ACID BASE BALANCE

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INTRODUCTION

- PH of blood is maintain between 7.35-7.45 by various means.
- Buffer – It is a system that consist of weak acid and base and it prevent PH from staying too far from normal on addition of significant amount of acid or base

- Buffers in the extracellular fluid (ECF):

  -Bicarbonate

  -plasma Proteins

  -Inorganic Phosphate

- Buffers in the intracellular fluid (ICF):

  1. Hemoglobin (Hb)

  2. Proteins

  3.Organic and Inorganic Phosphate[Most imp]

- Bones-

  - Important site of buffering

  -An acid load is associated with uptake of excess H^+ ion by bone which occurs in exchange for surface Na^+ and K^+ and by dissolution of bone minerals resulting in release of buffering compounds $NaHCO_3$, $CaHCO_3$, $CaHPO_4$

- 40% of buffering of acute acid load occurs in bone
- Chronic acid load – adverse eff. on bone mineralization

BICARBONATE BUFFER

- Main extracellular buffering system
- Weak acid- H2CO3 ; Base-HCO3

- $H^+ + HCO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow H_2O + CO_2$
- **Equilibrium**
- $[H^+] = Keq \times [H_2CO_3]/[HCO_3^-]$

HOW DOES BUFFER SYSTEM REGULATE PH

- $H_2O + CO_2 \Leftrightarrow H_2CO_3 \Leftrightarrow H^+ + HCO_3^-$

$$H_2O + CO_2 \Leftrightarrow H_2CO_3 \Leftrightarrow H^+ + HCO_3^-$$

1 - For acidosis

2 -For alkalosis

3 -Effect of CO2

4 -Effect of HCO3

- Kidney and lung compensate for change in PH

Four Main Acid-Base Disorders

Disorder	Primary Alteration	Secondary Response	Mechanism of Response
Metabolic Acidosis	▼ in plasma HCO_3	▼ in plasma pCO_2	Hyperventilation
Metabolic Alkalosis	▲ in plasma HCO_3	▲ increase in pCO_2	Hypoventilation
Respiratory Acidosis	▲ in plasma pCO_2	▲ in plasma HCO_3	Increase in acid excretion; increase in reabsorption of HCO_3
Respiratory Alkalosis	▼ in plasma pCO_2	▼ in plasma HCO_3	Suppression of acid excretion; decrease in reabsorption of HCO_3

- If PH and PCO2 are going in the opposite direction : then its respiratory, If PH and PCO2 are going in same directions then its metabolic.

Normal Values

Variable	Normal Range
pH	7.35 - 7.45
Pco2 Na K	35-45 135- 155 3.5-5.5
Bicarbonate CL	22-26 95-105
Anion gap	10-14
Albumin PO2	4 > 70

Steps for ABG analysis

1. What is the pH? Acidemia or Alkalemia?
2. What is the primary disorder present?
3. Is the compensation acute or chronic?
4. Is there an anion gap?
5. If there is a AG check the delta gap?
6. What is the differential for the clinical processes?

1. Determine the primary disturbance:
   - Acidemia or Alkalemia: look at the pH
     - < 7.40 = acidemia
     - > 7.40 = alkalemia
   2. Respiratory or Metabolic: look at HCO_3 and CO_2
      - ↓ HCO_3 = primary metabolic acidosis
      - ↑ pCO_2 = primary respiratory acidosis
      - and vice versa for alkalosis

3. Compensation

- Compensation attempts to normalize pH but can be present with an abnormal pH

- Expected change in pCO_2 best used for primary metabolic disturbance and expected change in HCO_3 for primary respiratory disturbance

Respiratory Disturbance Compensation

	pCO2	in pH	in HCO3
Acute Resp. Acidosis	↑ 10	↓ 0.08	↑ 1
Chronic Resp. Acidosis	↑ 10	↓ 0.03	↑ 3

	pCO2	in pH	in HCO3
Acute Resp. Alkalosis	↓ 10	↑ 0.08	↓ 2
Chronic Resp. Alkalosis	↓ 10	↑ 0.03	↓ 5

Compensations for Metabolic Disturbances

- Metabolic acidosis
  - $pCO_2 = 1.5 \times HCO_3 + 8 \ (\pm 2)$
  - If serum pCO2 > expected pCO2 -> additional respiratory acidosis

- Metabolic alkalosis
  - pCO_2 increases by 6 for every 10 mEq increases in HCO_3

Calculate the anion gap

- AG = Na –[Cl + HCO3] (normal 12 ± 2)
- AG corrected = AG + 2.5[4 – albumin]
- If there is an anion Gap then calculate the Delta/delta gap . Only need to calculate delta gap (excess anion gap) when there is an anion gap to determine additional hidden metabolic disorders (nongap metabolic acidosis or metabolic alkalosis)
- If there is no anion gap then start analyzing for non-anion acidosis

- **Delta gap = (actual AG – 12) + HCO3**
- Adjusted HCO3 should be 24 (+_ 6) {18-30}
- If delta gap > 30 -> additional metabolic alkalosis
- If delta gap < 18 -> additional non-gap metabolic acidosis
- If delta gap 18 – 30 -> no additional metabolic disorders

- For non-gap metabolic acidosis, calculate the urine anion gap
  - $\mathbf{U_{AG} = U_{NA} + U_{K} - U_{CL}}$
- If UAG>0: renal problem
- If UAG<0: nonrenal problem (most commonly GI)

Metabolic Acidosis Anion Gap "MUDPILERS"	Metabolic Acidosis Non-Gap "HARDUPS"	Acute Resp. Acidosis "anything causing hypoventilation"	Metabolic Alkalosis "CLEVERPD"	Respiratory Alkalosis "CHAMPS"
·Methanol ·Uremia ·DKA/Alcoholic ketoacidosis ·Paraldehyde ·Isoniazid ·Lactic acidosis ·Ethanol ·Renal failure/Rhabdo ·Salicylates	·Hyperalimentation ·Acetazolamide ·Renal Tubular Acidosis ·Diarrhea ·Uretero-Pelvic shunt ·Post-hypocapnia ·Spironolactone	·CNS depression ·Airway obstruction ·Pulmonary edema ·Pneumonia ·Hemo/Pneumo thorax ·Neuromuscular	·Contraction ·Licorice ·Endocrine (Conn/Cushing /Bartters) ·Vomiting ·Excess alkali ·Refeeding ·Post-hypercapnia ·Diuretics	·CNS disease ·Hypocapnia ·Anxiety ·Mech. Ventilation ·Progesterone ·Salicylates ·Sepsis

- I.Respiratory Alkalosis

A. Central nervous system stimulation 1. Pain 2. Anxiety, psychosis 3. Fever 4. Cerebrovascular accident 5. Meningitis, encephalitis 6. Tumor 7. Trauma

B. Hypoxemia or tissue hypoxia 1. High altitude 2. Pneumonia, pulmonary edema 3. Aspiration 4. Severe anemia

- C. Drugs or hormones 1. Pregnancy, progesterone 2. Salicylates 3. Cardiac failure
- D. Stimulation of chest receptors 1. Hemothorax 2. Flail chest 3. Cardiac failure 4. Pulmonary embolism

E. Miscellaneous

1. Septicemia
2. Hepatic failure
3. Mechanical hyperventilation
4. Heat exposure
5. Recovery from metabolic acidosis

II. Respiratory Acidosis

A. Central 1. Drugs (anesthetics, morphine, sedatives) 2. Stroke 3. Infection

B. Airway1. Obstruction 2. Asthma

C. Parenchyma 1. Emphysema 2. Pneumoconiosis 3. Bronchitis 4. Adult respiratory distress syndrome 5. Barotrauma

D. Neuromuscular

1. Poliomyelitis 2. Kyphoscoliosis 3. Myasthenia 4.Musculardystrophies

E. Miscellaneous

1. Obesity
2. Hypoventilation
3. Permissive hypercapnia

Metabolic alkalosis

- Calculate the urinary chloride to differentiate saline responsive vs saline resistant
- Must be off diuretics in order to interpret urine chloride

Saline responsive U_{Cl}<10	Saline-resistant U_{Cl} >10
Vomiting	If hypertensive: Cushings, Conn's, renal failure with alkali administartion
NG suction	If not hypertensive: severe hypokalemia, hypomagnesemia, Bartter's, Gittelman's, licorice ingestion
Over-diuresis	Exogenous corticosteroid administration
Post-hypercapnia	

Nongap metabolic acidosis

For non-gap metabolic acidosis, calculate the urine anion gap

$$U_{AG} = U_{NA} + U_K - U_{CL}$$

If UAG>0: renal problem

If UAG<0: nonrenal problem (most commonly GI)

I. Gastrointestinal bicarbonate loss

A. Diarrhea

B. External pancreatic or small-bowel drainage

C. Ureterosigmoidostomy, jejunal loop, ileal loop

D. Drugs

1. Calcium chloride (acidifying agent)
2. Magnesium sulfate (diarrhea)
3. Cholestyramine (bile acid diarrhea)

II. Renal acidosis

A. Hypokalemia

1. Proximal RTA (type 2)

Drug-induced: acetazolamide, topiramate

2. Distal (classic) RTA (type 1)

Drug induced: amphotericin B, ifosfamide

B. Hyperkalemia

1. Generalized distal nephron dysfunction (type 4 RTA)

a. Mineralocorticoid deficiency

b. Mineralocorticoid resistance (autosomal dominant PHA I)

c. Voltage defect (autosomal dominant PHA I and PHA II)

d. Tubulointerstitial disease

III. Drug-induced hyperkalemia (with renal insufficiency)

A. Potassium-sparing diuretics (amiloride, triamterene,spironolactone)

B. Trimethoprim

C. Pentamidine

D. ACE-Is and ARBs

E. Nonsteroidal anti-inflammatory drugs

F. Cyclosporine and tacrolimus

IV. Other

A. Acid loads (ammonium chloride, hyperalimentation)

B. Loss of potential bicarbonate: ketosis with ketone excretion

C. Expansion acidosis (rapid saline administration)

D. Hippurate

E. Cation exchange resins

Metabolic acidosis: Anion gap acidosis

Differential for Anion Gap Metabolic Acidosis - MUDPILERS
Methanol
Uremia
Diabetic ketoacidosis, starvation ketoacidosis, EtOH ketoacidosis
Paraldehyde
INH, iron toxicity
Lactic acidosis
Ethylene glycol
Rhabdomyolysis
Salicylates

THANK YOU