Quick Review: Acid Base Disorders

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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:

$PaCO_2 = 0.9 \times HCO_3^- + 9$

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: HCO_3^- increases by 1 mmol/L for each 10 mm Hg increase in $PaCO_2$
Chronic: HCO_3^- increases by 4 mmol/L for each 10 mm Hg increase in $PaCO_2$

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: HCO_3^- decreases by 2 mmol/L for each 10 mm Hg decrease in $PaCO_2$
Chronic: HCO_3^- decreases by 5-7 mmol/L for each 10 mm Hg decrease in $PaCO_2$

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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