(Renal Physiology 11)

Acid-Base Balance 3

Acid Base Disorders

Dr. Mona Soliman, MBBS, MSc, PhD
Head, Medical Education Department
Associate Professor of Physiology
Chair of Cardiovascular Block
College of Medicine
King Saud University

Dr.Ahmad Ahmeda

aahmeda@ksu.edu.sa

Learning Objectives:

- To explain the principles of blood gas and acid-base analysis
- To interpret blood gas analysis and diagnose various acid base disorders
- Describe causes of acid base disorders
- Understand use of acid base nomograms

Disturbances of Acid-Base Balance

Disturbances of Acid-Base Balance

- Acid-base disturbances may be either RESPIRATORY or METABOLIC.

- pH problems due to a respiratory disorder result in **RESPIRATORY** acidosis or alkalosis.
  - pH problems arising from acids or bases of a non-CO_2 origin result in **METABOLIC** acidosis or alkalosis.

Respiratory Acidosis

- **Respiratory Acidosis**
  - Associated with **RESPIRATORY FAILURE** (*e.g.* COPDs like emphysema).
    - Inadequate alveolar ventilation
    - Impaired gas diffusion (*e.g.* pulmonary oedema)
  - **Characterised by ↑ Pco_2 (hypercapnia) and ↓ plasma pH.**

- Initial response is increased conversion of CO_2 to H^+ and HCO_3^-.
  - **INCREASE** in ECF [H^+] **and** plasma [HCO_3^-].

- **INCREASED** i) renal **SECRETION OF H^+** and ii) **ABSORPTION OF HCO_3^-** is **COMPENSATORY MECHANISM**

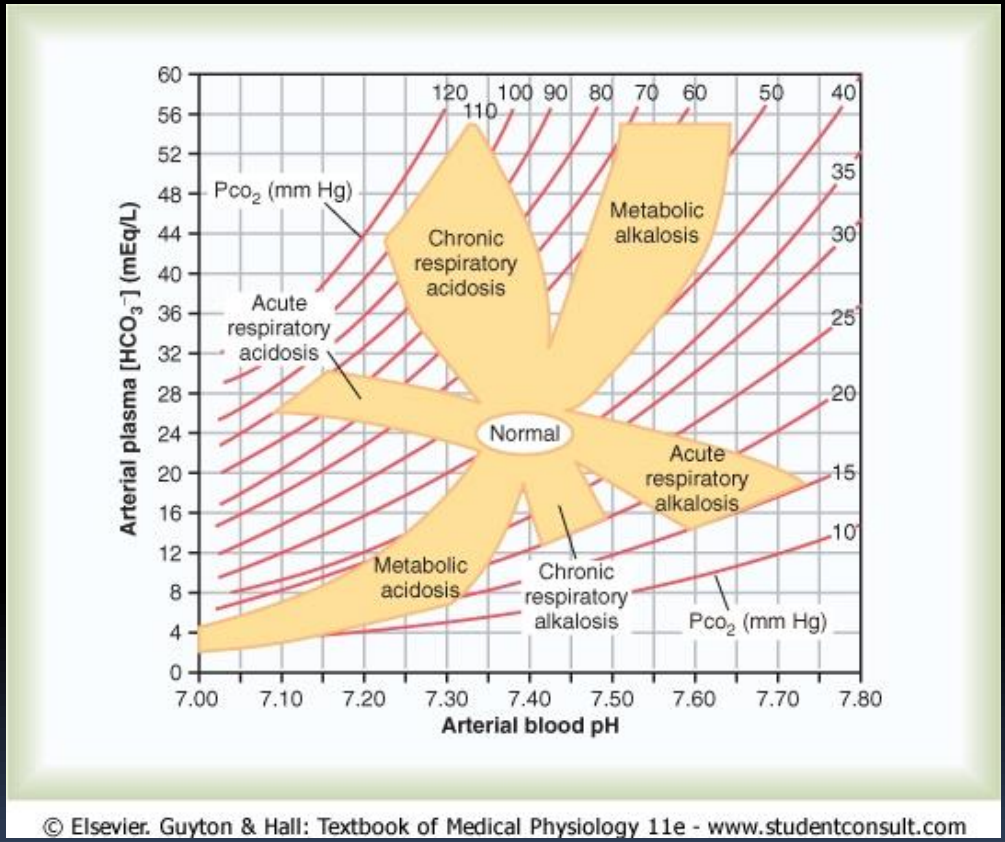
  - ACUTELY ,1 mEq/L [HCO_3^-] per 10 mm Hg ↑ in Pco_2 (tissue buffering)

  - CHRONICALLY, 3.5 mEq/L [HCO_3^-] per 10 mm Hg ↑ in Pco_2(renal acid excretion).

Davenport Diagram
Acid-base alterations

Respiratory Acidosis

↓ plasma pH, ↑ Pco_2, ↑ plasma [HCO_3^-]



Respiratory Alkalosis

- **Respiratory Alkalosis**
  - Reduced plasma Pco_2 (hypocapnia) and elevated pH
  - Caused by increased gas exchange mainly due to **HYPERVENTILATION**
    - Anxiety / fear
    - High altitude

- Characterised by ↓ Pco_2 and ↑ plasma pH.

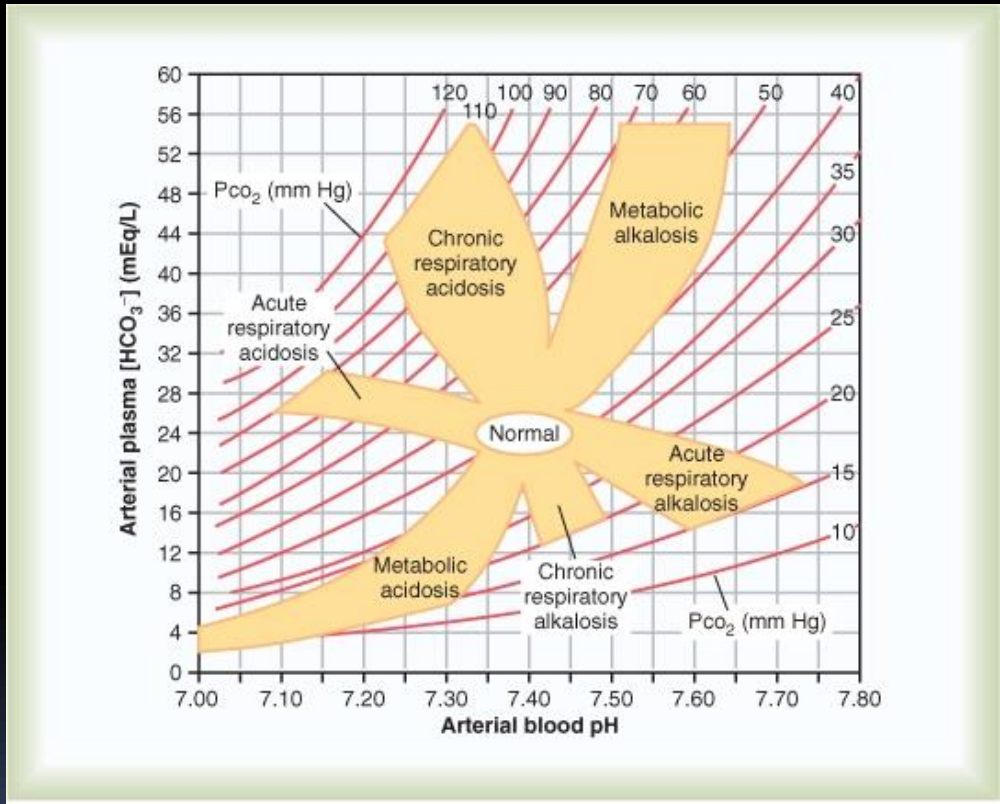
- Reduction in Pco_2 shifts buffering reaction to the left
  - **DECREASE** in ECF $[H^+]$ and plasma $[HCO_3^-]$
- **DECREASED** i) renal **SECRETION** of H^+ and ii) **ABSORPTION** of HCO_3^- (still an excess of HCO_3^- relative to H^+) is **COMPENSATORY MECHANISM.**

(see G &H, p.396-7).

Davenport Diagram
Acid-base alterations

Respiratory Alkalosis

↑ plasma pH,↓ Pco_2,,
↓ plasma [HCO_3^-]



Metabolic Acidosis & Alkalosis

- Metabolic acidosis and alkalosis includes all situations other than those in which primary problem is respiratory.

- By definition, metabolic acidosis and alkalosis cannot be due to excess retention or loss of CO_2
  - does arterial Pco_2 remain unchanged in these cases?

NO!

- ↑ $[H^+]$ in acidosis will reflexly stimulate ventilation to lower Pco_2. Conversely, ventilation will be inhibited in alkalosis to restore $[H^+]$.

- Remember, plasma Pco_2 changes during metabolic acidosis / alkalosis are a *result* of, not *cause* of, compensatory reflex responses to non-respiratory abnormalities.

Metabolic Acidosis

- Caused by either i) **INCREASED** acid ***production*** or

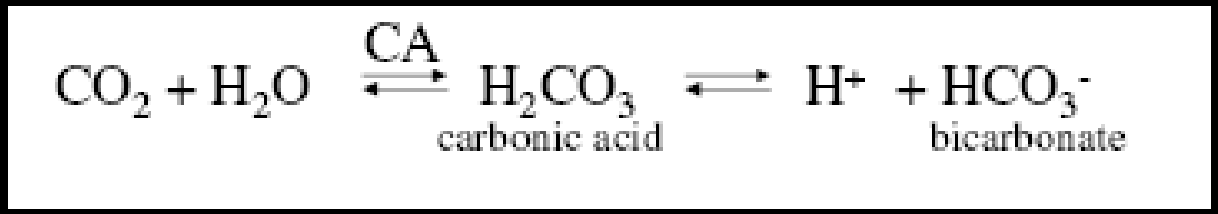
  ii) **IMPAIRED** acid ***excretion***.

Can occur in response to;

1) High protein diet - protein catabolism produces phosphoric acid and sulphuric acid.
2) High fat diet - fat catabolism produces fatty acids.
3) Heavy exercise – stimulates anaerobic metabolism, producing lactic acid.
4) Addition of fixed acids (*e.g.* diabetic ketoacidosis).
5) Severe diarrhoea – loss of bicarbonate from intestines.
6) Alterations in renal function (inability to excrete H^+).
7) Tissue hypoxia (produces lactic acid)
8) Ingested substances such as methanol, aspirin (acetylsalicylic acid), ethylene glycol.

Metabolic Acidosis

- First line of defence is shift of buffering reactions to the left to neutralise excess acid (for bicarbonate, cell* & bone buffers).

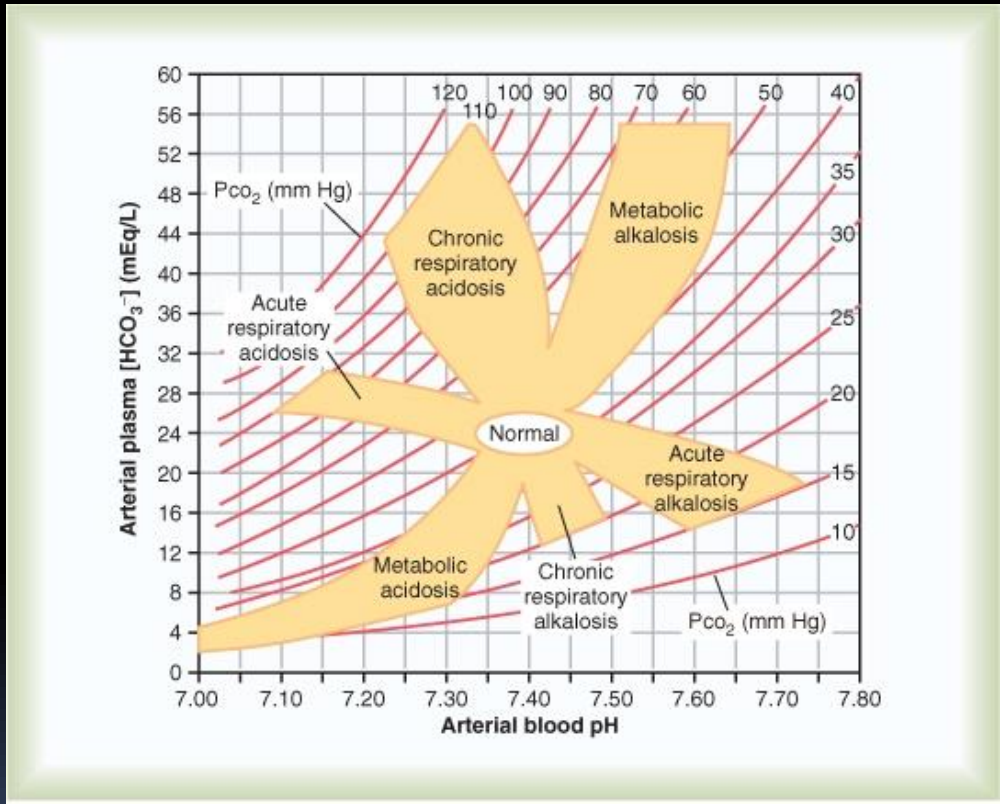


- Metabolic acidosis increases ventilation rate *via* chemoreceptor activation.
- Increased expiration of CO_2 reduces Pco_2 levels which increases pH of ECF (respiratory compensation).
- Acidosis **INCREASES** renal **SECRETION** of H^+ and **ABSORPTION** of HCO_3^- $\Rightarrow$ ECF $[HCO_3^-]$ increases.
- **Characterised by DECREASED $[HCO_3^-]$ (<25mM) and pH.**

Davenport Diagram
Acid-base alterations

Metabolic Acidosis

↓ plasma pH,
↓ plasma [HCO_3^-]
↓ Pco_2,



Metabolic Acidosis

* Uptake of excess H^+ by cells is accompanied, in part, by **LOSS** of ***intra***cellular K^+ (and Na^+) to ***extra***cellular fluid to maintain **ELECTRONEUTRALITY**.

- Thus, metabolic acidosis often associated with **INCREASED plasma [K^+]** relative to that expected from state of potassium balance.
- **HYPERKALEMIA** can develop even though body K^+ stores are diminished.
- Cation shift is **REVERSED** with correction of acidosis.

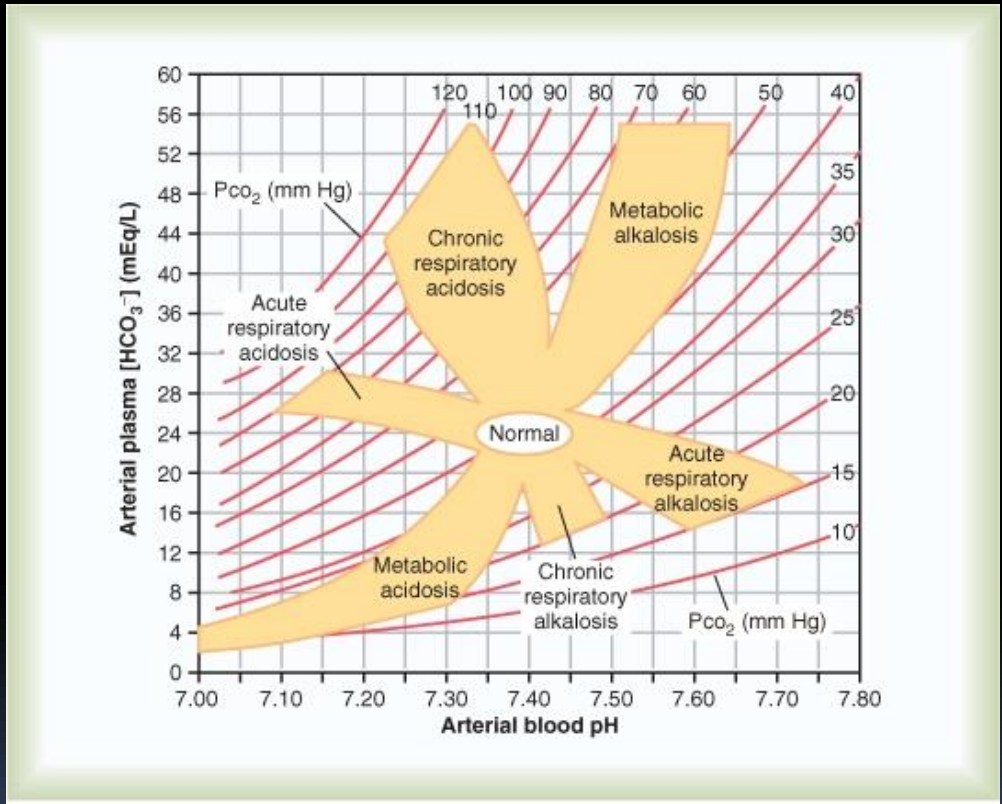
Metabolic Alkalosis

- Relatively rare phenomenon that can occur in response to;
  1) Excessive vomiting – loss of HCl from stomach ($\therefore$ retention of (duodenal) bicarbonate in circulation).
  2) Alterations in renal function ($\uparrow$ excretion of titratable acid *e.g.*, thiazide and loop diuretics $\uparrow Na^+$ reabsorption $\rightarrow \uparrow$excretion of H^+).
  3) Excessive ingestion of bicarbonate antacids paired with renal failure.
  4) Volume contraction (*e.g via* diuretic therapy $\uparrow$ plasma $[HCO_3^-]$).
  5) Excess aldosterone (stimulates collecting duct H^+-ATPases to excrete H^+) .
- Loss of acid $\uparrow$ dissociation of $H_2CO_3 \Rightarrow \uparrow HCO_3^-$.
- Increase in pH **REDUCES** ventilation rate, elevating Pco_2 levels.
- Reduction in renal absorption and $\uparrow$ excretion of HCO_3^- in the nephron.
- **Characterised by ELEVATED plasma $[HCO_3^-]$ and pH.**

Davenport Diagram
Acid-base alterations

Metabolic Alkalosis

–plasma pH, ↑
↑ plasma [HCO_3^-] ,↑ Pco_2,



Acid-Base Imbalances

(Summary)

- Acid-base balance can be altered in one of four ways:
  i) respiratory acidosis or alkalosis
  ii) metabolic acidosis or alkalosis

- **RESPIRATORY ACIDOSIS** caused by ↓ elimination of CO_2, secondary to either ↓ respiration or gas exchange.

- **RESPIRATORY ALKALOSIS** caused by ↑ elimination of CO_2 (by hyperventilation).

- **METABOLIC ACIDOSIS** caused by ↑ H^+ production / intake or ↓[bicarbonate].

- **METABOLIC ALKALOSIS** caused by ↓ H^+ production, ↑ elimination of H^+ or ↑ in [bicarbonate].

Acid / Base Disorders
Summary

Table 28-3. The Four Major Acid-Base Disorders

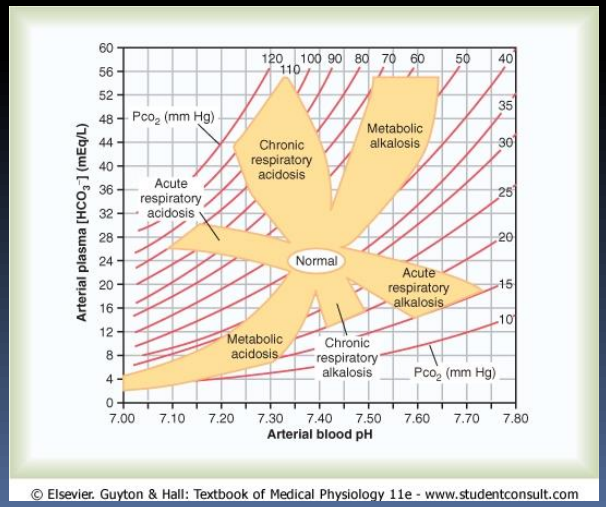
Disorder	Proximate Causes	Clinical Causes	Changes in Arterial Acid-Base Parameters
Respiratory acidosis	Increased P_{CO_2}	Decreased alveolar ventilation (e.g., drug overdose) ↓ Lung diffusing capacity (e.g., pulmonary edema) Ventilation-perfusion mismatch	pH ↓ [HCO_3^-] ↑ P_{CO_2} ↑
Respiratory alkalosis	Decreased P_{CO_2}	Increased alveolar ventilation caused by: Hypoxia (e.g., acclimatization to high altitude) Anxiety Aspirin intoxication	pH ↑ [HCO_3^-] ↓ P_{CO_2} ↓
Metabolic acidosis	Addition of acids other than CO_2 or H_2CO_3 Removal of alkali (fixed P_{CO_2})	↓Urinary secretion of H^+ (e.g., renal failure) Ketoacidosis (e.g., diabetes mellitus) Lactic acidosis (e.g., shock) HCO_3^- loss (e.g., severe diarrhea)	pH ↓ [HCO_3^-] ↓ P_{CO_2}: no change
Metabolic alkalosis	Addition of alkali Removal of acids other than CO_2 or H_2CO_3 (fixed P_{CO_2})	HCO_3^- load (e.g., $NaHCO_3$ therapy) Loss of H^+ (e.g., severe vomiting)	pH ↑ [HCO_3^-] ↑ P_{CO_2}: no change

Analysis of Acid-Base Disorders

- Analysis aimed at identifying underlying cause of disorder such that appropriate therapy can be initiated.

- In addition to usual history taking and physical findings, sampling of arterial blood can yield valuable information.

- Analysis of blood sample data is straightforward if approached systematically either using the Davenport nomogram or flow diagram.



How to Analyze an ABG

1. PO_2 NL = 80 – 100 mmHg

2. pH NL = 7.35 – 7.45
   Acidotic <7.35
   Alkalotic >7.45

3. PCO_2 NL = 35 – 45 mmHg
   Acidotic >45
   Alkalotic <35

4. HCO_3 NL = 22 – 26 mmol/L
   Acidotic < 22
   Alkalotic > 26

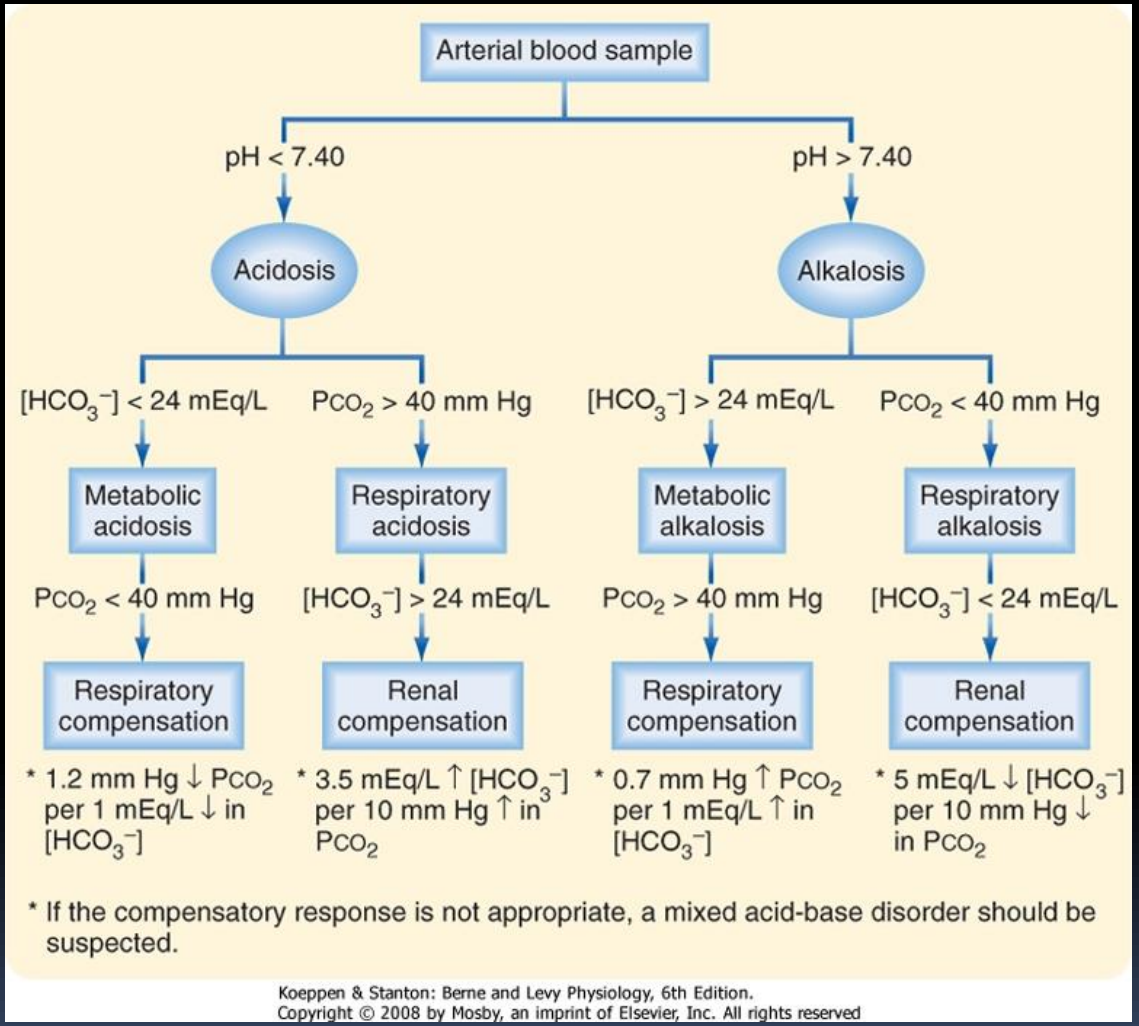
Analysis of Acid-Base Disorders

e.g. pH = 7.35
$[HCO_3^-]$ = 16mEq/L
PCO2 = 30 mm Hg

1) Evaluate pH - acid

2) Metabolic or respiratory source?
$[HCO_3^-]$ < 24mM = metabolic

3) Analysis of compensatory response.
↓ PCO_2 – respiratory compensation



- Mixed acid-base disorders can also occur (*e.g.* emphysema with diarrhea) in which an appropriate compensatory response has not occurred.

1) A 50 year-old man with history of type 2 diabetes was admitted to the emergency department with history of polyuria. On examination he had rapid and deep breathing. Blood analysis showed glucose level of 400 mg/dl.

- **The following is the arterial blood analysis report of this patient:**
- **pH = 7.1, PCO^2 = 40 mmHg and HCO^{3-} = 18 mmol/L**
- **(Normal reference ranges: PCO2 = 36.0-46.0 mmHg, HCO3- = 22.0-26.0 mmol/L)**

- **What is the acid base disturbance in this case?**

2) PH= 7.12, $PaCO_2$= 60mmHg, HCO_3^- = 24meq/L.

a) Compensated metabolic acidosis.
b) Uncompensated metabolic acidosis,
c) Compensated respiratory acidosis,
d) Uncompensated respiratory acidosis,

3) **PH= 7.51, $PaCO_2$= 40mmHg, HCO_3^- = 31meq/L.**

a) **Normal,**
b) **Compensated respiratory acidosis,**
c) **Uncompensated respiratory alkalosis.**
d) **Uncompensated metabolic alkalosis,**

74 y-o ♀ with hx chronic renal failure and chronic diuretic therapy was admitted to ICU comatose and severely dehydrated. On 40% oxygen her ABG & Vital Sign:

pH	7.52	BP	130/90 mmHg
PCO_2	55 mmHg	Pulse	120/min
PO_2	92 mmHg	RR	25/min
HCO_3	42 mmol/L		

Interpretation: Partly compensated metabolic alkalosis.


Thanks

Table 5.2 Primary and Compensatory Changes in Different Acid–Base Disorders

Disorder	Primary Change	Compensatory Response
Metabolic acidosis	Fall in plasma bicarbonate concentration	Reduction in PCO_2 averaging 1.2 mm Hg per 1-mEq/L reduction in plasma bicarbonate concentration
Metabolic alkalosis	Rise in plasma bicarbonate concentration	Elevation in PCO_2 averaging 0.6–0.7 mm Hg per 1-mEq/L rise in plasma bicarbonate concentration
Respiratory acidosis	Elevation in PCO_2	**Acute**: Rise in plasma bicarbonate concentration averaging 1 mEq/L per 10 mm Hg elevation in PCO_2 **Chronic**: Increase in plasma bicarbonate concentration averaging 3.5 mEq/L per 10 mm Hg rise in PCO_2
Respiratory alkalosis	Reduction in PCO_2	**Acute**: Fall in plasma bicarbonate concentration averaging 2 mEq/L per 10 mm Hg decline in PCO_2 **Chronic**: Fall in plasma bicarbonate concentration averaging 4 mEq/L per 10 mm Hg decline in PCO_2

Major Causes of Metabolic Acidosis

Increased acid production

A. **Lactic acidosis**
B. **Ketoacidosis, most often due to uncontrolled diabetes mellitus**
C. **Ingestions**
    1. Aspirin
    2. Ethylene glycol, a component of antifreeze and solvents
    3. Methanol (wood alcohol), a component of shellac and de-icing solutions
D. **Loss of bicarbonate**
    1. Gastrointestinal—diarrhea, pancreatic, biliary or intestinal fistulas, ureterosigmoidostomy
    2. Renal—type 2 (proximal) renal tubular acidosis

Decreased acid excretion

A. **Renal failure—decreased NH_4^+ excretion**
B. **Type 1 (distal) renal tubular acidosis**
C. **Type 4 renal tubular acidosis (hypoaldosteronism)**

Major Causes of Metabolic Alkalosis

I. **Hydrogen loss**
   A. **Gastrointestinal loss**
      1. Removal of gastric secretions due to vomiting or nasogastric suction
      2. Antacids in advanced renal failure
   B. **Urinary loss**
      1. Loop or thiazide-type diuretics
      2. Primary mineralocorticoid excess (hyperaldosteronism)
      3. Posthypercapnic alkalosis
      4. Hypercalcemia and milk alkali syndrome
   C. **Movement of H^+ into the cells**
      1. Hypokalemia

II. **Administration of bicarbonate or an organic ion that can be metabolized to bicarbonate, such as citrate in blood transfusions**

III. **Contraction alkalosis**
   A. **Loop or thiazide-type diuretics in edematous patients**
   B. **Vomiting or nasogastric suction in achlorhydria**
   C. **Sweat losses in cystic fibrosis**