# Quick Review: Acid Base Disorders

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**Citation**

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**Abstract**

Normal blood pH is 7.40 (7.36 - 7.44), which corresponds to a [H+] of 40 nEq/L (44-36).
Systemic arterial pH is maintained by complex buffering mechanisms as well as renal and respiratory compensatory responses. This brief article reviews the basics of acid-base disorders.

## GENERAL CONCEPTS

The kidneys regulate $HCO3^-$ by the following mechanisms:

Reabsorption of filtered $HCO3^-$

Formation of titrable acid
Excretion of NH4+ in the urine

Acidemia: serum pH < 7.36
Alkalemia: serum pH > 7.44

Acidosis: pathophysiologic processes, which favor development of acidemia
Alkalosis: pathophysiologic processes, which favor development of alkalosis

Buffer: A substance, which can absorb or donate H+ ions in order to mitigate changes pH.

$H2CO3^-$ H+ + $HCO3^-$ H2O + CO2
* Remember: [H+] ion concentration and pH are inversely related.

## HENDERSON-HASSELBALCH EQUATION:

pH = pK + log ($HCO3^-$) pK = 6.1 (PaCO2)

## KASSIRER-BLEICH EQUATION:

H+ = 24 x PCO2/$HCO3^-$

Reflects how the acidity of blood is determined by the relative availability of acid and alkali, i.e. $HCO3^-$, PaCO2. Stresses how H+ ion concentration is determined by the ratio of PCO2/HCO3, rather than the absolute value of either value alone.

Remember: Metabolic Acidosis/Alkalosis = disturbances of blood bicarbonate Respiratory Acidosis/Alkalosis = disturbances of PaCO2

## METABOLIC ACIDOSIS

Anion Gap: Na+ - ($Cl^-$ + $HCO3^-$) (represents unmeasured anions in plasma, normally 10-12 mmol/L)

**Figure 1**

| Elevated AG | Decreased AG | Normal AG *(hyperchloremic)* |
|---|---|---|
| Ethylene glycol | Hypercalcemia | Diarrhea |
| Lactic acid | Hypermagnesemia | RTA |
| Methanol | Hyperkalemia | Acetazolamide |
| Paraldehyde | Hypoalbuminemia | Diversions (ureteral) |
| ASA | Paraproteinemia | Renal failure |
| Renal failure | Lithium toxicity | HCL administration |
| Ketoacidosis | | |

## COMPENSATION:

Winter's formula: PaCO2 = 1.5 x $HCO3^-$ + 8 (+/-2)
(PaCO2 = last 2 digits of pH - chronic metabolic acidosis)

## TREATMENT:

Should be directed at the underlying cause Bicarbonate therapy can be considered with severe acidosis with physiologic compromise:

Bicarbonate deficit (mEq) = LBW x 0.5 x (Desired $HCO3^-$ – actual $HCO3^-$)

## OSMOLAL GAP:

Measured OSM – Calculated OSM

## CALCULATED OSMOLALITY:

2 x Na + Glc/18 + BUN/2.8 + ETOH/4.6

Elevated OG (> 10 mOsm/L):

Methanol
Ethylene glycol
Paraldehyde
ETOH ketoacidosis
Isopropyl alcohol
Mannitol

## METABOLIC ALKALOSIS

### Figure 2

| $Cl^-$ responsive (Ucl <20) | | $Cl^-$ resistant (Ucl > 30) |
|---|---|---|
| GI: | NG suction | Primary mineralocorticoid excess: |
| | Vomiting/diarrhea | Primary aldosteronism |
| | Laxative abuse | Cushing's syndrome |
| | Villous adenoma | Licorice |
| Renal: | Diuretics | Alkali load: |
| | Post hypercapnea | Citrate (transfusions) |
| | Refeeding alkalosis | Acetate (TPN) |
| Cystic fibrosis (sweat losses) | | Bartter's syndrome |
| | | Severe hypokalemia/-magnesemia |

### COMPENSATION:

$PaCO2 = 0.9 \times HCO3^- + 9$

### TREATMENT:

Acetazolamide (Diamox): 250 -375 mg po qd-bid
HCl infusion: 0.1-0.2 N @ < 0.2 mEq/hour via central line (=100-200 mEq H+/L)

HCL (mmol) = (LBW x 0.5) x Actual HCO3- desired HCO3)
Hemodialysis: severe alkalosis with cardiac/renal dysfunction

## RESPIRATORY ACIDOSIS

### CNS:

Sedatives, morphine, anesthetics
Trauma, Stroke
Infection

### NM DISORDERS:

Myopathies (MD, K+ depletion)
Neuropathies (GB, polio)

### ACUTE-CHRONIC LUNG DISEASE

COPD
PNA, pulmonary edema
ARDS
Acute obstruction (aspiration, tumor, spasm)
Obesity
Pneumothorax
Pleural effusion
Kyphoscoliosis
Scleroderma
Crush injury
Mechanical ventilation
Cardiopulmonary arrest

### COMPENSATION:

Acute: $HCO3^-$ increases by 1 mmol/L for each 10 mm Hg increase in PaCO2
Chronic: $HCO3^-$ increases by 4 mmol/L for each 10 mm Hg increase in PaCO2

## RESPIRATORY ALKALOSIS

- Anxiety, Pain
- CNS Disorders (CVA, tumor, infection)
- Lung Disease:
    - Restrictive disorders
    - Pulmonary embolus
    - PNA
- Sepsis, fever
- Hyperthryoidism
- Hypoxia
- Hepatic insufficiency
- Pregnancy
- Salicylates, Catecholamines
- Mechanical ventilation

### COMPENSATION:

Acute: $HCO3^-$ decreases by 2 mmol/L for each 10 mm Hg decrease in PaCO2
Chronic: $HCO3^-$ decreases by 5-7 mmol/L for each 10 mm Hg decrease in PaCO2

### DELTA GAP:

Identifies triple acid base disorders

**Figure 3**

| Δ gap = Δ AG = Δ HCO3 | | |
|---|---|---|
| ΔAG/ΔHCO3: | < 1 | >1 |
| | Non AG acidosis | Metabolic alkalosis + AG acidosis |
| | DKA | Lactic acidosis |
| | Chronic renal failure | |

## SUMMARY OF ACID BASE COMPENSATORY RESPONSES

**Figure 4**

| Primary disorder | Primary | | Expected | Response |
|---|---|---|---|---|
| Metabolic Acidosis | ↓HCO3- | ↓PaCO2 | PCO2 = 1.5 x HCO3 + 8 (+/-2) | |
| | | | PaCO2 = last 2 digits of pH | |
| | | | PaCO2 ↓1.25mm Hg ~ 1 mmol/L ↓ HCO3 | |
| Metabolic Alkalosis | ↑HCO3⁻ | ↑PaCO2 | PaCO2 = 0.9 x HCO3 + 9 | |
| | | | PaCO2 = HCO3 + 15 | |
| | | | PaCO2 ↑6 mm Hg ~ 10 mmol/L ↑ HCO3 | |
| Respiratory Acidosis | ↑PaCO2 | ↓HCO3⁻ | Acute: | |
| | | | HCO3 ↑1 mmol/L ~ 10 mm Hg ↑ PaCO2 | |
| | | | Chronic: | |
| | | | HCO3 ↑4 mmol/L ~ 10 mm Hg ↑ PaCO2 | |
| Respiratory Alkalosis | ↓PaCO2 | ↑HCO3⁻ | Acute: | |
| | | | HCO3⁻ ↓2 mmol/L ~ 10 mm Hg ↓ PaCO2 | |
| | | | Chronic: | |
| | | | HCO3⁻ ↓5 mmol/L ~ 10 mm Hg ↓ PaCO2 | |

## References

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