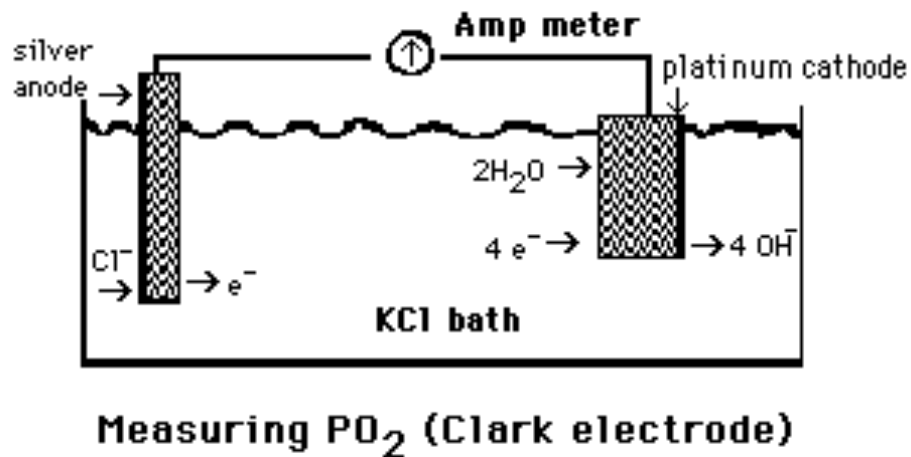


Measuring pH (Sanz electrode)

b. The  $\text{PCO}_2$  measurement relies upon the Severinghouse electrode:



c. The  $\text{PO}_2$  measurement relies upon the Clark electrode:



6. (Back to work!) While normal blood gas values vary according to age, sex, and barometric pressure, we sometimes need to make quick assessments in clinical practice and institute treatments without consulting tables for normal values. In other words, we need to carry certain "normal" values in our heads to effectively deal with clinical problems. Some of these numbers have been adjusted slightly to make them easier to remember.

### Normal values

<b>pH</b>	<b>7.35-7.45</b>
<b><math>\text{PaO}_2</math></b>	<b>75-100 mm Hg</b>
<b><math>\text{HCO}_3</math></b>	<b>20-26 mm Hg</b>
<b><math>\text{PvO}_2</math></b>	<b>35-45 mm Hg</b>
<b><math>\text{SaO}_2</math></b>	<b>95-98%</b>
<b><math>\text{SvO}_2</math></b>	<b>65-75%</b>
<b><math>\text{O}_2\text{con-a}</math></b>	<b>18-22 cc/100cc</b>

7. For the purposes of further discussion, let's now define some important terms.

Acidemia: Blood pH less than 7.35

Alkalemia: Blood pH greater than 7.45

Acidosis: A process which causes acid to accumulate. (This does not necessarily imply an abnormal pH.)

Alkalosis: A process which causes alkali accumulation. (This does not necessarily imply an abnormal pH.)

Buffer: A substance which can absorb or donate H<sup>+</sup>, mitigating changes in pH.

8. There are many buffering systems in the body:

#### MAJOR BUFFER SYSTEMS

**H<sub>2</sub>CO<sub>3</sub>/HCO<sub>3</sub>** (carbonic acid/bicarbonate)

**H<sub>2</sub>PO<sub>4</sub>/HPO<sub>4</sub>** (phosphate)

**HPr/PR-** (serum protein)

**Hemoglobin buffer system**

Of these, the most important buffering system is the carbonic acid-bicarbonate buffering system. The reason is because the concentrations of its components can be independently regulated, PCO<sub>2</sub> by the lungs and bicarbonate by the kidneys.

#### **"Let's Go For a Ranger Run. . ."**

"We shall not cease from exploration. And the end of our exploring will be to arrive where we started and know the place for the first time." (T.S. Eliot)

After years of frustration on standardized tests, I now understand why my performance usually fell short of my expectations. After almost forty years of exploring, I feel that I finally understand for the first time why my work in the standardized exam room always lagged my work in the classroom.

Casual reading and random, aimless study have no place in preparing for a standardized examination. We did well in the classroom by commanding a body of information, anticipating and focusing upon what was expected, and then demonstrating excellence when it was tested.

The same must be done for standardized tests. One must relentlessly focus upon test content.

The focus is here, in these pages. You must now command it. At the tutorial, we will efficiently review this and additional information in an examination format. This process will take us to the heart of the exam and will allow us to achieve the same standards of excellence which have enabled us to travel where we have.

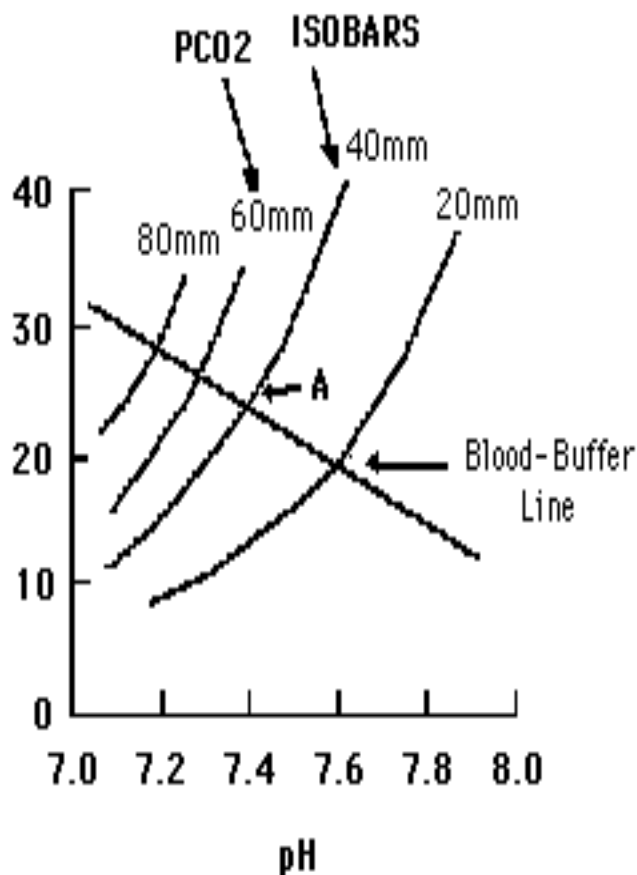
9. What about compensation? The lungs attempt to compensate for pH abnormalities by hypo or hyperventilation. This reflex primarily involves the medulla and the carotid bodies. The kidneys compensate for pH abnormalities in two ways: They vary the reabsorption of filtered bicarbonate and, in certain cases, they add new bicarbonate to plasma flowing through the kidneys.

10. An understanding of the Henderson-Hasselbalch equation is very important in the study of acid-base disturbances:

$$\text{pH} = 6.1 + \log \frac{\text{Base}}{\text{Acid}} = 6.1 + \log \frac{\text{HCO}_3}{(0.03) \text{PaCO}_2}$$

a. Recall that the  $pK$  is the  $pH$  at which the acids in the blood are 50% ionized. Since the  $pK$  is constant (6.1), it is obvious the  $pH$  is determined solely by the ratio of base to acid and not by the absolute concentration of either one. The  $pH$  notation is a useful means of expressing the  $H^+$  concentration in the body because the  $H^+$  concentration happens to be so low relative to other cations. In fact, the concentration of hydrogen ions is about a millionth the concentration of most other ions. The  $pH$ , that is the negative logarithm of this concentration, is about 7.4. The negative log scale helps us to deal efficiently with these small quantities and numbers.

11. The  $pH$ -bicarbonate diagram is the most powerful tool to clarify and simplify the changing relationship between  $pH$ ,  $PCO_2$ , and bicarbonate that occurs in various acid-base disturbances. A mental picture of this diagram enables one to visualize and understand at the bedside complex acid-base problems.



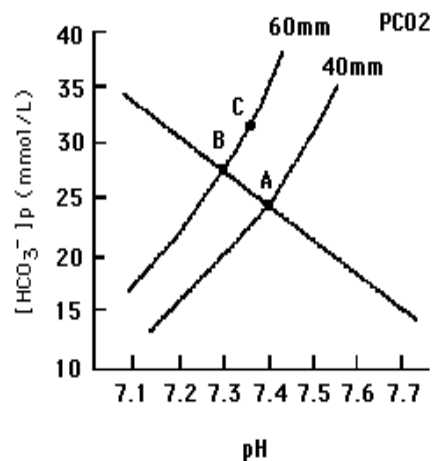
a. On the X axis is  $pH$ . On the Y axis is bicarbonate in mmoles/liter. The blood-buffer line, shown above, illustrates the changes in  $pH$  and bicarbonate of normal blood that occur when  $PCO_2$  is varied. Blood buffer lines must be experimentally determined. The point is that the relationship between  $pH$ ,  $PCO_2$ , and Bicarbonate is a changing one.

b. The  $PCO_2$  isobars, shown above, illustrate the relationship between  $pH$  and bicarbonate at a constant  $PCO_2$ . Since the normal arterial  $PCO_2$  is 40 mmHg and the normal bicarbonate level is 24 mmoles/liter, point A represents the normal arterial point, with a  $pH$  of 7.4.

c. Recall that for every 10 mmHg increase in arterial PCO<sub>2</sub>, the pH decreases by about 0.08- 0.1 unit and for every 10 mmHg decrease in the PCO<sub>2</sub>, the pH increases by 0.08-0.1 unit.

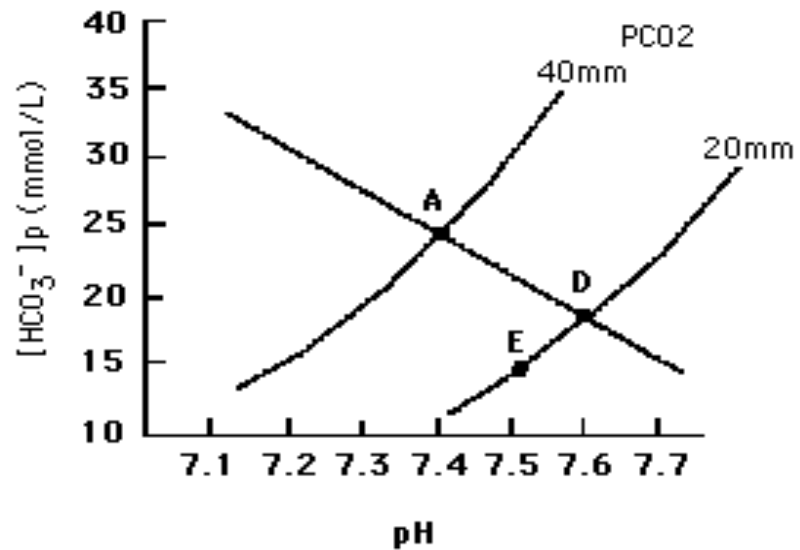
d. Lets look at the four primary acid-base disturbances using the pH-bicarbonate diagram.

1) The primary abnormality in respiratory acidosis is an increase in arterial PCO<sub>2</sub>. Given the inverse relationship between alveolar ventilation and arterial PCO<sub>2</sub>, a major cause of respiratory acidosis is reduced minute ventilation and/or alveolar hypoventilation. Other causes are increased alveolar dead space, increased carbon dioxide production, and increased mechanical dead space. You must be familiar with these "other causes."

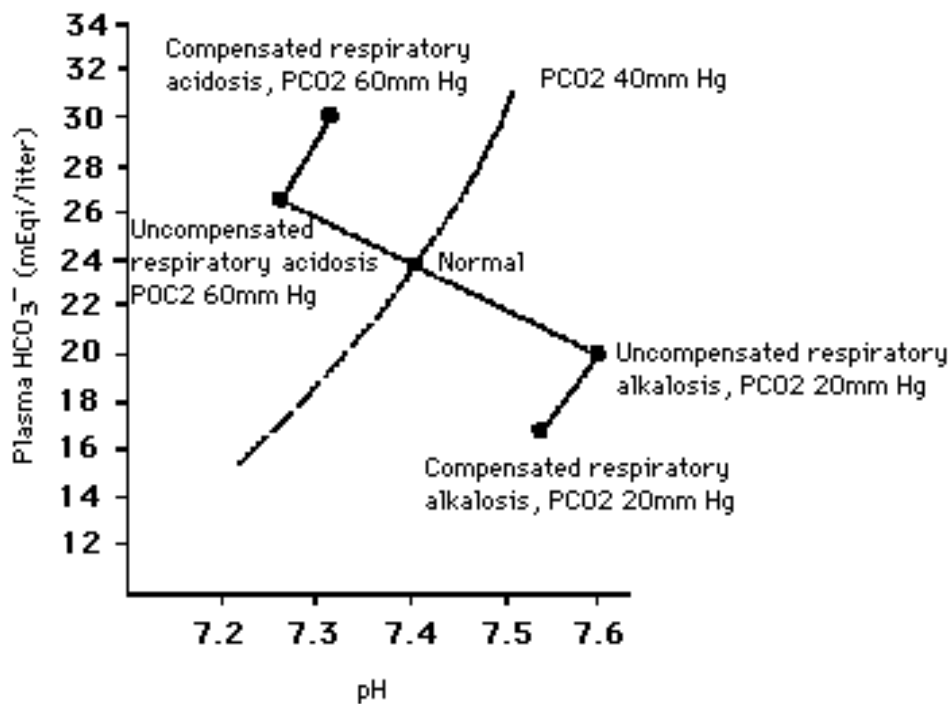


a) Point A represents a normal pH, bicarbonate, and PCO<sub>2</sub>. Point B represents an acute increase in PCO<sub>2</sub> from 40 to 60 mmHg with a corresponding decrease in pH and increase in bicarbonate.  
 b) Point C reveals what happens if the arterial PCO<sub>2</sub> remains elevated, the cause of the respiratory acidosis persists. The kidneys attempt to restore the pH to normal by increasing H<sup>+</sup> secretion and generating additional bicarbonate (over a period of days). Although the arterial PCO<sub>2</sub> remains elevated, the bicarbonate/CO<sub>2</sub> ratio and hence the pH, increases toward normal.

2) In respiratory alkalosis, the primary abnormality is a decrease in arterial PCO<sub>2</sub> and virtually all cases result from hyperventilation, caused for example by hypoxic conditions, CNS disorders, anxiety, and, most often, by too aggressive of ventilatory support.



- a) Notice the fall in arterial PaCO<sub>2</sub> from 40 to 20, at point D.
- b) Once again, the kidneys attempt to restore the pH toward normal by failing to generate any new bicarbonate and failing to resorb all of the filtered bicarbonate. The fall in plasma bicarbonate is seen in point E, chronic respiratory alkalosis.
- c) Notice that these respiratory processes involve moving across PCO<sub>2</sub> isobars and the compensation involves moving up or down along these isobars.



3) In metabolic acidosis, the primary abnormality is a decrease in bicarbonate. There are two types of metabolic acidosis, an increased anion gap and a normal anion gap.

a) What is the anion gap? The sum of all positive charges in the body must be counterbalanced by the sum of all negative charges. However, since all of the anions are not measured, there is an inequality and this

is the anion gap, which is normally  $12 \pm 4$  or between 8 and 16. Anion gap =  $\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$

b) An increased anion gap implies the presence of an unmeasured anion and its conjugate  $\text{H}^+$ , producing a metabolic acidosis. There are seven causes of an increased anion gap acidosis: I find the mnemonic LUK SEMP to be very useful in helping me to remember these:

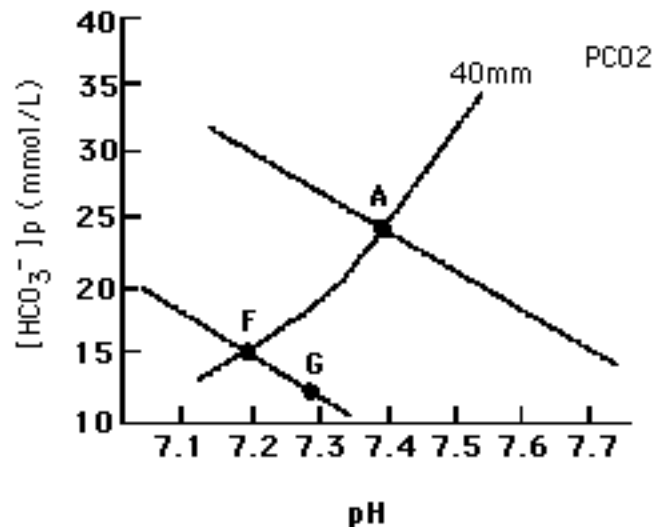
c) Causes of Increased Anion Gap Acidosis (LUK SEMP)

Lactic Acid  
 Uremia  
 Ketoacidosis  
     Diabetic ketosis  
     Alcoholic ketosis  
     Starvation ketosis  
 Salicylates  
 Ethylene Glycol (anti-freeze)  
 Methanol (paint thinner)  
 Paraldehyde (anticonvulsant)

d) Causes of a Normal Anion Gap Acidosis (BADR)

Bicarbonate loss such as GI tract losses  
 Acid loads  
 Dilution of  $\text{HCO}_3^-$  with saline  
 Renal Defects: Poor  $\text{HCO}_3^-$  reabsorption and acid secretion.

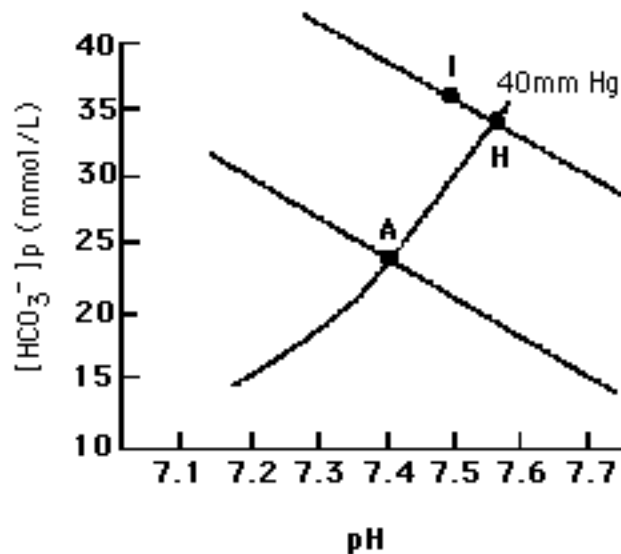
e) Consider what happens when the bicarbonate falls abruptly from its normal value of 24 to 15 mmoles/liter. Note that the bicarbonate decreases along the isobar for a  $\text{PCO}_2$  of 40 mm Hg, since respiratory function remains unchanged. The compensatory response is orchestrated by both the medulla as well as chemoreceptors in the carotid bodies, which initiate reflex alveolar ventilation causing a decrease in arterial  $\text{PCO}_2$  and an increase in the pH toward normal.



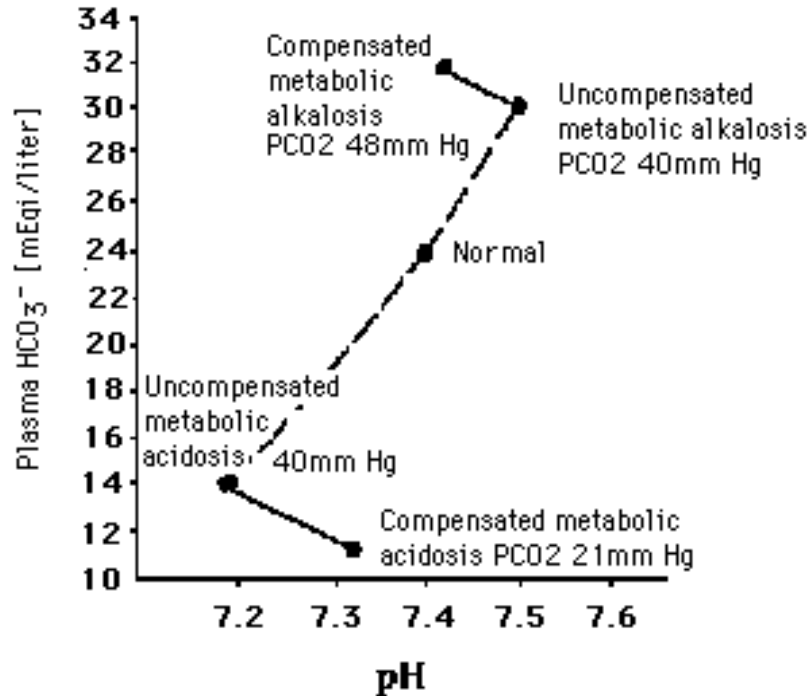
i. How can we determine if the reflex compensatory response of the lungs is appropriate. There is a useful rule: In the case of a metabolic acidosis, the  $\text{PCO}_2$  should be equal to the last two digits of the pH. We will return to this rule, when we evaluate sample cases. This is a coincidental but useful thing to remember.

4) Finally, in a metabolic alkalosis, the primary abnormality is an increase in bicarbonate usually from the loss of bicarb poor fluid. The body store of bicarbonate is therefore contained in a smaller volume and bicarbonate concentration increased. Common causes of metabolic alkalosis include vomiting and nasogastric suction in which  $H^+$  rich gastric fluid is lost and diuretic drugs that result in the excretion of a large volume of acidic urine, such as loop diuretics and thiazides.

a) Consider the acute state as well as the compensation.  
 Consider the acute increase from 24 mmoles/liter to 35 mmoles/liter. The increase occurs along the isobar for  $PCO_2$  of 40 mmHg. The compensatory response to the increase in pH is reflex alveolar hypoventilation, as reflected by point i. The kidneys eventually aid in the compensation by excreting bicarbonate-- however this only occurs after the plasma volume has been corrected by the administration of suitable fluids.



ii. These metabolic processes and their compensation are summarized below. Notice that the primary processes involve alterations in bicarbonate along one  $PCO_2$  isobar and the compensatory processes involve movement across  $PCO_2$  isobars.



12. In a general way, the treatments for these problems are obvious from the causes but we need to consider sodium bicarbonate in the treatment of acute lactic acidosis, a controversial area.

- a. The development of a lactic acidosis during the course of an illness signals a dangerous disruption of normal oxygen use. If not reversed, there is progressive intracellular and extracellular acidosis and eventually death. In the past, an acute lactic acidosis was treated with sodium bicarbonate. In vitro data generally demonstrate that either respiratory or metabolic acidosis below a pH of about 7.1 decreases myocardial contractility and causes CNS depression.
- b. The role of sodium bicarbonate has recently been challenged with several studies showing it to be ineffective and potentially detrimental by actually promoting lactic acid production. The American Heart Association now recommends that sodium bicarbonate be used only late in the course of the cardiopulmonary resuscitation, if at all.
- c. The major problem is that when exogenous bicarbonate is administered during acidemia, bicarbonate reacts with hydrogen ions to form carbonic acid. The carbonic acid dissociates to CO<sub>2</sub> and water and the CO<sub>2</sub> partial pressure increases. When CO<sub>2</sub> cannot be eliminated, the pH of the system is only minimally changed or in fact worsened.
- d. Therefore, during states of severe hypoperfusion, such as CPR, there is very little benefit from sodium bicarbonate administration.
- e. In other settings, sodium bicarbonate does have a role in the treatment of lactic acidosis because of the severe hemodynamic deterioration that can occur with a pH much below 7.1. Small titrated doses of bicarbonate should be used to temporize while attempts to improve tissue oxygenation continue.

1) In such cases, generally treat if the pH is less than 7.20, as long as a respiratory acidosis does not exist. If it does, treat the respiratory acidosis first. The dose of bicarbonate can be determined by the following :

Body weight in kg. X deviation of HCO<sub>3</sub> from 24 X 0.2

0.2 = Extracellular fluid volume as a fraction of body wt.

0.4 = Extracellular fluid volume as a fraction of body weight in infants.

2) Besides the problems mentioned, other problems with bicarbonate include:

Intraventricular hemorrhage  
Hypernatremia  
Hyperosmolarity  
Left shift of the oxy-Hb dissociation curve due to rebound alkalosis

13. Temperature correction of ABGs:

**1998 Written Board Beach Pillbox: Lock 'n Load--Relevant Questions at Course**

- a. The solubility of oxygen and carbon dioxide are temperature dependent. At lower temperature, the solubility of oxygen and carbon dioxide in solution is higher, there are less molecules in the gas phase, and the partial pressure of both is decreased. With the partial pressure of carbon dioxide less, pH is higher at lower temperature.
- b. When a blood gas sample arrives in the laboratory for analysis, it is warmed to 37° C. and analysis is undertaken. It is not "temperature corrected" as the blood gas analyzer assumes a patient temperature of 37° C. If patient temperature is significantly less than 37° C. and if desired a second "temperature corrected" reading can be performed. This reading utilizes a computer normogram which corrects for solubility and pH changes which occur with temperature.
- c. The terms alpha stat and pH stat describe strategies for managing the results of arterial blood gas results. The alpha stat strategy relies upon the uncorrected arterial blood gas values; no attempt is made to correct partial pressures of oxygen and carbon dioxide for differences in temperature. The pH stat strategy relies upon the temperature corrected values and involves administering carbon dioxide gas systemically to the patient to correct for lower partial carbon dioxide pressure secondary to its increased solubility in solution. It aims to keep pH at about 7.40 and PaCO<sub>2</sub> at about 40 mm Hg.
- d. Alpha stat versus pH stat arguments are important for Oral Boards and will be reviewed in Big Red. Our present purposes dictate only a firm command of points a-c.

14. There is considerable confusion when it comes to defining acid-base abnormalities as primary, secondary, or compensatory processes. If both respiratory and metabolic problems are an acidosis or if both are an alkalosis, then both processes are primary. If they are opposite, then the primary problem is defined by the pH. (see ABA-ASA Lock 'n Load Notes and Tape in Ranger Blue.)

15. Besides being able to read a blood gas systematically, it is critical that you know how to use the basic information which blood gases convey at the bedside in taking care of patients. We must be able to apply the information that we gain from an arterial blood gas: to evaluate the status of the acid-base balance, the status of oxygenation and ventilation, the need for emergent intubation, and the appropriateness of extubation, to name a few.

a. Let's review the indications for **intubation**, some of the most important elements embodied in the results of an arterial blood gas. There are three factors to consider: mechanics, oxygenation and ventilation. In terms of . . . . .

1) Mechanics

- a) Respiratory rate greater than 35/min.
- b) VC less than 15 cc/kg for adults and 10 cc/kg for children
- c) MIF less than 20 cm H<sub>2</sub>O

2) Oxygenation

- a) PaO<sub>2</sub> less than 70 mmHg on FiO<sub>2</sub> 40%
- b) The A-a gradient is greater than 350 torr with FiO<sub>2</sub> 100%

3) Ventilation

- a)  $\text{PaCO}_2$  is greater than 55 except in patients with chronic hypercarbia.
- b)  $\text{Vd/Vt}$  is greater than 0.6 (normal is 0.3)

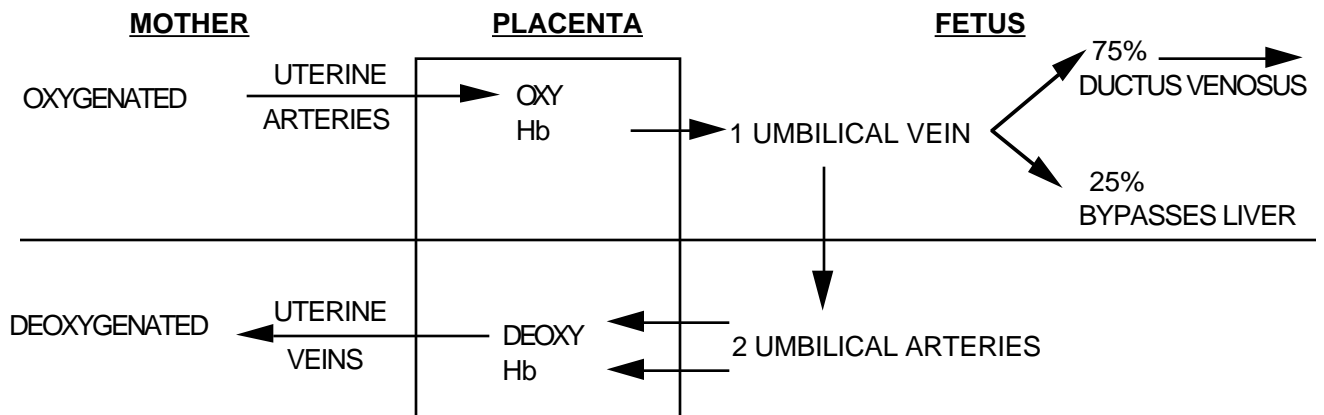
b. Decisions about extubation frequently are also aided by the results of a blood gas. Criteria for extubation:

- 1) Awake and alert with stable vital signs, good grip, and sustained head lift.
  - a) Stable vital signs includes respiratory rate less than 30-35
  - b) Stable blood pressure and pulse
  - c) No inotropic support
  - d) Patient afebrile
- 2) ABG good on 40%, with  $\text{PaO}_2$  greater than 70 and  $\text{PaCO}_2$  less than 55
- 3) MIF more negative than a negative 20 cm H<sub>2</sub>O.
- 4) VC greater than 15 cc/kg.

c. Weaning from mechanical ventilation is accomplished in a number of ways, blood gases being one important parameter of many of them. There are two basic techniques, the T-piece technique and the intermittent mandatory ventilation technique. You should review these at this time.

- 1) T-piece technique
  - a) If patient meets extubation criteria a T-piece adaptor and is attached to the endotracheal tube.
  - b) Sit patient up.
  - c) Set  $\text{FiO}_2$  slightly higher (about 5%) than the patient had with mechanical ventilation
  - d) Check vital signs (including respiratory rate, depth, and work) and saturation. This should be done on a very frequent basis during the first hour or two.
  - e) If the patient tolerates weaning, he is extubated after 2-4 hours.
- 2) Intermittent mandatory ventilation technique
  - a) The IMV is gradually decreased so that eventually the patient begins spontaneous ventilation.

16. In reviewing blood gas questions from previous exams, it is clear that a memorization of umbilical vein and artery gases is important. They expect you to know the normal values, so that you can interpret the gas and thereby the needs of the patient.



a. Normals

	UMBILICAL VEIN-BIRTH	UMBILICAL ARTERY-BIRTH	60 MIN
pH	7.35 (7.30-7.40)	7.28 (7.23-7.33)	7.30-7.35
pCO <sub>2</sub>	40 50 (33-43)	32-42 (42-58)	
pO <sub>2</sub>	30 20 (25-35)	50-70 (12-25)	

b. As one would expect, umbilical artery blood (which has circulated through the fetus) is lower in pH and oxygen and higher in carbon dioxide than umbilical venous blood.

17. Notes on acid-base-pKa-ionization-lipophilic issues. [This is important information for the Written examination. I've never seen it put together in quite this way. It may be worth 2-3 questions on the exam.]

a. Neutral pH 7.0

b. pKa: The pH at which 50% ionization occurs.

c. Two important questions:

1) What is the ionizing group?

2) It is charged with the proton on or off?

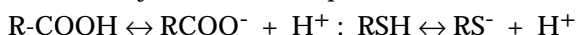
d. There are two common ionizing groups in biologic systems:

1) Amines (narcotics, local anesthetics):



The amines are the main ionizing group in most drugs. They are charged in the protonated form. Therefore, as the system becomes more acidic, they are more charged and less lipophilic. As the system becomes more basic, they are less charged and more lipophilic. This is how the term "free basing" comes about. Cocaine is an amine. When sodium hydroxide or sodium bicarbonate is added to make the solution more alkaline, the free base forms, which is uncharged, less water soluble (being ionized leads to greater water solubility), very lipid soluble, and rapidly absorbed through mucous membranes.

2) Carboxylic acids (Thiopental):



The carboxylic acids are the second most common ionizing group in biologic systems. They are uncharged in the protonated form. Therefore, as the physiologic system becomes acidic, they are less charged and more lipophilic. As the system becomes more basic, they are more charged and less lipophilic.

3) Thiols are another type of ionizing group. Consider them in the same way as carboxylic acids, except they have higher pKa. The major ionizing group on thiopental are thiols.

e. Recall this information:

	pH	pKa
Thiopental:	10.5	7.6
Narcotics (morphine):	2.5-6.0	6.1
Local anesthetics :	5.0-7.0	8.0-9.0

f. In assessing lipophilicity, one must therefore know the major ionizing group as well as the the pKa. If the pKa is high enough it moves it out of the range where pH

adjustments are relevant at all. If the pKa is 11 then at any physiologically reasonable pH the material will be almost exclusively in the protonated form. g. Local anesthetics and narcotics are subject to fetal ion trapping. In the more acidic fetal environment, the dissociation is to the left--the charged form which has difficulty crossing the placental barrier (and thus "traps" the compound). What about thiopental? It is not trapped. In the more acidic fetal environment, dissociation is the left--to the uncharged form which can more easily cross the placental barrier.

#### **A Question From Dr. Jensen's Tutorial**

K type

Which of the following would indicate the need for a mechanical ventilator?

- 1)  $V_d/V_t$  of 0.8
- 2) A-aDO<sub>2</sub> of 150 torr at FIO<sub>2</sub> 1.0
- 3) Inspiratory force of 5 cm H<sub>2</sub>O
- 4) Vital capacity of 20 ml/kg

B.

Know the list, indications for intubation. Two points:

1. They really do expect you to know this stuff!
2. Big Blue will serve you well if you serve it well.

#### **NFJs Key Quotes**

Life is what happens to you while you're making other plans  
--John Lennon