

ARTERIAL BLOOD GAS ANALYSIS



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INTRODUCTION

- Arterial blood gas analysis refers to the measurement of pH and the partial pressure of oxygen and CO₂ in arterial blood.
- It is an essential part of diagnosing and managing a patient's oxygenation status and acid–base balance.



DEFINITIONS & TERMINOLOGY

- **Acidemia** - decrease in the blood pH below normal range of 7.35 - 7.45
- **Alkalemia** - Elevation in blood pH above the normal range of 7.35 – 7.45)
- **Acidosis** – process that increases $[H^+]$ by increasing PCO_2 or by reducing $[HCO_3^-]$
- **Alkalosis** – process that reduces $[H^+]$ by reducing PCO_2 or by increasing $[HCO_3^-]$



DEFINITIONS AND TERMINOLOGY

- *An **acid base disorder** is a change in the normal value of extracellular pH that may result when renal or respiratory function is abnormal or when an acid or base load overwhelms excretory capacity.*

Normal acid base values

	pH	pCO ₂	HCO ₃ ⁻
Range	7.35-7.45	36-44	22-26
Optimal Value	7.4	40	24



○ **BUFFER**

- A buffer is a solution containing substances that have the ability to minimize changes in pH when acid or base is added to it .It consists of a weak acid mixed with salt of that acid and a strong base.



○ *There are three major buffer systems in the body*

- In the extracellular fluid (ECF)
- In the intracellular fluid(ICF)
- Bone.



EXTRACELLULAR BUFFERS

- The most important buffer in the ECF and the body is **HCO₃⁻** (bicarbonate) which combines with excess H⁺ ions to form carbonic acid.
- The **CO₂/HCO₃⁻** buffer system is considered very effective because of the vast quantity of bicarbonate in the body and the ability to excrete the CO₂ formed via ventilation.
- Other less important buffers in the ECF are **plasma proteins and inorganic phosphates**.



INTRACELLULAR BUFFERS

- The primary intracellular buffers are *proteins, organic and inorganic phosphates* and in the RBC, *hemoglobin (HB-)*
- Whereas buffering by plasma HCO_3^- occur almost immediately, approximately **2-4 hours** is required for buffering by cell buffers due to slow cell entry.



BONE

- Bone represents an important site of buffering acid load.
- An acid load, is associated with the uptake of excess H^+ ions by bone which occurs in exchange for surface Na^+ and K^+ and by the dissolution of bone mineral, resulting in the release of buffer compounds, such as $NaHCO_3$, $CaHCO_3$, and $CaHPO_4$.





It has been estimated that at least 40% of the buffering of an acute acid load takes place in bone. Chronic acidosis can have very adverse effects on bone mineralization due to this process and can result in bone diseases such as rickets, osteomalacia and osteopenia.



- **BASE EXCESS[BE]** -BE is index of magnitude of the metabolic contribution to an acid base disturbance
- **STANDARD BICARBONATE**-It is the calculated bicarbonate value that would exist if the patient's P_{aCO_2} were 40 mmhg and the Hb were 100% saturated with O_2



OVERVIEW OF ACID-BASE PHYSIOLOGY

- **Daily Acid Load**-There are 2 types of acids that can potentially contribute to the daily acid load;

Nonvolatile Acids	Volatile Acids (H_2CO_3)
Metabolism of amino acids, producing HCL and H_2SO_4	Metabolism of fats and carbohydrates producing CO_2
Intake of acid containing foods-sulphates, phosphates	Production of 15-20 mol of CO_2 per day
Daily loss of alkali in feces (minimal unless diarrhea)	
Generation of 50 -100 meq of H^+ per day	



Range of ECF [H⁺] variation very small

pH Vs. [H⁺]

pH		nanoeq [H ⁺]/L
7.00-7.38	Acidemia	100-44
7.38-7.44	Normal	44-36
7.44-7.80	Alkalemia	36-16

Relationship between pH and [H] at physiologic pH

pH	7.00	7.10	7.20	7.30	7.40	7.50	7.60	7.70
[H ⁺] (nM)	100	79	63	50	40	32	25	20



IMPORTANCE OF pH CONTROL

- pH (intracellular and ECF including blood) maintained in narrow range to preserve N cell, tissue and organ f_x

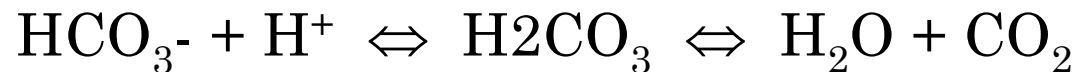
Intracellular pH (pH_i)

- Maintained at ~ 7.2 :
 1. To keep imp metabolic intermediates in ionized state and limit tendency to move out of cell
 2. Most intracellular enzymes taking part in cellular metabolism have pH optimum close to this value
 3. DNA, RNA & Protein synthesis \uparrow at slightly higher pH



Regulation of arterial pH

1. **BUFFERS** – presence of buffer systems minimize the change in pH resulting from production of acid and provide imm protection from acid load. Main buffer system in humans is HCO_3^-



2. **ROLE OF THE RESPIRATORY SYSTEM** – is in elimination of volatile acid -- CO_2 .
 - a. Respiratory centers in the brain respond to changes in pH of CSF and blood to affect ventilatory rate.
 - b. Ventilation directly controls the elimination of CO_2 .



3. **ROLE OF THE KIDNEY** - To retain and regenerate HCO_3^- thereby regenerating the body buffer with the net effect of eliminating the non-volatile acid load

a. **Excrete the daily acid load**

1. **Hydrogen secretion**

2. Excretion of hydrogen ions with **urinary buffers**(**titratable acids and ammonium**)

b. **HCO_3^- reabsorption**

1. Proximal tubule – 90%

2. Distal tubule

Factors affecting H^+ secretion/reabsorption HCO_3^-

a. CO_2 concentration, pH

b. Aldosterone

c. ECF volume

d. Potassium concentration

e. Chloride



- In order to approach acid base disorders, consider the following equations:

1) *Henderson Hasselbalch equation:*

- $$\text{pH} = 6.1 + \log \frac{\text{HCO}_3^-}{0.03 \text{ pCO}_2}$$

2) *Kassirer-Bleich equation:*

- $$\text{H}^+ = 24 \times \frac{\text{pCO}_2}{\text{HCO}_3^-}$$



3 Component Terminology

- I. Respiratory/Metabolic
- II. Compensated/decompensated
- III. Acidosis/Alkalosis



RESPIRATORY VS METABOLIC

- **Respiratory** – processes which lead to **acidosis or alkalosis** through a primary alteration in ventilation and resultant excessive elimination or retention of CO₂
- **Metabolic** – processes which lead to **acidosis or alkalosis** through their effects on kidneys and the consequent disruption of H⁺ and HCO₃⁻ control



COMPENSATORY RESPONSES

- ❑ Acid Base disorders are associated with defense mechanisms referred to as compensatory responses that function to reduce the effects of the particular disorder on the pH.
- ❑ **They do not restore the pH back to a normal value**
- ❑ This can only be done with correction of the underlying cause



COMPENSATION

- Disturbances in HCO_3^- (metabolic acidosis or alkalosis) result in respiratory compensation while changes in CO_2 (respiratory acidosis/alkalosis) are counteracted by renal compensation
- a. **Renal compensation** – kidneys adapt to alterations in pH by changing the amount of HCO_3^- generated/excreted. Full renal compensation takes 2-5 days
- b. **Respiratory compensation** – alteration in ventilation allow immediate compensation for metabolic acid-base disorders . It reaches its maximum in 24 hrs

SIMPLE VS. MIXED ACID-BASE DISORDER

- Simple acid-base disorder – a single primary process of acidosis or alkalosis
- Mixed acid-base disorder – presence of more than one acid base disorder simultaneously



Characteristics of 1° acid-base disorders

DISORDER	PRIMARY RESPONSES			COMPENSATORY RESPONSE
Metabolic acidosis	↑ [H ⁺]	↓ PH	↓ HCO ₃ ⁻	↓ pCO ₂
Metabolic alkalosis	↓ [H ⁺]	↑ PH	↑ HCO ₃ ⁻	↑ pCO ₂
Respiratory acidosis	↑ [H ⁺]	↓ PH	↑ pCO ₂	↑ HCO ₃ ⁻
Respiratory alkalosis	↓ [H ⁺]	↑ PH	↓ pCO ₂	↓ HCO ₃ ⁻



INDICATIONS

1. *ESTABLISHING DIAGNOSIS AND ASSESSING ILLNESS SEVERITY AS IN*
 - a. Suspected hypercapnia
 - i.e. in a drowsy patient with flapping tremors, bounding pulses or clinical deterioration in patient with chronic type II respiratory failure e.g. COPD
 - b. Suspected severe hypoxaemia
 - i.e. patient with very low or unrecordable oxygen saturation, cyanosis
 - c. Severe prolonged or worsening respiratory distress
 - d. Acute deterioration in consciousness
 - e. Hyperventilating patient to confirm decreased PaCO₂ and check for underlying metabolic acidosis



f. Any severely unwell patient

2. GUIDING TREATMENT AND MONITORING RESPONSE AS IN

a. Mechanically ventilated patient

b. Patients receiving non invasive assisted ventilation

c. Patients with respiratory failure

d. Patients with chronic hypercapnia receiving oxygen

e. Critically ill patients undergoing surgery

f. Candidates for long term oxygen therapy



COMPONENTS OF THE ARTERIAL BLOOD GAS

- The arterial blood gas provides the following values:

- **pH**

Measurement of acidity or alkalinity, based on the hydrogen (H^+) ions present.

The normal range is 7.35 to 7.45

- **PaO₂**

The partial pressure of oxygen that is dissolved in arterial blood.

The normal range is 80 to 100 mm Hg.

- **SaO₂**

The arterial oxygen saturation.

The normal range is 95% to 100%.



COMPONENTS OF THE ARTERIAL BLOOD GAS (CONTD.)

◦ PaCO_2

The amount of carbon dioxide dissolved in arterial blood.

The normal range is 35 to 45 mm Hg.

◦ HCO_3

The calculated value of the amount of bicarbonate in the bloodstream.

The normal range is 22 to 26 mEq/liter

◦ B.E.

The base excess indicates the amount of excess or insufficient level of bicarbonate in the system.

The normal range is -2 to $+2$ mEq/liter.

(A negative base excess indicates a base deficit in the blood.)



PERTAINING A SAMPLE FROM AN ARTERIAL PUNCTURE

PREPARATION

The sites of puncture

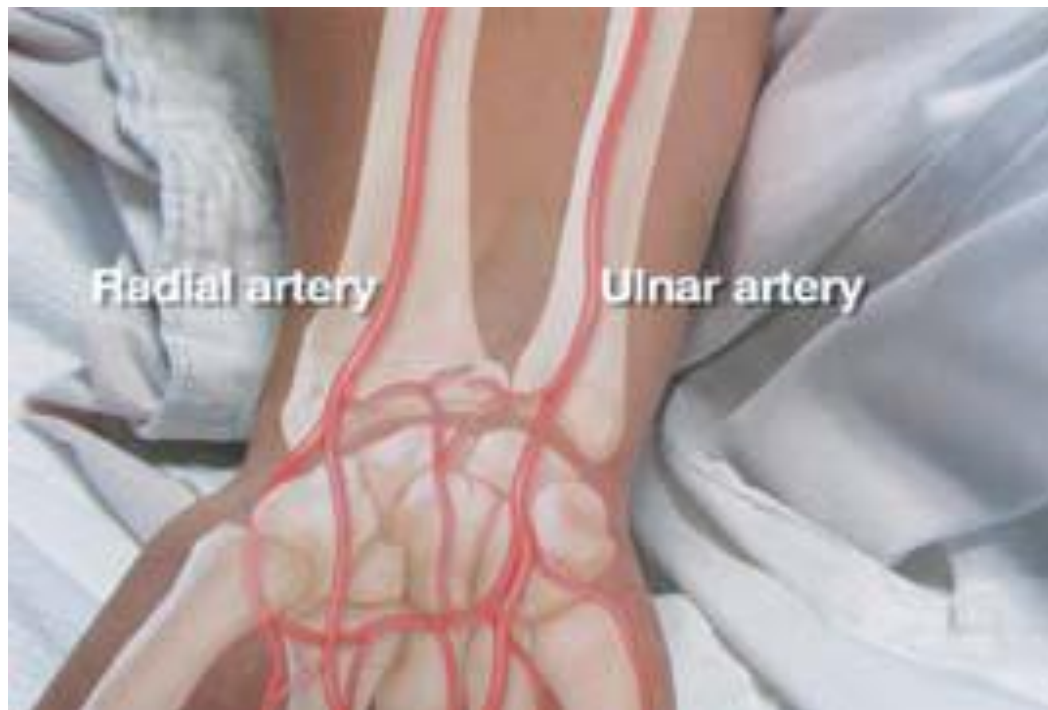
Three most commonly used sites are:

1. Radial artery
2. Brachial artery
3. Femoral artery

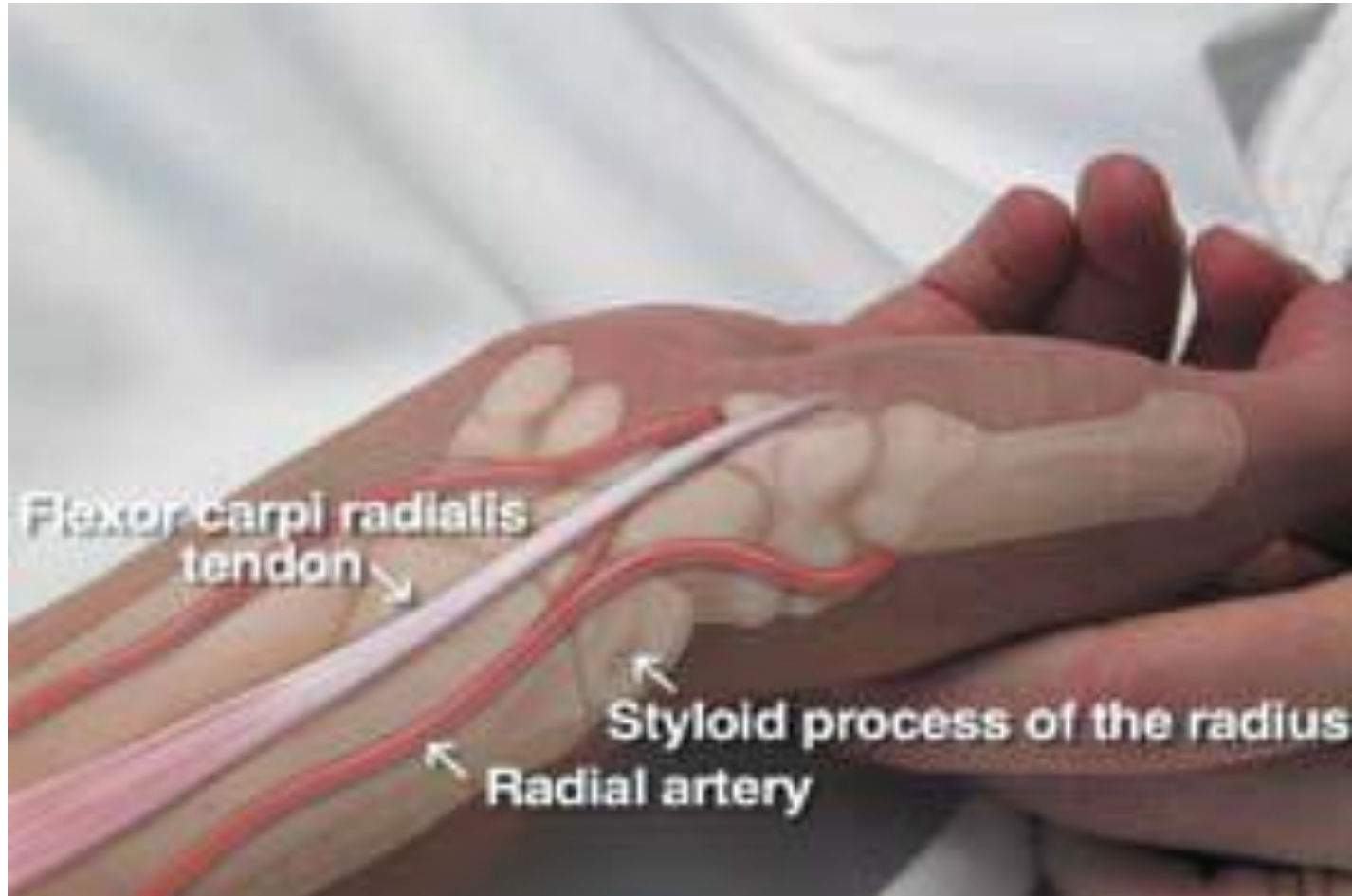


The radial artery is most oftenly used in conscious patients as

1. Easy to access
2. Superficial
3. Pressure can be applied to arrest bleeding



LOCATING LANDMARKS OF RADIAL ARTERY



EQUIPMENT

The following equipment is required to take an arterial blood sample:

1. Skin preparation fluid-alcohol or iodine based
2. 2ml syringe containing 0.5 % or 1 % plain lignocaine with 25G needle attached
3. 23G needle attached to a heparinised syringe
4. Sharps cutter and bin
5. Gauze swabs or cotton wool
6. Cap to seal syringe
7. Ice if transportation to the lab will take over 5 mins





PROCEDURE



Take the consent and assemble the equipment

Position the patient as per planned site of puncture(In case of radial artery, patient should lie semi-recumbent on bed with non-dominant arm supported on a pillow)

Wrist is extended by 20 to 30 degree to move the artery into a more superficial position.



Once the above tasks are completed, following sequence of events should be carried out:

- Clean the area & allow the agent to dry.
-
- Determine the point of maximum impulse
- Inject a analgesic around the artery and wait 30 to 60 s
- Relocate the maximum impulse with the index and middle fingers of your nondominant hand.
- Holding the ABG syringe with your dominant hand puncture the skin at a 30-to-45-degree angle at a point just below the index and middle fingers of your nondominant hand.
- Advance the needle slowly until the syringe passively fills with bright red blood

After the
blood
sample is
collected,

- Withdraw the syringe
- Apply pressure for 5 mins
- Expel air bubbles from the syringe.
- Apply the safety cap
- Make sure that the syringe is labeled with the patient's name and unit number.
- Affix the gauze with some tape.
- Dispose of all sharps in designated sharps containers.



Identification of radial pulse



Cleaning of desired radial artery puncture site



Insertion of needle at radial artery puncture site.

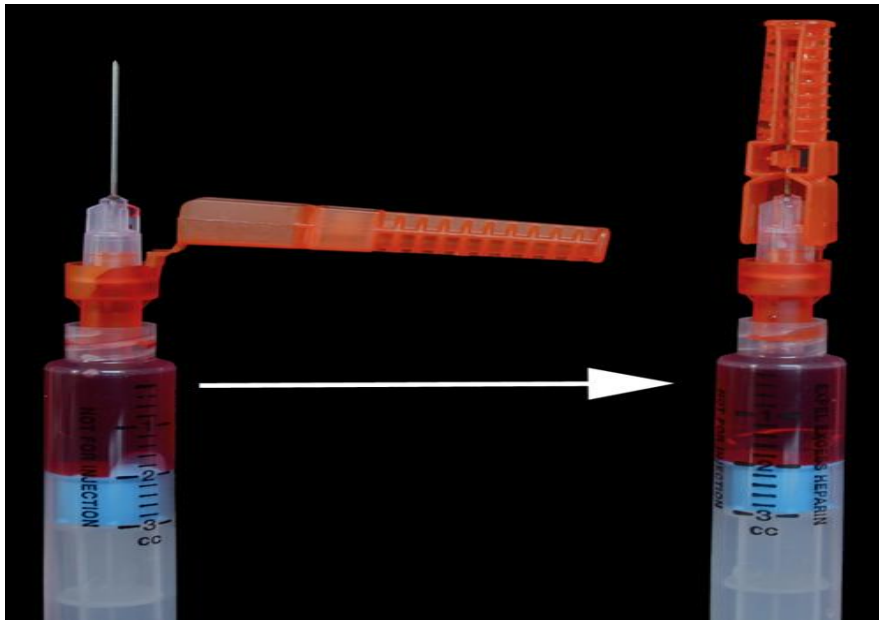




Radial artery puncture



Application of local pressure



Application of needle protective sleeve

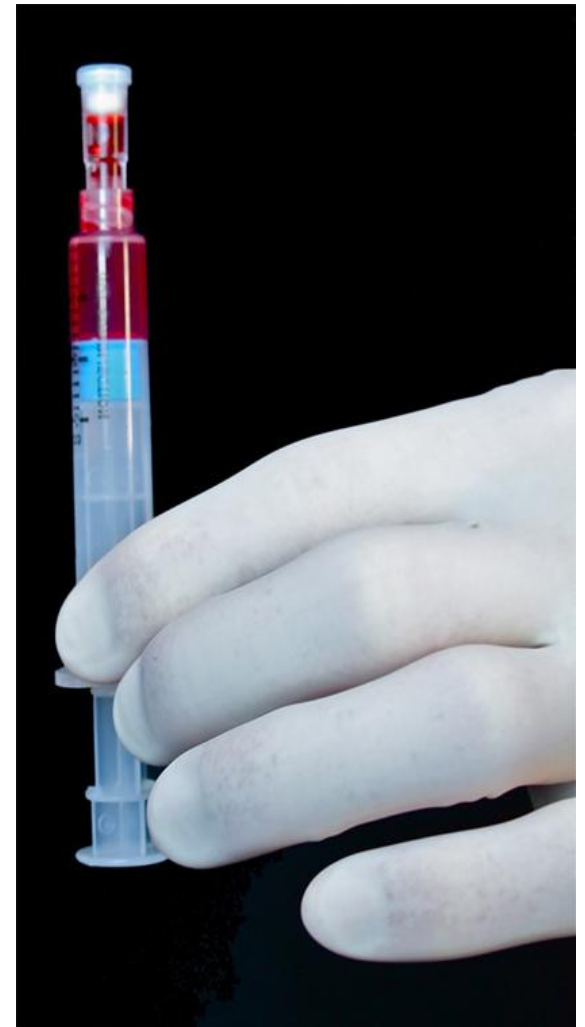




Disposal of needle



Removal of air bubbles from syringe



Capping of syringe

CONTRAINDICATIONS TO THE USE OF RADIAL ARTERY

- *Absent collateral circulation*
- Radial arterial puncture is contraindicated in the presence of a known deficiency of collateral circulation to the distal upper extremity.
- A *modified Allen's test* can be performed to assess the adequacy of the collateral circulation of the radial artery by the ulnar artery



MODIFIED ALLEN'S TEST

1) The hand is elevated and the patient/person is asked to make a fist for about 30 seconds.



2) Pressure is applied over the ulnar and the radial arteries so as to occlude both of them.



3) Still elevated, the hand is then opened. It should appear blanched (pallor can be observed at the finger nails).

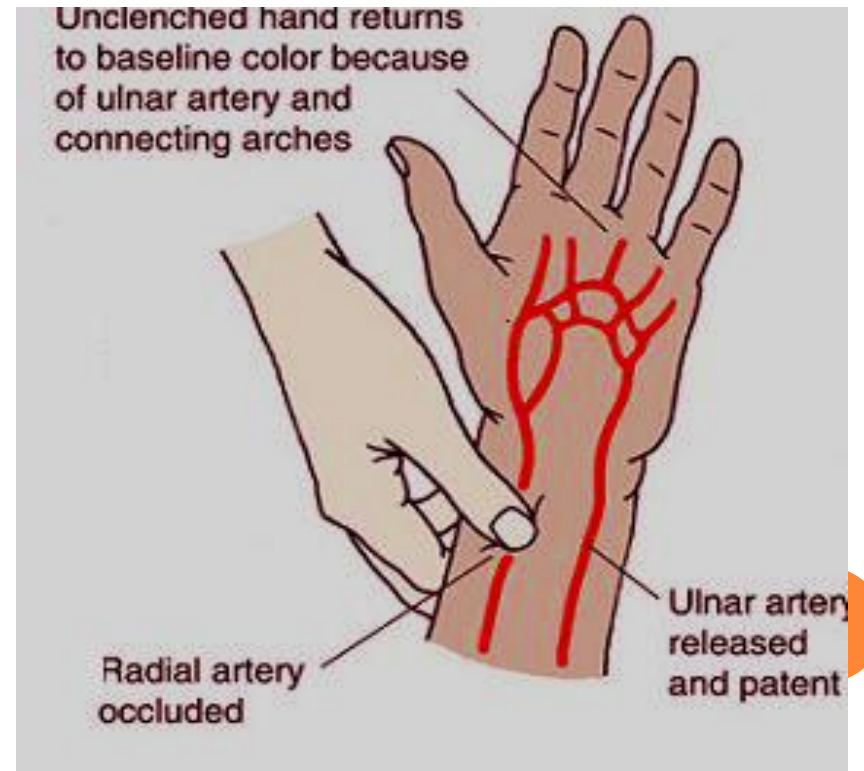
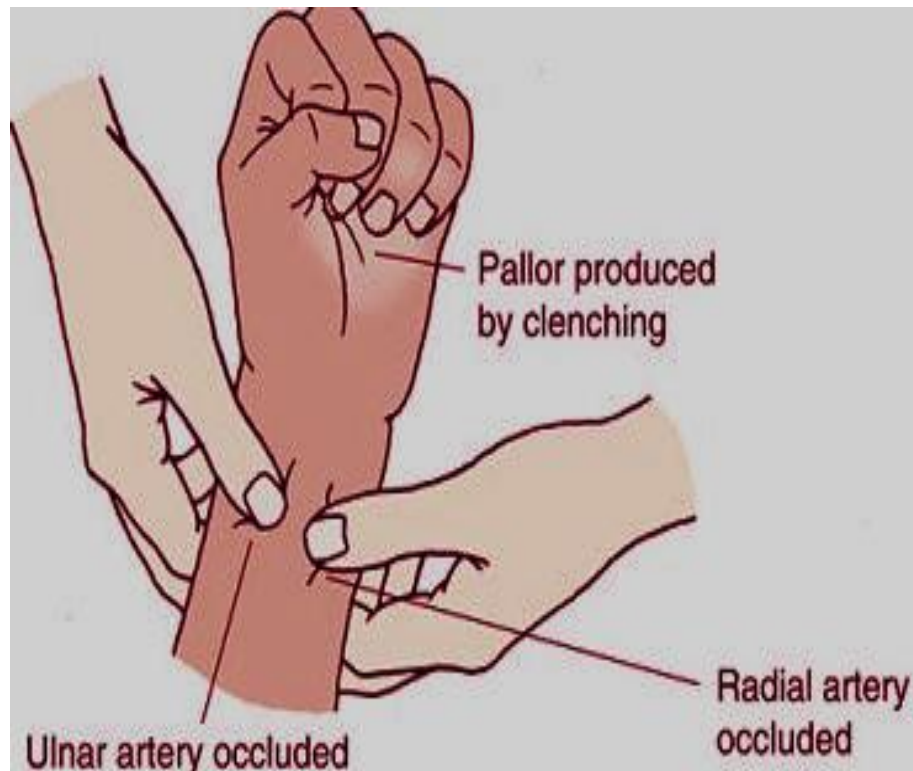


4) Ulnar pressure is released and the color should return in 7 seconds.

MODIFIED ALLEN'S TEST

Inference: Ulnar artery supply to the hand is sufficient and it is safe to cannulate/prick the radial

If color does not return or returns after 7–10 seconds, the test is considered negative and the ulnar artery supply to the hand is not sufficient. The radial artery therefore cannot be safely pricked/cannulated.



CONTRAINDICATIONS TO THE USE OF RADIAL ARTERY (CONTD.)

- Impaired circulation in the hand for e.g. Raynaud's disease or Buerger's disease
- Underlying skeletal trauma
- An arterio-venous fistula
- Overlying skin infection



ALTERNATIVES TO RADIAL ARTERY

- *Brachial artery*- its is used as an alternative to radial artery but it is deeper
- In this case needle is inserted medial to the biceps tendon over point of maximum pulsation.

Remember median nerve lies medial to the artery

C/I to using brachial artery:

1. Impaired circulation distally
2. Fracture around the elbow
3. An A-V fistula in the forearm



ALTERNATIVES TO THE RADIAL ARTERY (CONTD.)

- *The femoral artery is used*
 1. When the above two sites have failed
 2. The patient is in shock
 3. Peripheral arteries are difficult to palpate
- It is the deepest of the three arteries
- Here the needle is introduced at the mid-inguinal point, 2cm below the inguinal ligament over the point of maximum pulsation



COMPLICATIONS

- Local hematoma
- Artery vasospasm
- Arterial occlusion
- Air or thrombus embolism
- Local anesthetic anaphylactic reaction
- Infection at the puncture site



COMPLICATIONS CONTD.

- Needle stick injury to health care personnel
- Vessel laceration
- Vasovagal response
- Hemorrhage
- Local pain



POTENTIAL PREANALYTICAL ERRORS

During preparation prior to sampling

- Missing or wrong patient/sample identification
- Use of the incorrect type or amount of anticoagulant - heparin;
- Inadequate stabilization of the respiratory condition of the patient; and
- Inadequate removal of flush solution in arterial lines prior to blood collection

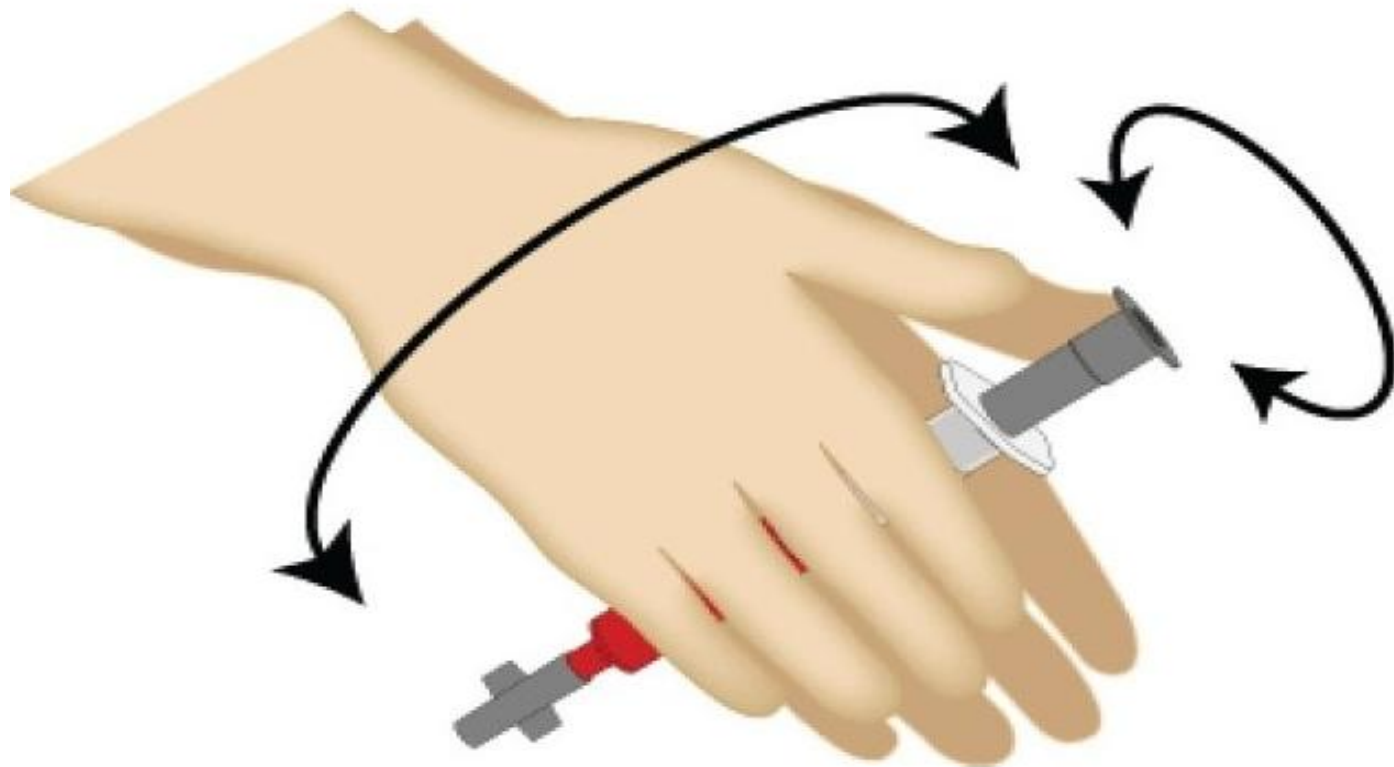


◦ *During sampling/handling*

- *Mixture of venous and arterial blood during puncturing;*
- *Air bubbles in the sample*
 - Air in the bubbles have a $PO_2 \sim 150$ mmHg & $PCO_2 \sim 0$ mm Hg
 - Mixing with sample lead to \uparrow PaO_2 & \downarrow $PaCO_2$



- *Insufficient mixing with heparin*-It is recommended to mix the blood sample thoroughly by inverting the syringe 10 times and rolling it between the palms



During storage/transport

- Incorrect storage
- Hemolysis of blood cells

General Storage Recommendation

- Do not cool the sample.
- Analyze within 30 min. For samples with high paO_2 e.g., shunt or with high leukocyte or platelet count also analyze within 5 min.
- When analysis is expected to be delayed for more than 30 minutes, use of glass syringes and ice slurry is recommended.




CHECKING FOR CONSISTENCY OF ABG

While making an interpretation of an ABG always check for the consistency of the report by the modified Henderson equation.

$$\frac{[H^+] [HCO_3^-]}{PaCO_2} = 24$$

The hydrogen ion is calculated by subtracting the two digits after the decimal point of pH from 80



The hydrogen can be calculated from

Table 1

pH value and corresponding H⁺ ion concentration

pH	H ⁺	pH	H ⁺
6.70	200	7.40	40
6.75	178	7.45	35
6.80	158	7.50	32
6.85	141	7.55	28
6.90	126	7.60	25
6.95	112	7.65	22
7.00	100	7.70	20
7.05	89	7.75	18
7.10	79	7.80	16
7.15	71	7.85	14
7.20	63	7.90	13
7.25	56	7.95	11
— — —	— —	— — —	— —



THIS IS THE TYPE OF ABG MACHINE IN OUR HOSPITAL





METABOLIC ACIDOSIS

- *A primary metabolic acidosis is characterized by low arterial pH (< 7.35), reduced plasma HCO_3^- concentration, and compensatory alveolar hyperventilation resulting in decreased PCO_2 .*
- In metabolic acidosis, this reduction in bicarbonate ions may result from increased extracellular buffering of an increased acid load or less commonly; loss of bicarbonate ions in the urine.
- The body responds to metabolic acidosis by trying to restore the $\text{PCO}_2 / [\text{HCO}_3^-]$ ratio. This is done by reducing the PCO_2 .
- The increase in ventilation is referred to as **Kussmaul Respiration**.



DISORDER	PRIMARY RESPONSES			COMPENSATORY RESPONSE
Metabolic acidosis	$\uparrow [\text{H}^+]$	$\downarrow \text{PH}$	$\downarrow \text{HCO}_3^-$	$\downarrow \text{pCO}_2$



- *In general, respiratory compensation results in a 1.2 mmHg reduction in PCO₂ for every 1.0 meq/L reduction in the plasma HCO₃⁻ concentration down to a minimum PCO₂ of 10 to 15mmHg.*

- **Winter's Formula**

To estimate the expected PCO₂ range based on respiratory compensation, one can also use the Winter's Formula which predicts:

$$PCO_2 = (1.5 \times [HCO_3^-]) + 8 \pm 2$$



- For example, if an acid load lowers the plasma HCO_3^- concentration to 9 meq/L, then:

Degree of HCO_3^- reduction is **24 (optimal value) – 9 = 15.**

Therefore, PCO_2 reduction should be **$15 \times 1.2 = 18$.**

Then PCO_2 measured should be **40 (optimal value) – 18 = 22mmHg.**



ANION GAP

- AG traditionally used to assess acid-base status esp in D/D of met acidosis

AG based on principle of electroneutrality:

- Total Serum Cations = Total Serum Anions
- $\text{Na} + (\text{K} + \text{Ca} + \text{Mg}) = \text{HCO}_3 + \text{Cl} + (\text{PO}_4 + \text{SO}_4 + \text{Protein} + \text{Organic Acids})$
- $\text{Na} + \text{UC} = \text{HCO}_3 + \text{Cl} + \text{UA}$
- $\text{Na} - (\text{HCO}_3 + \text{Cl}) = \text{UA} - \text{UC}$
- $\text{Na} - (\text{HCO}_3 + \text{Cl}) = \text{AG}$



- If the anion of the acid added to plasma is Cl^- , the anion gap will be normal (i.e., the decrease in $[\text{HCO}_3^-]$ is matched by an increase in $[\text{Cl}^-]$)
- In contrast, if the anion of the acid is not Cl^- (e.g. lactate, β -hydroxybutyrate), the anion gap will increase (i.e. the decrease in $[\text{HCO}_3^-]$ is not matched by an increase in the $[\text{Cl}^-]$ but rather by an increase in the [unmeasured anion])



Unmeasured Anions	vs	Unmeasured Cations
Proteins, mostly albumin 15 mEq/L		Calcium 5 mEq/L
Organic acids 5 mEq/L		Potassium 4.5 mEq/L
Phosphates 2 mEq/L		Magnesium 1.5 mEq/L
Sulfates 1 mEq/L		
Totals: 23 mEq/L		11 mEq/L

- Normal value of AG = 12 ± 4 meq/L
- Revised N value AG = 8 ± 4 meq/L
- Changes in methods of measurement of Na, Cl &

HCO₃ and resultant shift of Cl value to higher range.



DIFFERENTIAL DIAGNOSIS

Elevated Anion Gap (>16 meq)	Normal Anion Gap (8-16 meq)
<p>Increased Endogenous production:</p> <p>Ketoacidosis (Alcohol, Starvation, DKA)</p> <p>Lactic Acidosis</p> <p>Uremia</p>	<p>Loss of Bicarbonate: Diarrhea Carbonic anhydrase inhibitors Type 2 RTA (proximal) Pancreatic ileostomy Pancreatic, biliary, intestinal fistula</p> <p>Exogenous Administration: ammonium chloride or HCL</p> <p>Decreased Renal Acid Excretion: Type 1(distal) ,4 RTA Renal Failure</p>
<p>Intoxications: Methanol, Ethylene Glycol, Paraldehyde, Salicylates, INH</p>	<p>Miscellaneous: Hyperkalemia Recovery from DKA</p>



DELTA AG & DELTA HCO₃

- DELTA RATIO = $\frac{\text{DELTA AG}}{\text{DELTA HCO}_3}$
- ΔAG & ΔHCO_3^- used to assess mixed acid base disorders where

$$\Delta \text{AG} = \text{Observed AG} - \text{Upper normal AG} = \Delta \text{HCO}_3$$

i.e (AG - 12)

$$\Delta \text{HCO}_3 = \text{Lower normal HCO}_3 - \text{Observed HCO}_3$$

i.e (24 - [HCO₃-])

In uncomplicated high AG metabolic acidosis, Based on assumption that for each 1 meq/L increase in AG, HCO₃ will fall by 1 meq/L

$$\Delta \text{AG} = \Delta \text{HCO}_3^-$$

Any significant deviation from this rule implies a mixed acid base disorder



Delta ratio- $\frac{\Delta \text{ Anion gap}}{\Delta [\text{HCO}_3^-]}$	Assessment Guidelines
< 1	High AG & normal AG acidosis
<p>A delta-delta value below 1:1 indicates a greater fall in $[\text{HCO}_3^-]$ than one would expect given the increase in the anion gap. This can be explained by a mixed metabolic acidosis, i.e a combined elevated anion gap acidosis and a normal anion gap acidosis, as might occur when lactic acidosis is superimposed on severe diarrhea</p>	
1 to 2	Pure Anion Gap Acidosis Lactic acidosis: average value 1.6 DKA more likely to have a ratio closer to 1 due to urine ketone loss
> 2 (A value above 2:1 indicates a lesser fall in $[\text{HCO}_3^-]$ than one would expect given the change in the anion gap)	High AG acidosis and a concurrent metabolic alkalosis or a pre-existing compensated respiratory acidosis



RESPIRATORY ACIDOSIS

- Respiratory Acidosis is an acid base disturbance characterized by an elevation in the $p\text{CO}_2$ leading to an elevation in the $\text{PCO}_2/[\text{HCO}_3^-]$ ratio which subsequently increases the hydrogen ion concentration according to the following equation:

$$[\text{H}^+] = 24 \times \text{PCO}_2 / [\text{HCO}_3^-]$$

- In Respiratory Acidosis, the elevation in PCO_2 result from a reduction in alveolar ventilation. **Elevation in PCO_2 is never due to an increase in CO_2 production.**



Respiratory
acidosis

↑ [H⁺]

↓ PH

↑ pCO₂

↑ HCO₃⁻



- In response to the increase in $[H^+]$ and reduction of the pH, the body responds by trying to increase the plasma $[HCO_3^-]$ to match the increase in PCO_2 and thus maintain the PCO_2/HCO_3^- ratio.
- This is accomplished via two mechanisms;
 - a) **rapid cell buffering**
 - b) **an increase in net acid excretion.**
- Because these mechanisms occur at different moments in time, acute respiratory acidosis can be distinguished from chronic respiratory acidosis

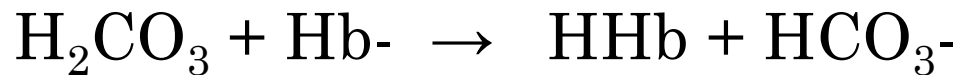


ACUTE RESPIRATORY ACIDOSIS

- Cell buffering occur within minutes after the onset of respiratory acidosis.
- The elevation in CO₂ levels lead to an increase in carbonic acid in the plasma.
- Unlike nonvolatile acids, carbonic acid (H₂CO₃) cannot be buffered by HCO₃⁻ in the extracellular fluid.
- **Therefore, in contrast to metabolic acidosis, bicarbonate levels do not fall in respiratory acidosis.**



- In this setting, carbonic acid (H_2CO_3) can only be buffered by the limited **intracellular buffers (primarily hemoglobin and proteins)**.



- As shown above, each buffering reaction produces HCO_3^- , which leads to an increase in plasma $[\text{HCO}_3^-]$.
- *Due to this process, acutely, there is an increase in the plasma $[\text{HCO}_3^-]$, averaging 1 meq/L for every 10 mmHg rise in the PCO_2 .*



CHRONIC RESPIRATORY ACIDOSIS

- In chronic respiratory acidosis, the persistent elevation in PCO_2 stimulates increased excretion of titratable acid and ammonium, resulting in the addition of new HCO_3^- to the extracellular fluid.
- This process is complete after 3-5 days resulting in a new steady state in which there is approximately *a 3.5 meq/L increase in the plasma HCO_3^- concentration for every 10 mmHg increase in the PCO_2 .*



- If the PCO₂ is acutely increased to 80 mmHg

Change in PCO₂ = 80 - 40 = 40.

Therefore elevation in [HCO₃⁻] = 40/10 × 1 = 4

According to the Henderson-Hasselbach equation,

$$\text{pH} = 6.1 + \log[\text{HCO}_3^-]/0.03 \text{ PCO}_2$$

$$\text{Hence pH} = 6.1 + \log (28/ 0.03 \times 80) = 7.17.$$

- In another example: If the PCO₂ were chronically increased to 80 mmHg, the plasma [HCO₃⁻] should rise by 14 ([(80-40)/10] × 3.5) to a new concentration of 38 meq/L (24+14)

The pH in this situation would be:

$$\text{pH} = 6.1 + \log (38/ 0.03 \times 80) = 7.30$$





Thus renal compensation offers more significant pH protection in the setting of chronic respiratory acidosis in contrast to intracellular buffering in the acute situation.

Chronic respiratory acidosis is commonly caused by COPD. These patients can tolerate a PCO₂ of up to 90-110 mmHg and not have a severe reduction in pH due to renal compensation.



METABOLIC ALKALOSIS

- Metabolic alkalosis is an acid base disorder characterized by an elevation in $[\text{HCO}_3^-]$ above the normal range, which leads to a reduction in the $\text{PCO}_2/[\text{HCO}_3^-]$ ratio and subsequently a reduction in hydrogen ion concentration according to the following equation:
$$[\text{H}^+] = 24 \times (\text{PCO}_2 / [\text{HCO}_3^-])$$
- This elevation in bicarbonate ions is due to an addition in alkali to the body which then cannot be excreted by the kidney.



Metabolic
alkalosis

↓ [H⁺]

↑ PH

↑ HCO₃⁻

↑ pCO₂



- Metabolic alkalosis is always associated with renal impairment of some kind because the kidney has a vast capacity in excreting excess alkali.
- Loss of acid from the body as occurs in vomiting induced metabolic alkalosis is equivalent to adding alkali to the body.
- In response to the reduction in $[H^+]$ and elevation in pH, the body responds by trying to increase the PCO_2 to match the increase in $[HCO_3^-]$ and thus maintain the $PCO_2/[HCO_3^-]$ ratio. Elevation in PCO_2 is accomplished by lowering alveolar ventilation.



- The development of alkalemia is sensed by central and peripheral chemoreceptors, resulting in a reduction in the rate of ventilation and a reduction in tidal volume and thus an elevation in the $p\text{CO}_2$.
- *On average the $p\text{CO}_2$ rises 0.7 mmHg for every 1.0 meq/L increment in the plasma $[\text{HCO}_3^-]$.*



RESPIRATORY ALKALOSIS

- Respiratory alkalosis is caused by an elevation in the frequency of alveolar ventilation and more importantly tidal volume that result in an increase in minute ventilation. The increase in ventilation leads to the excretion of CO₂ at a rate greater than that of cellular CO₂ production.
- This leads to a net reduction in PCO₂ and subsequently to a reduction in the PCO₂ / [HCO₃⁻] ratio which reduces the hydrogen ion concentration (and increases the pH) according to the following equation:
- $[H^+] = 24 \times PCO_2 / [HCO_3^-]$



Respiratory
alkalosis

↓ $[H^+]$

↑ PH

↓ pCO₂

↓ HCO₃⁻

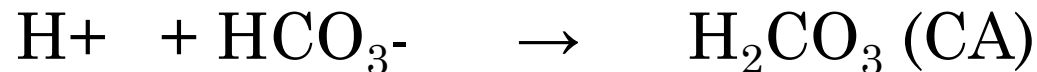


- In response to the decrease in $[H^+]$ and elevation in pH, the body responds by trying to reduce the plasma $[HCO_3^-]$ to match the reduction in PCO_2 and thus maintain the ratio. There are two mechanisms responsible for this compensation to respiratory alkalosis;
 - 1) rapid cell buffering**
 - 2) a decrease in net renal acid excretion.**
- As in respiratory acidosis, these responses occur in different moments of time, distinguishing acute respiratory alkalosis from chronic respiratory alkalosis.



ACUTE RESPIRATORY ALKALOSIS

- About 10 minutes after the onset of respiratory alkalosis, hydrogen ions move from the cells into the extracellular fluid, where they combine with HCO_3^- to form carbonic acid in the following reaction:



- The hydrogen ions are primarily derived from intracellular buffers such as hemoglobin, protein and phosphates. The reaction with bicarbonate ions in this reaction leads to a mild reduction in plasma $[\text{HCO}_3^-]$.



- *In acute respiratory alkalosis, as a result of cell buffering, for every 10 mmHg decrease in the PCO₂, there is a 2meq/L decrease in the plasma HCO₃⁻ concentration.*
- If the PCO₂ were reduced to 20 mmHg, the change in PCO₂ would be 20 (40-20) and therefore the fall in plasma [HCO₃⁻] would be 4 meq/L (20/10 × 2). The new plasma [HCO₃⁻] would be 20 meq/L (24-4).
- The pH in this circumstance would be:
$$\text{pH} = 6.1 + \log (20 / 0.03 \times 20) = 7.63$$

Had no cell buffering occurred, then the pH would be
$$\text{pH} = 6.1 + \log (24 / 0.03 \times 20) = 7.70$$



CHRONIC RESPIRATORY ALKALOSIS

- If respiratory alkalosis persist for longer than 2- 6 hours, the kidney will respond by lowering hydrogen secretion, excretion of titratable acids, ammonium production and ammonium excretion. There will also be an increase in the amount of HCO_3^- excreted due to decreased reabsorption of filtered HCO_3^- .
- *Renal compensation result in a 4 meq/L reduction in plasma $[\text{HCO}_3^-]$ for every 10 mmHg reduction in PCO_2*



- In comparison, to acute respiratory alkalosis, this compensation offers a much better protection of the arterial pH.
- Consider the same 20 mmHg fall in PCO₂ as before in the acute scenario. Now, due to renal compensation, the plasma [HCO₃⁻] falls by 8 meq/L to 16 meq/L. The pH now in the chronic situation would be :

$$\text{pH} = 6.1 + \log (16 / 0.03 \times 20) = 7.53.$$



- 1. Compensatory responses never return the pH to normal or overshoot.
- 2. The basis of compensatory responses is to maintain the $\text{PCO}_2/[\text{HCO}_3^-]$ ratio.
- 3. Therefore, the direction of the compensatory response is always the same as that of the initial change.



- 4. Compensatory response to respiratory disorders is two-fold; a fast response due to cell buffering and a significantly slower response due to renal adaptation.
- 5. Compensatory response to metabolic disorders involves only an alteration in alveolar ventilation.
- 6. Metabolic responses cannot be defined as acute or chronic in terms of respiratory compensation because the extent of compensation is the same in each case.



Primary disorder	Initial chemical change	Compensatory response	Compensatory Mechanism	Expected level of compensation
Metabolic Acidosis	↓HCO ₃ ⁻	↓PCO ₂	Hyperventilation	$PCO_2 = (1.5 \times [HCO_3^-]) + 8 \pm 2$
				$\downarrow PCO_2 = 1.2 \times \Delta [HCO_3^-]$
				PCO ₂ = last 2 digits of pH
Metabolic Alkalosis	↑HCO ₃ ⁻	↑PCO ₂	Hypoventilation	$PCO_2 = (0.9 \times [HCO_3^-]) + 16 \pm 2$
				$\uparrow PCO_2 = 0.7 \times \Delta [HCO_3^-]$
Respiratory Acidosis	↑PCO ₂	↑HCO ₃ ⁻		
Acute			Intracellular Buffering (hemoglobin, intracellular proteins)	$\uparrow [HCO_3^-] = 1 \text{ mEq/L for every } 10 \text{ mm Hg } \Delta PCO_2$
Chronic			Generation of new HCO ₃ ⁻ due to the increased excretion of ammonium.	$\uparrow [HCO_3^-] = 3.5 \text{ mEq/L for every } 10 \text{ mm Hg } \Delta PCO_2$
Respiratory Alkalosis	↓PCO ₂	↓HCO ₃ ⁻		
Acute			Intracellular Buffering	$\downarrow [HCO_3^-] = 2 \text{ mEq/L for every } 10 \text{ mm Hg } \Delta PCO_2$
Chronic			Decreased reabsorption of HCO ₃ ⁻ , decreased excretion of ammonium	$\downarrow [HCO_3^-] = 4 \text{ mEq/L for every } 10 \text{ mm Hg } \Delta PCO_2$

Also: In acute respiratory acidosis, $\downarrow pH = 0.008 \times \Delta PCO_2$

In chronic respiratory acidosis, $\downarrow pH = 0.003 \times \Delta PCO_2$.

GAS EXCHANGE: 3 KEY EQUATIONS FOR EVALUATION

- 1) PaCO₂ equation : Evaluating alveolar ventilation...
- 2) Alveolar gas equation: Evaluating oxygen transfer at the alveolar level...
- 3) Oxygen content equation: Evaluating oxygen transfer at the tissue level...



PACO₂ EQUATION: PACO₂ REFLECTS RATIO OF METABOLIC CO₂ PRODUCTION TO ALVEOLAR VENTILATION

$$PaCO_2 = \frac{VCO_2 \times 0.863}{VA}$$

VCO₂ = CO₂ production

VA = VE – VD

VE = minute (total) ventilation

VD = dead space ventilation

0.863 converts units to mm Hg

<u>PaCO₂</u>	Condition in blood	State of alveolar ventilation
>45 mm Hg	Hypercapnia	Hypoventilation
35 - 45 mm Hg	Eucapnia	Normal ventilation
<35 mm Hg	Hypocapnia	Hyperventilation



HYPERCAPNIA

$$\text{PaCO}_2 = \frac{\text{VCO}_2 \times 0.863}{\text{VA}}$$

The only physiologic reason for elevated PaCO_2 is inadequate alveolar ventilation (VA) for the amount of the body's CO_2 production (VCO_2). Since alveolar ventilation (VA) equals minute ventilation (VE) minus dead space ventilation (VD), hypercapnia can arise from insufficient VE, increased VD, or a combination.



HYPERCAPNIA

$$\text{PaCO}_2 = \frac{\text{VCO}_2 \times 0.863}{\text{VA}}$$

$$\text{VA} = \text{VE} - \text{VD}$$

- **Examples of inadequate VE leading to decreased VA and increased PaCO₂:**
sedative drug overdose; respiratory muscle paralysis; central hypoventilation
- **Examples of increased VD leading to decreased VA and increased PaCO₂:**
chronic obstructive pulmonary disease; severe pulmonary embolism, pulmonary edema.



ALVEOLAR GAS EQUATION

$$PAO_2 = PIO_2 - 1.2 (PaCO_2)$$


where PAO_2 is the average alveolar PO_2 , and PIO_2 is the partial pressure of inspired oxygen in the trachea

$$PIO_2 = FIO_2 (P_B - 47 \text{ mm Hg})$$

FIO_2 is fraction of inspired oxygen and P_B is the barometric pressure. 47 mm Hg is the water vapor pressure at normal body temperature.



ALVEOLAR GAS EQUATION

- $PAO_2 = PIO_2 - 1.2 (PaCO_2)$
 - $PIO_2 = FIO_2 (P_B - 47 \text{ mm Hg})$
 - **$PAO_2 = FIO_2 (P_B - 47 \text{ mm Hg}) - 1.2 (PaCO_2)$**
 - If FIO_2 and P_B are constant, then as $PaCO_2$ increases both PAO_2 and PaO_2 will decrease (hypercapnia causes hypoxemia).
 - If FIO_2 decreases and P_B and $PaCO_2$ are constant, both PAO_2 and PaO_2 will decrease.
 - If P_B decreases (e.g., with altitude), and $PaCO_2$ and FIO_2 are constant, both PAO_2 and PaO_2 will decrease (mountain climbing causes hypoxemia).
- 

$P(A-a)O_2$

- $P(A-a)O_2$ is the alveolar-arterial difference in partial pressure of oxygen. It is commonly called the “A-a gradient”. It results from gravity-related blood flow changes within the lungs
- Normal $P(A-a)O_2$ ranges from 5 to 25 mm Hg breathing room air (it increases with age). A higher than normal $P(A-a)O_2$ means the lungs are not transferring oxygen properly from alveoli into the pulmonary capillaries.



SAO₂ AND OXYGEN CONTENT

How much oxygen is in the blood? Oxygen content = CaO₂ (mlO₂/dl).

CaO₂ = quantity O₂ bound to hemoglobin + quantity O₂ dissolved in plasma

$$\text{CaO}_2 = (\text{Hb} \times 1.34 \times \text{SaO}_2) + (.003 \times \text{PaO}_2)$$

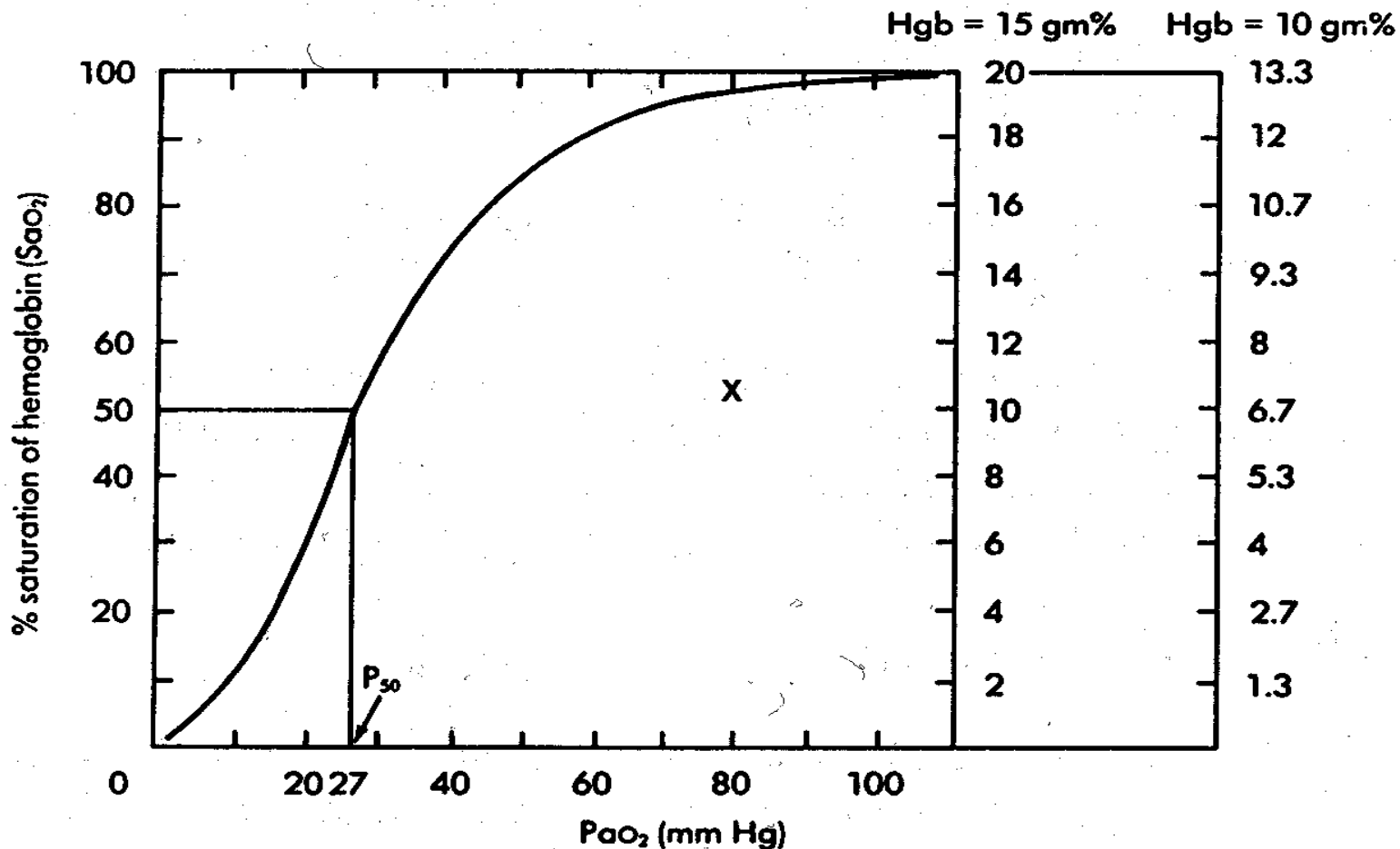
- Hb = hemoglobin in gm%; 1.34 = ml O₂ that can be bound to each gm of Hb; SaO₂ is percent saturation of hemoglobin with oxygen; .003 is solubility coefficient of oxygen in plasma: .003 ml dissolved O₂/mm Hg PO₂.



OXYGEN DISSOCIATION CURVE: SAO_2 VS. PAO_2

50%. ALSO SHOWN ARE CAO_2 VS. PAO_2 FOR TWO DIFFERENT HEMOGLOBIN CONTENTS: 15 GM%


AND 10 GM%. CAO_2 UNITS ARE ML O_2 /DL. P_{50} IS THE PAO_2 AT WHICH SAO_2 IS



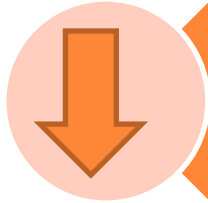
SAO₂ – IS IT CALCULATED OR MEASURED?

SaO₂ is measured in a 'co-oximeter'. The traditional 'blood gas machine' measures only pH, PaCO₂ and PaO₂, whereas the co-oximeter measures SaO₂, carboxyhemoglobin, methemoglobin and hemoglobin content. Newer 'blood gas' consoles incorporate a co-oximeter, and so offer the latter group of measurements as well as pH, PaCO₂ and PaO₂.

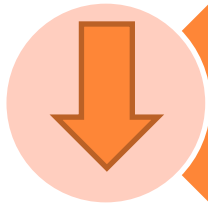
Always make sure the SaO₂ is measured, not calculated. If it is calculated from the PaO₂ and the O₂-dissociation curve, it provides no new information, and could be inaccurate -- especially in states of CO intoxication or excess methemoglobin. CO and metHb do not affect PaO₂, but do lower the SaO₂.



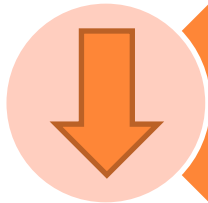
STEPWISE APPROACH TO ABG ANALYSIS



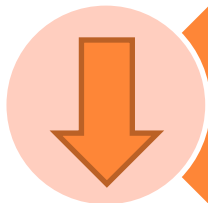
Determine whether patient is alkalemic or acidemic using the arterial pH measurement



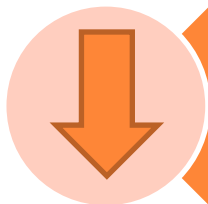
Determine whether the acid-base disorder is a primary respiratory or metabolic disturbance based on the $p\text{CO}_2$ and serum HCO_3^- level



If a primary respiratory disorder is present, determine whether it is chronic or acute



In metabolic disorders, determine if there is adequate compensation of the respiratory system



In respiratory disorders, determine if there is adequate compensation of the metabolic system





Determine pt's oxygenation status (PaO₂ & SaO₂) – hypoxemic or not

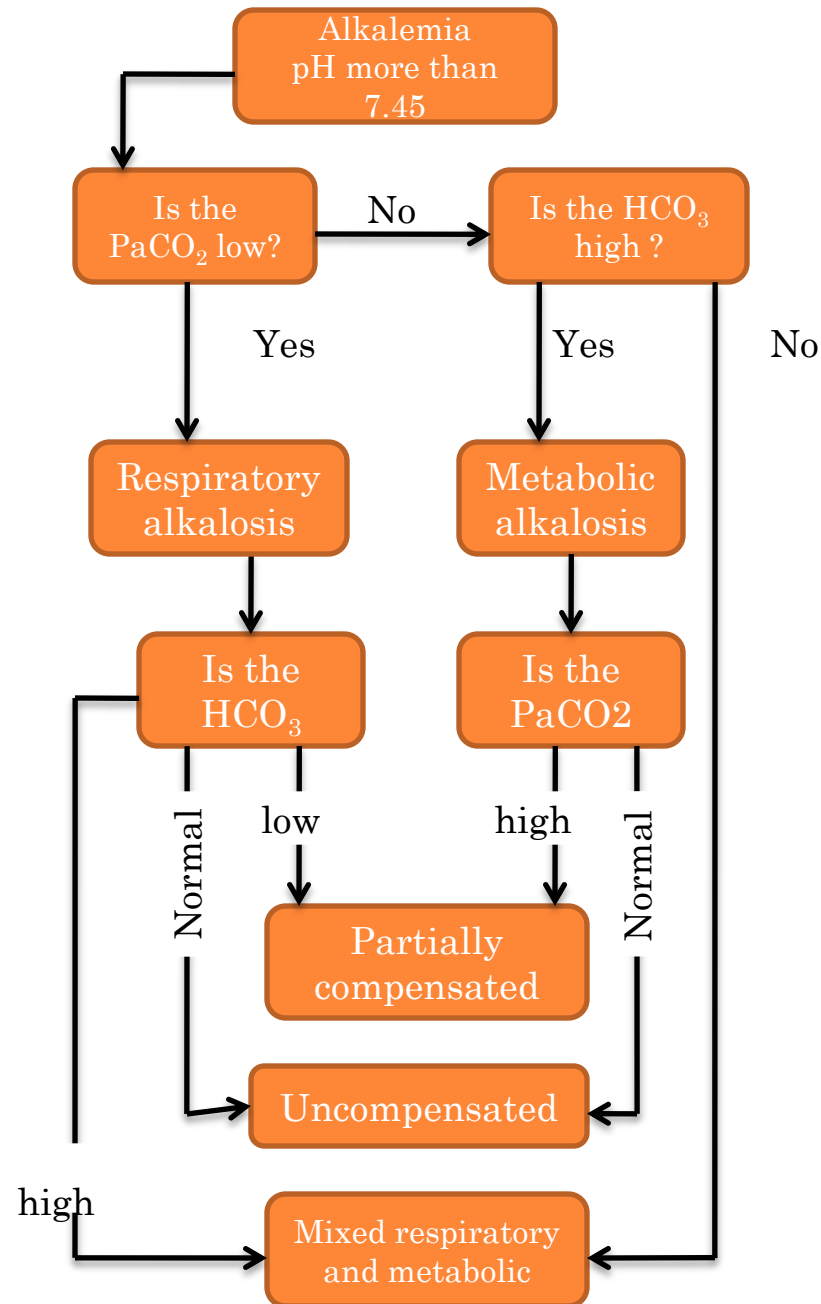


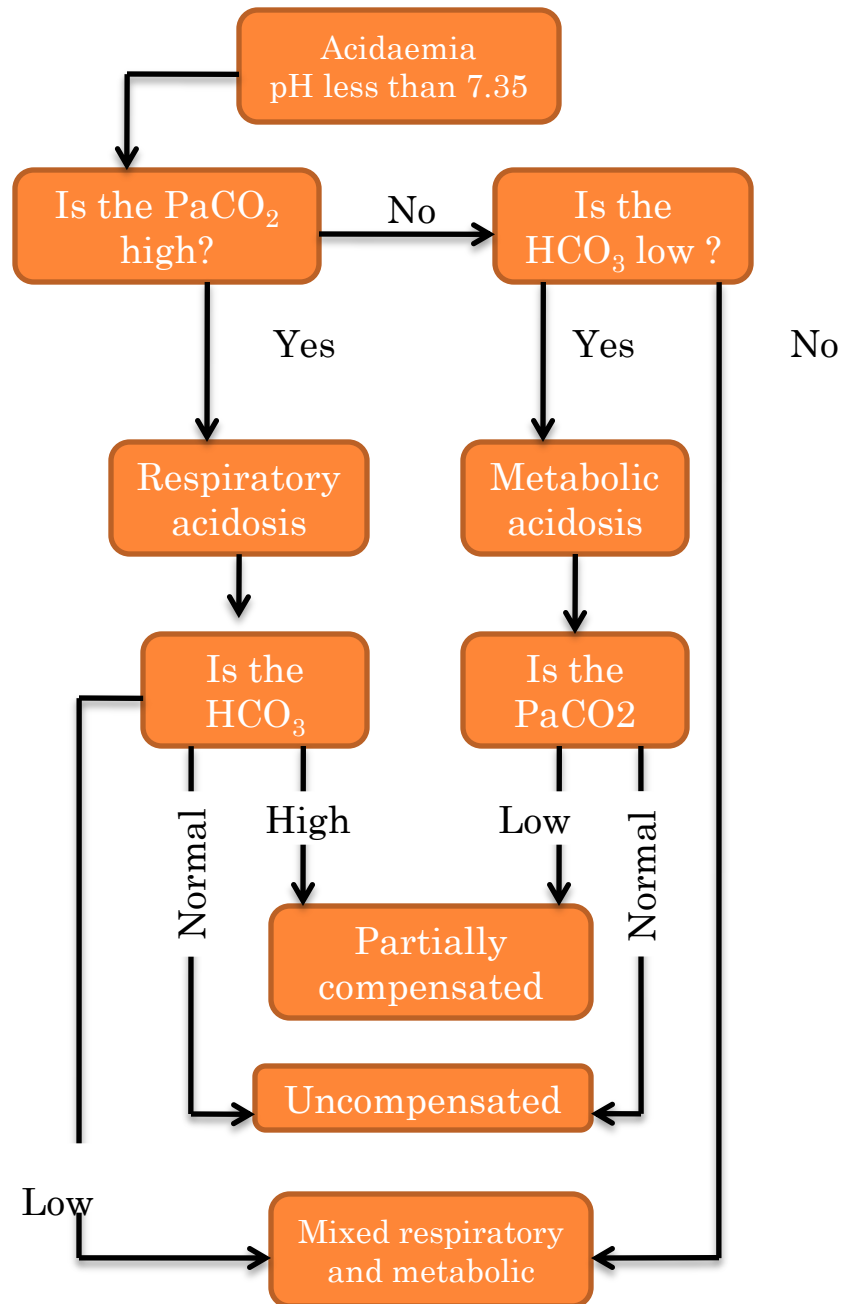
If a metabolic acidosis is present, determine the anion gap and osmolar gap



In high anion gap acidosis, determine the change in anion gap (Δ AG) & Δ HCO₃⁻ in order to assess for the presence of coexisting metabolic disturbances







CONDITIONS INVALIDATING OR MODIFYING ABG RESULTS

○ DELAYED ANALYSIS

Consumption of O₂ & Production of CO₂ continues after blood drawn into syringe

Iced Sample maintains values for 1-2 hours

Uniced sample quickly becomes invalid


PaCO₂ ↑ 3-10 mmHg/hour

PaO₂ ↓ at a rate related to initial value & dependant on Hb Sat



EFFECT OF TEMP ON RATE OF CHANGE IN ABG VALUES

Parameter	37 C (Change every 10 min)	4 C (Change every 10 min)
↓ pH	0.01	0.001
↑ PCO ₂	1 mm Hg	0.1 mm Hg
↓ PO ₂	0.1 vol %	0.01 vol %



○ EXCESSIVE HEPARIN

Dilutional effect on results ↓ HCO_3^- & PaCO_2

Syringe be emptied of heparin after flushing

Risk of alteration of results ↑ with:

1. ↑ size of syringe/needle

2. ↓ vol of sample

25% lower values if 1ml sample taken in 10 ml syringe (0.25 ml heparin in needle)

Syringes must be > 50% full with blood sample

○ HYPERVENTILATION OR BREATH HOLDING

May lead to erroneous lab results



○ AIR BUBBLES

1. $PO_2 \sim 150$ mmHg & $PCO_2 \sim 0$ mm Hg in air bubble(R.A.)
2. Mixing with sample lead to \uparrow PaO_2 & \downarrow $PaCO_2$
3. Mixing/Agitation \uparrow S.A. for diffusion \rightarrow more erroneous results
4. Discard sample if excessive air bubbles
5. Seal with cork/cap imm after taking sample



○ FEVER OR HYPOTHERMIA

1. Most ABG analyzers report data at N body temp
2. If severe hyper/hypothermia, values of pH & PCO₂ at 37 C can be significantly diff from pt's actual values
3. Changes in PO₂ values with temp predictable
4. No significant change of HCO₃⁻, O₂ Sat, O₂ capacity/content, CO₂ content values with temp



- WBC COUNT

0.1 ml of O₂ consumed/dL of blood in 10 min in pts with N TLC

Marked increase in pts with very high TLC/plt counts – hence imm chilling/analysis essential



○ TYPE OF SYRINGE

1. pH & PCO₂ values unaffected
2. PO₂ values drop more rapidly in plastic syringes (ONLY if PO₂ > 400 mm Hg)
3. Other adv of glass syringes:
Min friction of barrel with syringe wall

Usually no need to 'pull back' barrel – less chance of air bubbles entering syringe

Small air bubbles adhere to sides of plastic syringes – difficult to expel

Though glass syringes preferred, differences usually not of clinical significance → plastic syringes can be and continue to be used



SUMMARY

SERIAL ABGs

CLINICAL PROFILE

SUPPORTING LAB DATA/
INVESTIGATIONAL TOOLS

CLINICIAN'S JUDGEMENT

CORRECT INTERPRETATION

SIMPLE DISORDER
(DEG OF COMPENSATION)

MIXED DISORDER
(ORDER OF PRIMARY &
SUBSEQUENT DISORDERS)

OXYGENATION /VENTILATORY STATUS



CLINICAL PROBLEM

- Jane Doe is a patient with chronic COPD being admitted for surgery. Her admission labwork reveals an arterial blood gas with the following values:

Clinical Laboratory	
PATIENT:	DOE, JANE
DATE:	2/16/03 17:30
pH	7.35
PaCO ₂	48
HCO ₃ ⁻	28
STAT LAB	



- 1. Assess the pH. It is within the normal range, but on the low side of neutral (<7.40).
- 2. Assess the PaCO₂ (48). It is high (normal 35-45). We would expect the pH and PaCO₂ to move in opposite directions if the primary problem is respiratory.
- 3. Assess the HCO₃ (28). It is also high (22-26). Normally the pH and HCO₃ should move in the same direction. Because they are moving in opposite directions, it confirms that the primary acid-base disorder is respiratory and that the kidneys are attempting to compensate by retaining HCO₃. Because the pH has returned into the low normal range, we would interpret
- **This ABG as a *compensated respiratory acidosis*.**



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THANK U

