ACID-BASE

BALANCE

DR.SIRILUK CHUMNANVEJ

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ACID-BASE

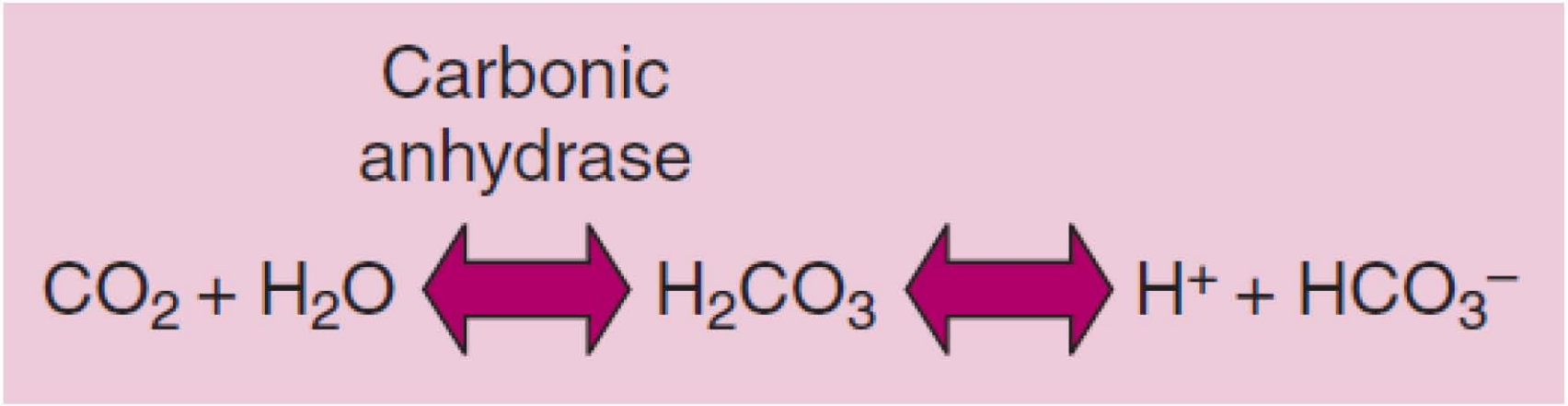
ACID-BASE

- ความสำคัญ
  - Cell function
  - Ionization
  - Pharmacokinatic & Pharmacodynamics
  - Electrolytes → K^+

1. Acid–Base Interpretation and Treatment
2. Practical Approach to Acid–Base Interpretation

Acid-Base Homeostasis

1. Buffer systems → first line of defense against the fall in pH
   1) $CO_2 + H_2O \overset{CA}{\longleftrightarrow} H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$
   2) $H_2PO_4^- \longleftrightarrow HPO_4^{2-}$
   3) Protein
   4) Hemoglobin
2. Respiratory system → within 15–30 min
3. Kidney → requires 3–5 days to complete compensation
   1) HCO_3^- reabsorption at proximal tubule
   2) Excretion H^+ ; $Na_2HPO_4 \longleftrightarrow NaH_2PO_4$, $NH_3 \longleftrightarrow NH_4^+$



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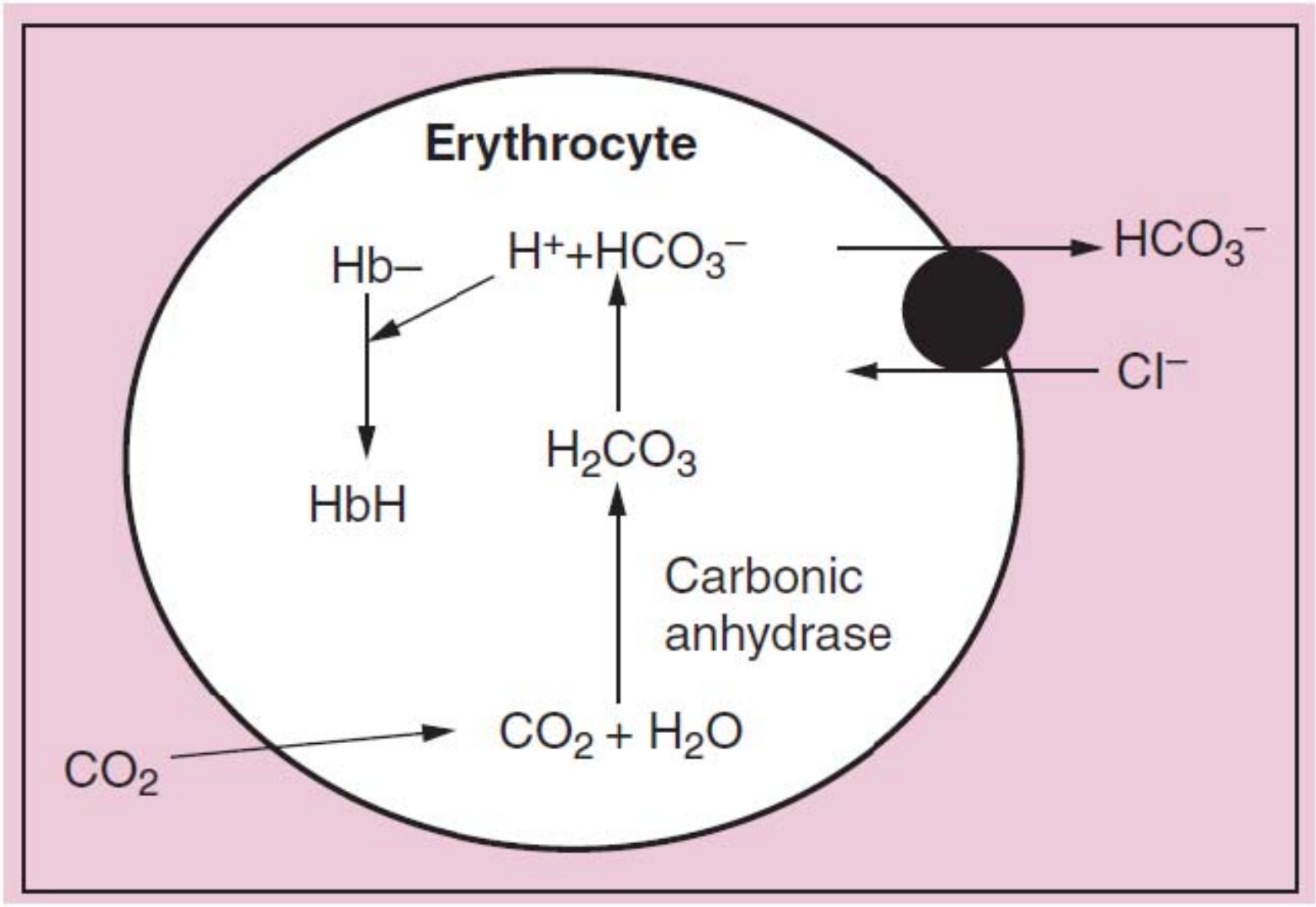
Hemoglobin buffering system



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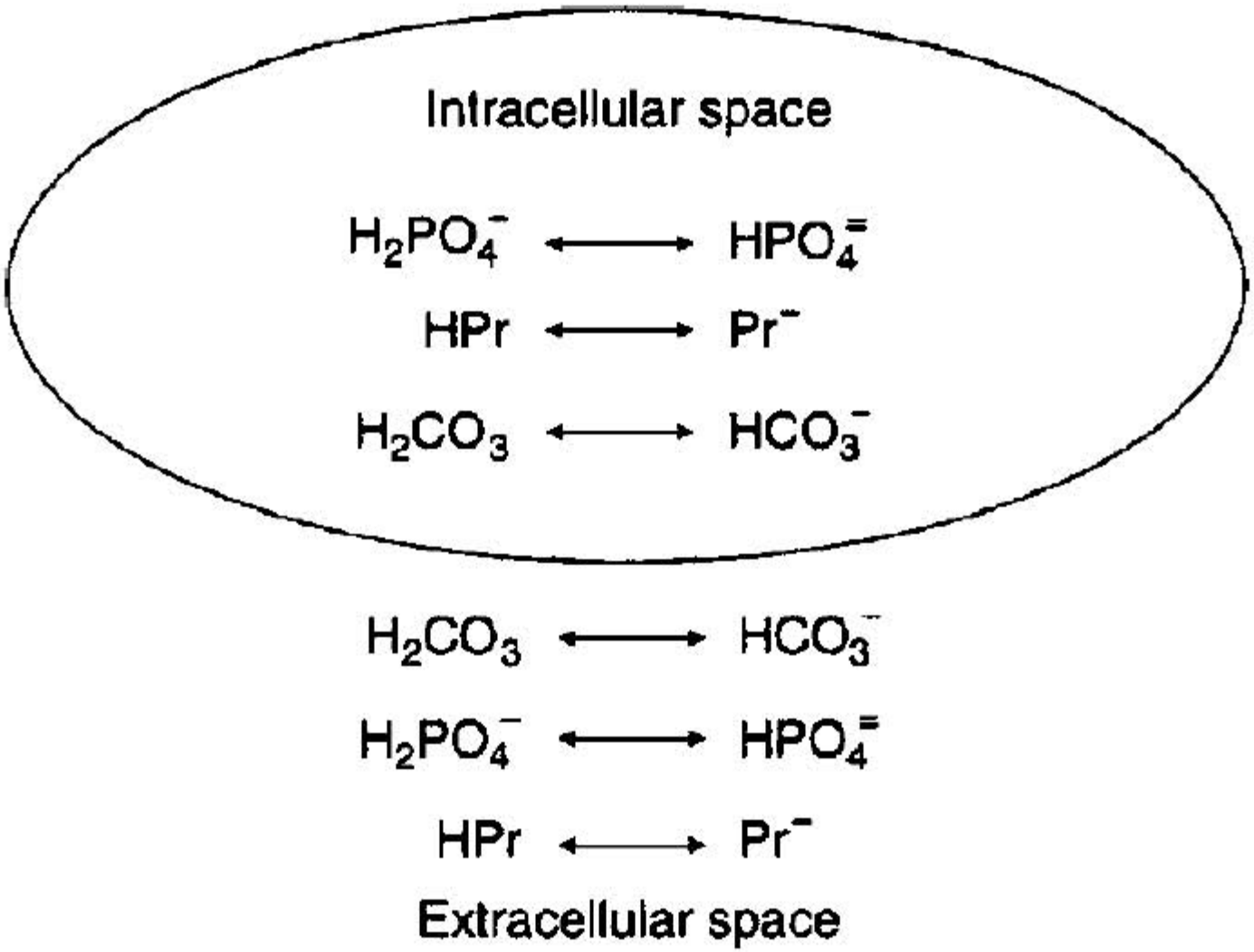
Buffering in the Intracellular



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Respiratory system

1. Central chemoreceptors
   - Locate at anterolateral surface of the medulla
   - respond to changes in cerebrospinal fluid pH
   - MV increases 1 to 4 L/min for every 1 mm Hg increase in $PaCO_2$
2. Peripheral chemoreceptors
   - Locate at bifurcation of the common carotid arteries and surrounding the aortic arch
   - most sensitive to PaO_2

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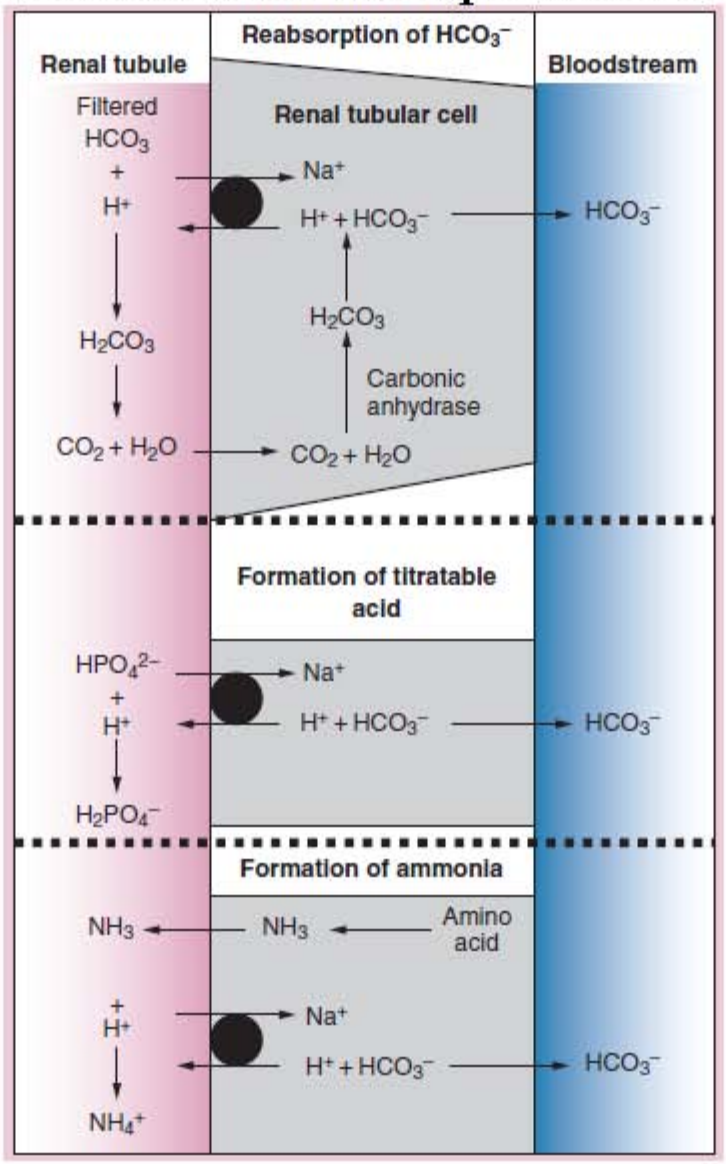
Three mechanisms of renal compensation during acidosis



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Henderson-Hasselbalch equation

$$\mathbf{pH} = 6.1 + \log_{10} \frac{[HCO_3^-]}{0.03 \times PaCO_2}$$

Henderson equation

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

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- Primary abnormalities of CO_2 tension are considered respiratory disturbances, whereas primary derangements of $[HCO_3^-]$ are metabolic disturbances

$$\mathbf{pH} = pK + \log_{10} \frac{[A^-]}{[HA]}$$

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Conversion of pH to Proton Concentration (nM/L)

pH	Proton Concentration (nM/L)
6.9	125
7.0	100
7.1	80
7.2	63
7.3	50
7.4	40
7.5	32
7.6	25

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General Principles of Acid-Base Homeostasis

- Acid-base homeostasis consists of the precise regulation of
  - CO_2 tension by the respiratory system → eliminating H^+ ~ 14,000 mEq/Day
  - plasma bicarbonate concentration [HCO_3^-] by the kidney
- The kidney regulates plasma [HCO_3^-] by
  - altering HCO_3^- reabsorption
  - eliminating protons (H^+) ~ 70 mEq/Day
- Body fluid pH is determined by CO_2 tension and [HCO_3^-]

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Serum Anion Gap (SAG)

$$SAG = [Na+] - [Cl^-] - [HCO_3^-]$$

Normally = 6 and 10 mEq/L

- Extremely elevate; SAG > 25 mEq/L) always reflects the presence of an organic acidosis
- low SAG 4 types;
  1. a reduction in the concentration of unmeasured anions (primarily albumin)
  2. increased unmeasured cations (hyperkalemia, hypermagnesemia, hypercalcemia, lithium toxicity, or a cationic paraprotein)
  3. underestimation of the serum Na+ concentration (severe hypernatremia)
  4. overestimation of the serum Cl^- concentration (bromide intoxication and marked hyperlipidemia)

each 1 g/dL decrease in serum Alb → SAG will decrease by 2.5 mEq/L

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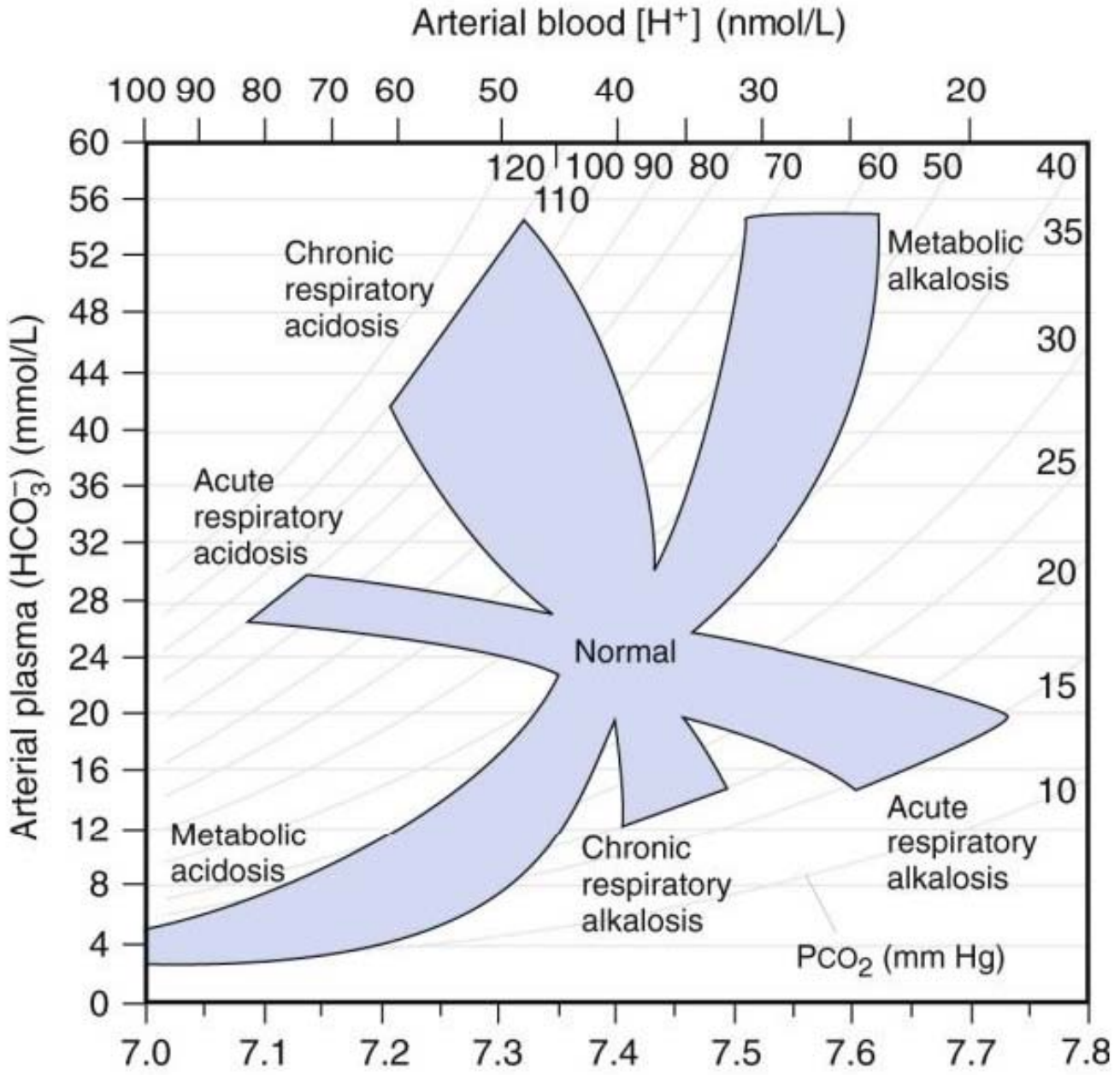

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Acid-Base Disturbances Commonly Seen Perioperatively

Disorder	Cause
Respiratory acidosis	Hypoventilation—narcosis, incomplete reversal of neuromuscular blockade
Respiratory alkalosis	Hyperventilation—anxiety, pain
Metabolic acidosis secondary to unmeasured anions (widened gap acidosis)	Hypoperfusion—lactic acidosis; diabetic ketoacidosis; renal failure
Metabolic acidosis secondary to measured anions (non-gap hyperchloremic acidosis)	Hyperchloremia—"normal" saline, hetastarch, or albumin infusions; renal tubular acidosis; bladder reconstructions
Metabolic acidosis secondary to free water excess (hyponatremia, dilution acidosis)	Hypotonic fluid administration; sodium loss—diarrhea; administration of hyperosmolar fluids—mannitol, alcohol, hyperproteinemia
Metabolic alkalosis	Hyperventilation of patient with history of carbon dioxide retention (COPD); sodium gain (sodium bicarbonate massive blood transfusion); chloride loss—nasogastric suctioning

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Causes of Respiratory Acidosis

Decreased Alveolar Ventilation

- Central nervous system depression (opioids, general anesthetics)
- Peripheral skeletal muscle weakness (neuromuscular blockers, myasthenia gravis)
- Chronic obstructive pulmonary disease
- Acute respiratory failure

Increased Carbon Dioxide Production

- Hypermetabolic states
  - Fever
  - Malignant hyperthermia
  - Sepsis
  - Multiple trauma
  - Hyperalimentation

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Causes of Respiratory Acidosis

Increased production
Malignant hyperthermia
Hyperthyroidism
Sepsis
Overfeeding
Decreased elimination
Intrinsic pulmonary disease (pneumonia, ARDS, fibrosis, edema)
Upper airway obstruction (laryngospasm, foreign body, OSA)
Lower airway obstruction (asthma, COPD)
Chest wall restriction (obesity, scoliosis, burns)
CNS depression (anesthetics, opioids, CNS lesions)
Decreased skeletal muscle strength (residual effects of neuromuscular blocking drugs, myopathy, neuropathy)
Increased rebreathing or absorption
Exhausted soda lime
Incompetent one-way valve
Laparoscopic surgery

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Causes of Respiratory Alkalosis

- Hyperventilation syndrome (diagnosis of exclusion; most often encountered in the emergency department)
- Iatrogenic hyperventilation
- Pain
- Anxiety
- Arterial hypoxemia
- Central nervous system disease
- Systemic sepsis

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Causes of Respiratory Alkalosis

Increased minute ventilation
Hypoxia (high altitude, low F_{IO_2}, severe anemia)
Iatrogenic (mechanical ventilation)
Anxiety and pain
CNS disease (tumor, infection, trauma)
Fever, sepsis
Drugs (salicylates, progesterone, doxapram)
Liver disease
Pregnancy
Restrictive lung disease
Pulmonary embolism
Decreased production
Hypothermia
Skeletal muscle paralysis

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Physiologic Effects Produced by Respiratory Alkalosis

- Hypokalemia (potentiates toxicity of digoxin)
- Hypocalcemia
- Cardiac dysrhythmias
- Bronchoconstriction
- Hypotension
- Decreased cerebral blood flow (returns to normal > 8 to 24 hrs corresponding to the return of cerebrospinal fluid pH to normal)

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Rules of Thumb for [HCO_3^-] and pH Changes in Response to Acute and Chronic Changes in $PaCO_2$

Decreased $PaCO_2$

1. pH increases 0.10 for every 10 mmHg decrease in $PaCO_2$.
2. [HCO_3^-] decreases 2 mEq/L for every 10 mmHg decrease in $PaCO_2$.
3. pH will nearly normalize if hypocarbia is sustained.
4. [HCO_3^-] will decrease 5 to 6 mEq/L for each chronic 10 mmHg ↓ in $PaCO_2$.[a]

Increased $PaCO_2$

1. pH will decrease 0.05 for every acute $PaCO_2$ increase of 10 mmHg.
2. [HCO_3^-] will increase 1.0 mEq/L for every $PaCO_2$ increase of 10 mmHg.
3. pH will return toward normal if hypercarbia is sustained.
4. [HCO_3^-] will increase 4 to 5 mEq/L for each chronic 10 mmHg increase in $PaCO_2$.

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Changes in Paco2 and [HCO3-] in Response to Acute and Chronic Acid-Base Disturbances

Disturbances	$[HCO_3^-]$ versus $Paco_2$
Acute respiratory acidosis	$\Delta HCO_3^- = 0.2\ \Delta Paco_2$
Acute respiratory alkalosis	$\Delta HCO_3^- = 0.2\ \Delta Paco_2$
Chronic respiratory acidosis	$\Delta HCO_3^- = 0.5\ \Delta Paco_2$
Metabolic acidosis	$\Delta Paco_2 = 1.3\ \Delta HCO_3^-$
Metabolic alkalosis	$\Delta Paco_2 = 0.75\ \Delta HCO_3^-$

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Determining Whether Respiratory Process Is Acute or Chronic

Acute Process

pH △ 0.08 for every 10 mm Hg △ in Pco_2 from 40 mm Hg

Chronic Process

pH △ 0.03 for every 10 mm Hg △ in Pco_2 from 40 mm Hg

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Physiologic Effects Produced by Metabolic Acidosis

- Decreased myocardial contractility
- Increased pulmonary vascular resistance
- Decreased systemic vascular resistance
- Impaired response of the cardiovascular system to endogenous and exogenous catecholamines
- Compensatory hyperventilation

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Causes of Metabolic Acidosis

Anion Gap Acidosis

M methanol, ethylene glycol
U uremia
L lactic acidosis = CHF, sepsis, cyanide toxicity
E ethanol
P paraldehyde
A aspirin, INH
K ketones = starvation, diabetic

Nongap Acidosis

Administration of 0.9% NaCl
GI losses—diarrhea, ileostomy, neobladder, pancreatic fistula
Renal losses—RTA
Drugs—acetazolamide

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Differential Diagnosis of Metabolic Acidosis

·ELEVATED ANION GAP	·NORMAL ANION GAP
Three diseases	
1. Uremia 2. Ketoacidosis 3. Lactic acidosis	1. Renal tubular acidosis 2. Diarrhea 3. Carbonic anhydrase inhibition 4. Ureteral diversions 5. Early renal failure 6. Hydronephrosis 7. HCl administration 8. Saline administration
Toxins	
1. Methanol 2. Ethylene glycol 3. Salicylates 4. Paraldehyde	

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Evaluation of a Patient with Metabolic Acidosis

1. Is acidosis being caused by measured or unmeasured anions (chloride)?
   - Look at blood chemistry

Calculate anion gap: Na + K - Cl = 10-12

If gap is normal → excess chloride , excess loss of sodium (diarrhea, ileostomy), renal tubular acidosis

If gap is wide (>16) → unmeasured anions causing acidosis

- Check serum lactate

if >2 probably lactic acidosis ; circulatory insufficiency (shock, hypovolemia, oliguria, under-resuscitation, anemia, carbon monoxide poisoning, seizures) → "type A" lactic acidosis

If not → "type B (rare)" ; biguanides, fructose, sorbitol, nitroprusside, ethylene glycol, cancer, liver disease

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Evaluation of a Patient with Metabolic Acidosis(cont.)

- Look at creatinine & urine output

If patient is in acute renal failure → renal acids

- Look at blood glucose & urinary ketones

If patient is hyperglycemic & ketotic → diabetic ketoacidosis

If patient is ketotic (unmeasured anion) & normoglycemic → alcoholic or starvation ketosis

Check for presence of chronic alcohol abuse—high mean corpuscular volume, increased γ-glutamyl transferase on liver panel

2. If all of these tests are negative, think of intoxication

   – Send toxicology laboratory tests (particularly salicylates) and serum osmolality → calculate osmolality :

$$2(\mathrm{Na} + \mathrm{K}) + \frac{\mathrm{Glucose}}{18} + \frac{\mathrm{BUN}}{2.8}$$

   – Look for unmeasured source of osmoles

if gap between measured and calculated serum osmolality >12, think of alcohol, particularly ethylene glycol, isopropyl alcohol, and methanol

Causes of Increased Anion Gap (Organic) Metabolic Acidosis

Increased acid production

- Lactic acidosis
- Ketoacidosis ; Diabetic ketoacidosis, Starvation, Alcoholic ketoacidosis
- Toxic alcohol ingestion
- Salicylate overdose
- Pyroglutamic acidosis
- Other intoxications (e.g., toluene, isoniazid and propylene glycol)
- Inborn errors of metabolism

Failure of acid excretion

- Acute kidney injury
- Chronic kidney disease

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Anesthetic Implications of Metabolic Acidosis

- Monitor arterial blood gases and pH
- Possible exaggerated hypotensive responses to drugs and PPV of the patient's lungs (reflects hypovolemia)
- Consider monitoring with an intra-arterial catheter and pulmonary artery catheter
- Maintain previous degree of compensatory hyperventilation

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Calculation of Sodium Bicarbonate Dose

$$\text{Sodium Bicarbonate (mEq/L)} = \frac{\text{weight (kg)} \times 0.3 \times (24\ \text{mEq/L [actual bicarbonate]})}{2}$$

infants & children → initial dose = 1.0 to 2.0 mEq/kg of body weight

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Generation of Metabolic Alkalosis

·GENERATION	·EXAMPLE	·MAINTENANCE
I. Loss of acid from extracellular space		
A. Loss of gastric fluid	Vomiting; nasogastric drainage	↓ effective arterial volume (EAV)
B. Loss of acid into urine; continued Na^+ delivery to the distal tubule in presence of hyperaldosteronism	1. Primary aldosteronism 2. Diuretic administration	1. K^+ depletion + aldosterone excess 2. ↓ EAV + K^+ depletion

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·GENERATION	·EXAMPLE	·MAINTENANCE
II. Excessive HCO_3^- loads		
A. Absolute	$NaHCO_3$ administration	↓EAV
1. HCO_3^-	Lactate, acetate, citrate administration	↓EAV
2. Metabolic conversion of salts of organic acid anions to HCO_3^-	Alkali administration to patients with renal failure	Renal failure
B. Relative		
1. Alkaline loads in renal failure		
III. Posthypercapnic state	Abrupt correction of chronic hypercapnia	↓EAV

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Causes of Metabolic Alkalosis

Chloride Responsive

Renal loss—diuretic therapy

GI loss—vomiting, NG suction

Alkali administration—citrate in blood products, acetate in TPN, bicarbonate

Chloride Resistant

Hyperaldosteronism

Refeeding syndrome

Profound hypokalemia

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Rules of Thumb for Respiratory Compensation in Response to Metabolic Alkalosis and Metabolic Acidosis

Metabolic alkalosis

1. $PaCO_2$ increases approximately 0.5 to 0.6 mmHg for each 1.0 mEq/L increase in $[HCO^-_3]$
2. The last two digits of the pH should equal the $[HCO^-_3] + 15$

Metabolic acidosis

1. $PaCO_2 = [HCO^-_3] \times 1.5 + 8$
2. $PaCO_2$ decreases 1.2 mmHg for every 1.0 mEq/L in $[HCO^-_3]$ to a minimum of 10 to 15 mmHg
3. The last two digits of the pH equal $[HCO^-_3] + 15$

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Factors that Maintain Metabolic Alkalosis

Factor	Proposed Mechanism
Decreased GFR	Increases fractional bicarbonate reabsorption and prevents elevated plasma bicarbonate concentrations from exceeding Tm
Volume contraction	Stimulates proximal tubular bicarbonate reabsorption
Hypokalemia	Decreases GFR and increases proximal tubular bicarbonate reabsorption Stimulates sodium-independent/potassium-dependent (low) secretion in cortical collecting tubules
Hypochloremia*	Increases renin Decreases distal chloride delivery
Passive backflux of bicarbonate	Creates a favorable concentration gradient for passive bicarbonate movement from proximal tubular lumen to blood
Aldosterone	Increases sodium-dependent proton secretion in cortical collecting tubules and sodium-independent proton secretion in cortical collecting tubules and medullary collecting tubules

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Physiologic Effects Produced by Metabolic Alkalosis

- Hypokalemia (potentiates effects of digoxin; evokes ventricular cardiac dysrhythmias)
- Decreased serum ionized calcium concentration
- Compensatory hypoventilation (may be exaggerated in patients with COPD or those who have received opioids; compensatory hypoventilation rarely results in $PaCO_2$ >55 mm Hg)
- Arterial hypoxemia (reflects effect of compensatory hypoventilation)
- Increased bronchial tone (may contribute to atelectasis)
- Leftward shift of oxyhemoglobin dissociation curve (oxygen less available to tissues)
- Decreased cardiac output
- Cardiovascular depression and cardiac dysrhythmias (result of inadvertent iatrogenic respiratory alkalosis to pre-existing metabolic alkalosis during anesthetic management)

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Treatment of Metabolic Alkalosis

Etiologic Therapy

- Expand intravascular fluid volume (intraoperative fluid management with 0.9% saline; lactated Ringer's solution provides an additional substrate for generation of bicarbonate).
- Administer potassium.
- Avoid iatrogenic hyperventilation of the patient's lungs.

Nonetiologic Therapy

- Administer acetazolamide (causes renal bicarbonate wasting).
- Administer hydrogen (ammonium chloride, arginine hydrochloride, hydrochloric acid [must be injected into a central vein]).

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Determining Appropriate Compensation in Acid-Base Disorders

Metabolic Acidosis

Winter's formula:

$$Pco_2 = (1.5 \times HCO_3^-) + 8$$

If measured $Pco_2 >$ calculated Pco_2, then concurrent respiratory acidosis is present.

If measured $Pco_2 <$ calculated Pco_2, then concurrent respiratory alkalosis is present.

Metabolic Alkalosis

$$Pco_2 = (0.7 \times HCO_3^-) + 21$$

If measured $Pco_2 >$ calculated Pco_2, then concurrent respiratory acidosis is present.

If measured $Pco_2 <$ calculated Pco_2, then concurrent respiratory alkalosis is present.

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Sequential Approach to Acid-Base Interpretation

1. Is the pH life threatening, requiring immediate intervention?
2. Is the pH acidemic or alkalemic?
3. Could the entire arterial blood gas picture represent only an acute increase or decrease in $PaCO_2$?
4. If the answer to question #3 is "No," is there evidence of a chronic respiratory disturbance or of an acute metabolic disturbance?
5. Are appropriate compensatory changes present?
6. Is an anion gap present?
7. Do the clinical data fit the acid-base picture?

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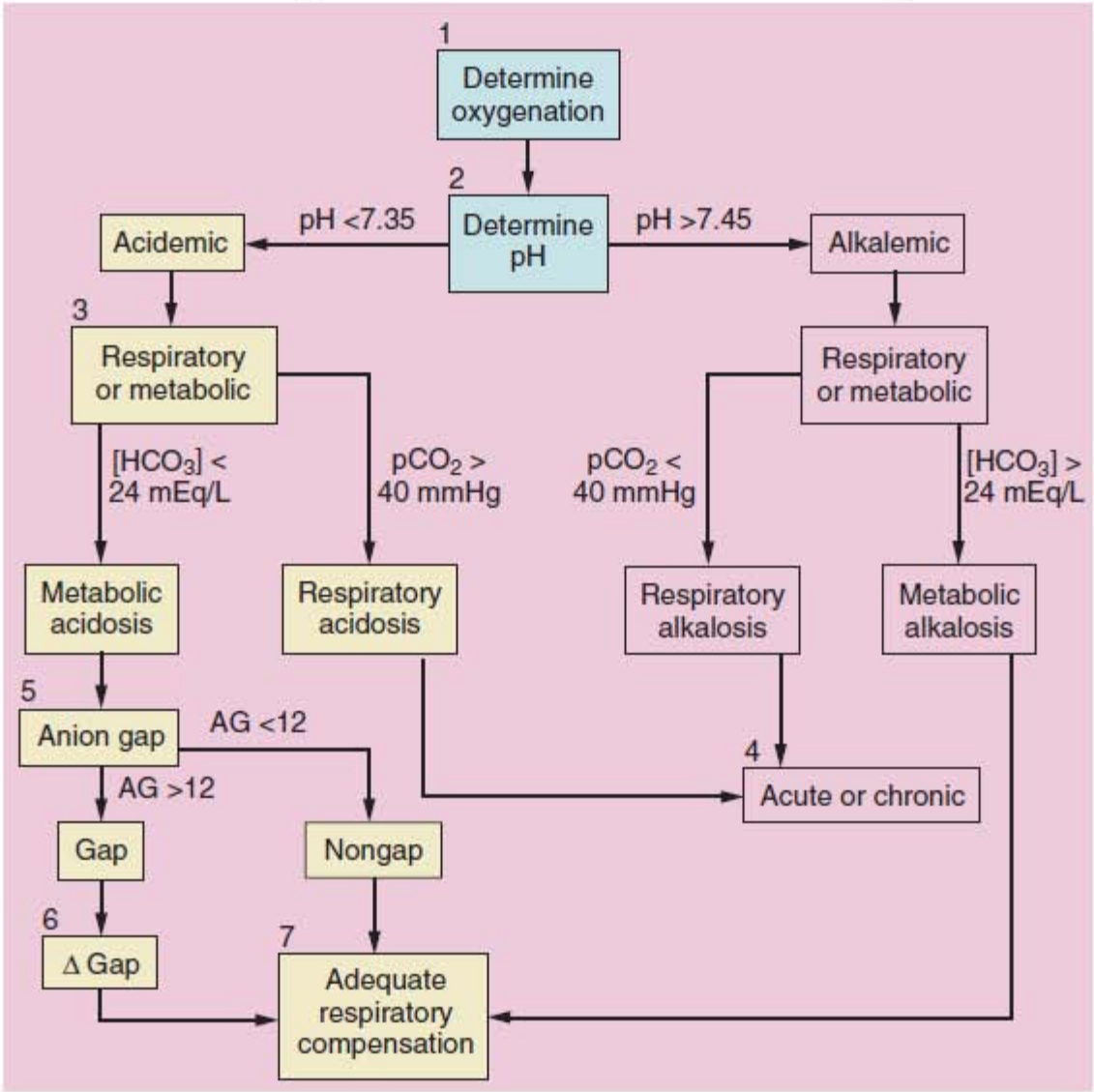
Seven steps for acid-base diagnosis



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Acid-Base Problems in Different Clinical Settings

Step 1. Look at the pH (three possibilities):

- <7.35—acidosis
- 7.35-7.45—normal or compensated acidosis
- >7.45—alkalosis

Step 2. Look for respiratory component (volatile acid = CO_2):

- Pco_2 <35 mm Hg—respiratory alkalosis or compensation for metabolic acidosis (if so, BD * > -5)
- Pco_2 35-45 mm Hg—normal range
- Pco_2 >45 mm Hg—respiratory acidosis (acute if pH <7.35, chronic if pH in normal range and BE[†]> +5)

Step 3. Look for a metabolic component (i.e., buffer base utilization):

- BD >-5—metabolic acidosis
- BE -5 to +5—normal range
- BE >5—alkalosis

Put this information together

1. Acidosis, CO_2 <35 mm Hg, ± BD >-5 → acute metabolic acidosis
2. Normal range pH CO_2 <35, BD >-5 → acute metabolic acidosis plus compensation
3. Acidosis, Pco_2 >45 mm Hg, normal range BE → acute respiratory acidosis
4. Normal range pH, Pco_2 >45 mm Hg, BE >+5 → prolonged respiratory acidosis
5. Alkalosis, Pco_2 >45 mm Hg, BE >+5 → metabolic alkalosis
6. Alkalosis, Pco_2 <35 mm Hg, BDE normal range → acute respiratory alkalosis
7. If the acid-base picture does not conform to any of these, a mixed picture is present.

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The alveolar gas equation,

calculation of alveolar-arterial (A-a) gradient

Alveolar gas equation: $P_AO_2 = (P_b\text{-}P_{H_2O})FIO_2 - PaCO_2/RQ$

P_AO_2 = alveolar partial pressure oxygen (mm Hg)
P_b = barometric pressure (760 mm Hg at sea level)
P_{H_2O} = partial pressure of water vapor (47 mm Hg at 37° C)
FIO_2 = fraction inspired oxygen concentration
RQ = respiratory quotient (0.8 for normal diet)

A-a gradient $= P_AO_2 - PaO_2$

For patient with PaO_2 of 363 mm Hg and $PaCO_2$ of 40 mm Hg breathing FIO_2 1.0

$P_AO_2 = (760 - 47)(1.0) - 40/0.8$
$= (713) - 50$
$= 663$ mm Hg

A-a gradient $= 663 - 363$
$= 300$ mm Hg

% shunt = 1% for every 20 mm Hg of A-a gradient
$= 300/20$
$= 15\%$

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Normally, the A-a gradient is

less than 15 mm Hg [breathing room air]

up to 60mm Hg [breathing FIO2 1.0]

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P/F RATIO

- The PaO2/ FIO2 (P/F) ratio is a simple alternative to the A-a gradient to communicate the degree of hypoxia for acute lung injury (ALI) versus
- ALI → P/F ratio below 300
- ARDS → P/F ratio below 200
- A ratio under 200 suggests a shunt fraction greater than 20%.

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Referrences

- Paul G. Barash, Chapter 9- Fluids, Electrolytes and Acid–Base Physiology , Clinical anesthesia, 5th ed., 2006.
- Paul G. Barash, Chapter 14- Fluids, Electrolytes and Acid–Base Physiology , Clinical anesthesia, 6th ed., 2009.
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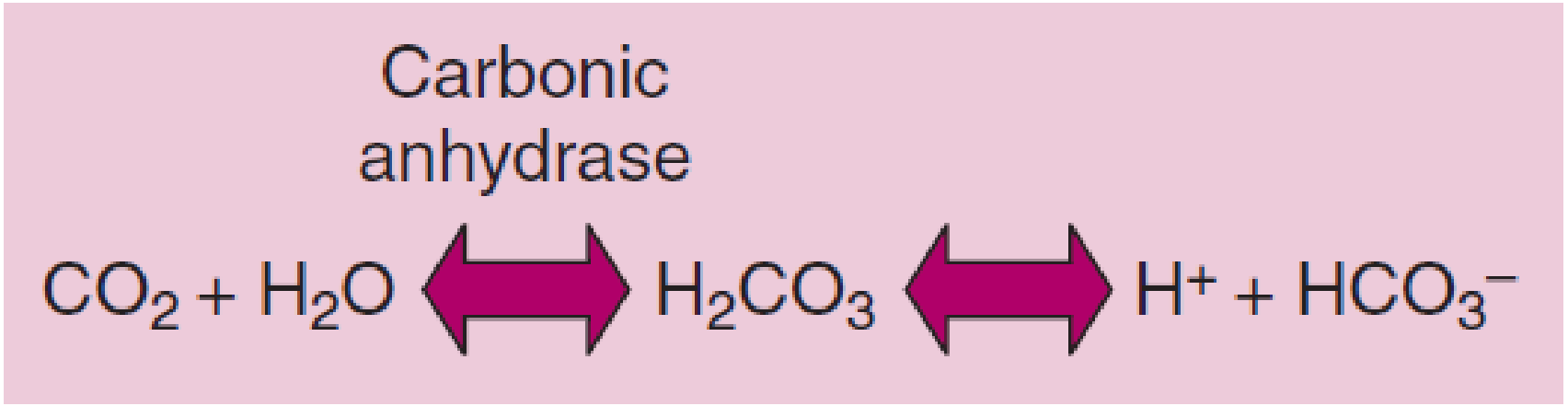
2. Practical Approach to Acid–Base Interpretation

Acid-Base Homeostasis

1. Buffer systems → first line of defense against the fall in pH
   1) $CO_2 + H_2O \overset{CA}{\longleftrightarrow} H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$
   2) $H_2PO_4^- \longleftrightarrow HPO_4^{2-}$
   3) Protein
   4) Hemoglobin
2. Respiratory system → within 15–30 min
3. Kidney → requires 3–5 days to complete compensation
   1) HCO_3^- reabsorption at proximal tubule
   2) Excretion H^+ ; $Na_2HPO_4 \longleftrightarrow NaH_2PO_4$, $NH_3 \longleftrightarrow NH_4^+$



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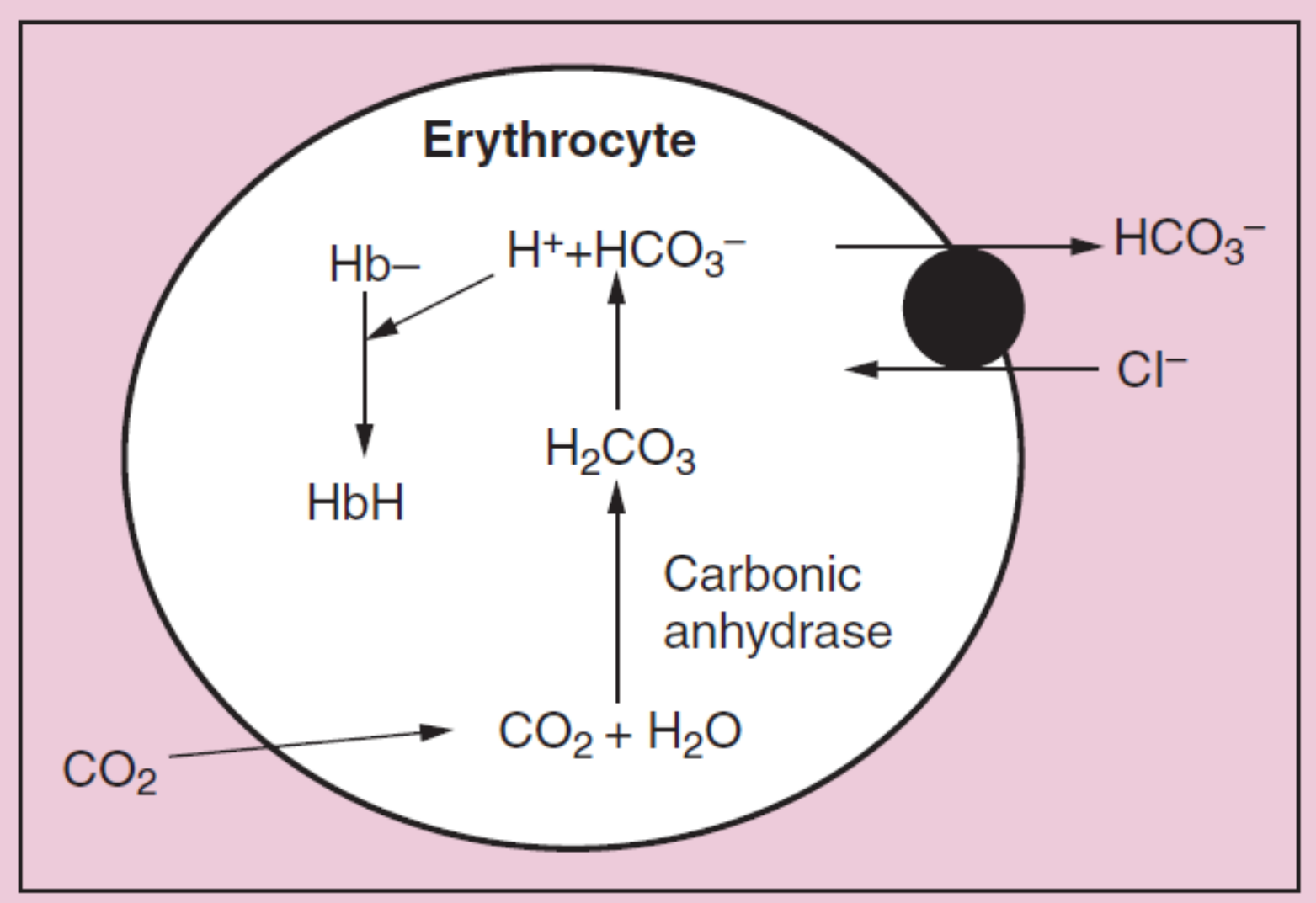
Hemoglobin buffering system



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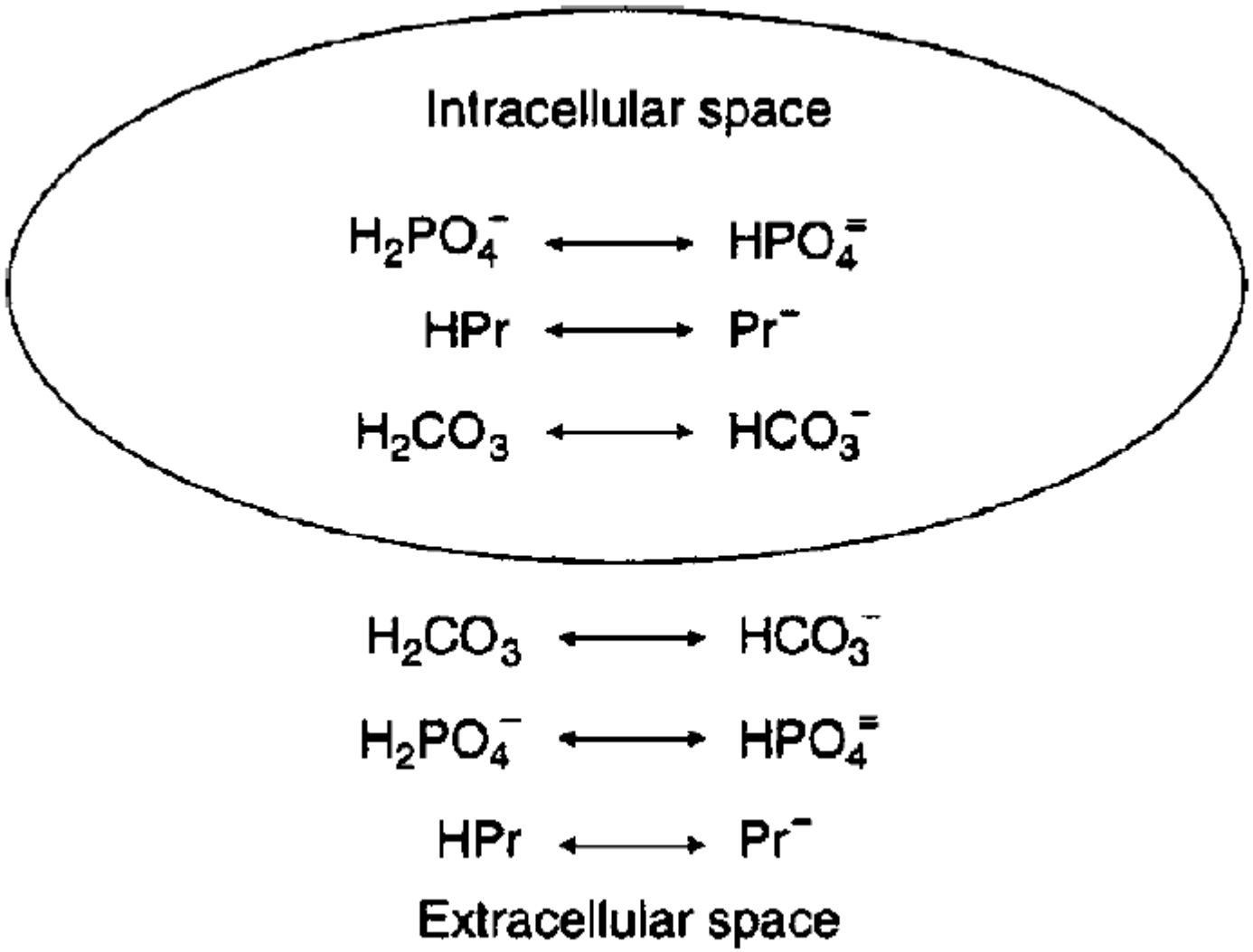
Buffering in the Intracellular



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Respiratory system

1. Central chemoreceptors
   - Locate at anterolateral surface of the medulla
   - respond to changes in cerebrospinal fluid pH
   - MV increases 1 to 4 L/min for every 1 mm Hg increase in $PaCO_2$
2. Peripheral chemoreceptors
   - Locate at bifurcation of the common carotid arteries and surrounding the aortic arch
   - most sensitive to PaO_2

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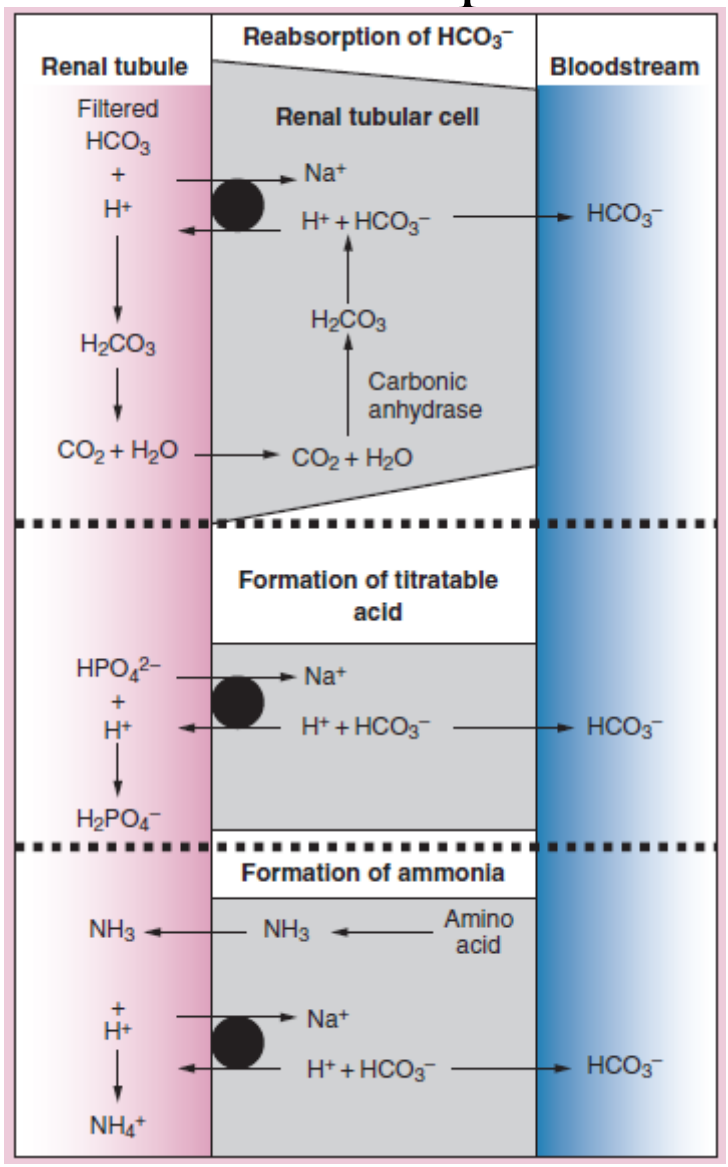
Three mechanisms of renal compensation during acidosis

1

2

3



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Henderson-Hasselbalch equation

$$\mathbf{pH} = 6.1 + \log_{10} \frac{[HCO_3^-]}{0.03 \times PaCO_2}$$

Henderson equation

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

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- Primary abnormalities of CO_2 tension are considered respiratory disturbances, whereas primary derangements of $[HCO_3^-]$ are metabolic disturbances

$$\mathbf{pH} = pK + \log_{10} \frac{[A^-]}{[HA]}$$

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Conversion of pH to Proton Concentration (nM/L)

pH	Proton Concentration (nM/L)
6.9	125
7.0	100
7.1	80
7.2	63
7.3	50
7.4	40
7.5	32
7.6	25

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General Principles of Acid-Base Homeostasis

- Acid-base homeostasis consists of the precise regulation of
  - CO_2 tension by the respiratory system → eliminating H^+ ~ 14,000 mEq/Day
  - plasma bicarbonate concentration [HCO_3^-] by the kidney
- The kidney regulates plasma [HCO_3^-] by
  - altering HCO_3^- reabsorption
  - eliminating protons (H^+) ~ 70 mEq/Day
- Body fluid pH is determined by CO_2 tension and [HCO_3^-]

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Serum Anion Gap (SAG)

$$SAG = [Na+] - [Cl^-] - [HCO_3^-]$$

Normally = 6 and 10 mEq/L

- Extremely elevate; SAG > 25 mEq/L) always reflects the presence of an organic acidosis
- low SAG 4 types;
  1. a reduction in the concentration of unmeasured anions (primarily albumin)
  2. increased unmeasured cations (hyperkalemia, hypermagnesemia, hypercalcemia, lithium toxicity, or a cationic paraprotein)
  3. underestimation of the serum Na+ concentration (severe hypernatremia)
  4. overestimation of the serum Cl^- concentration (bromide intoxication and marked hyperlipidemia)

each 1 g/dL decrease in serum Alb → SAG will decrease by 2.5 mEq/L

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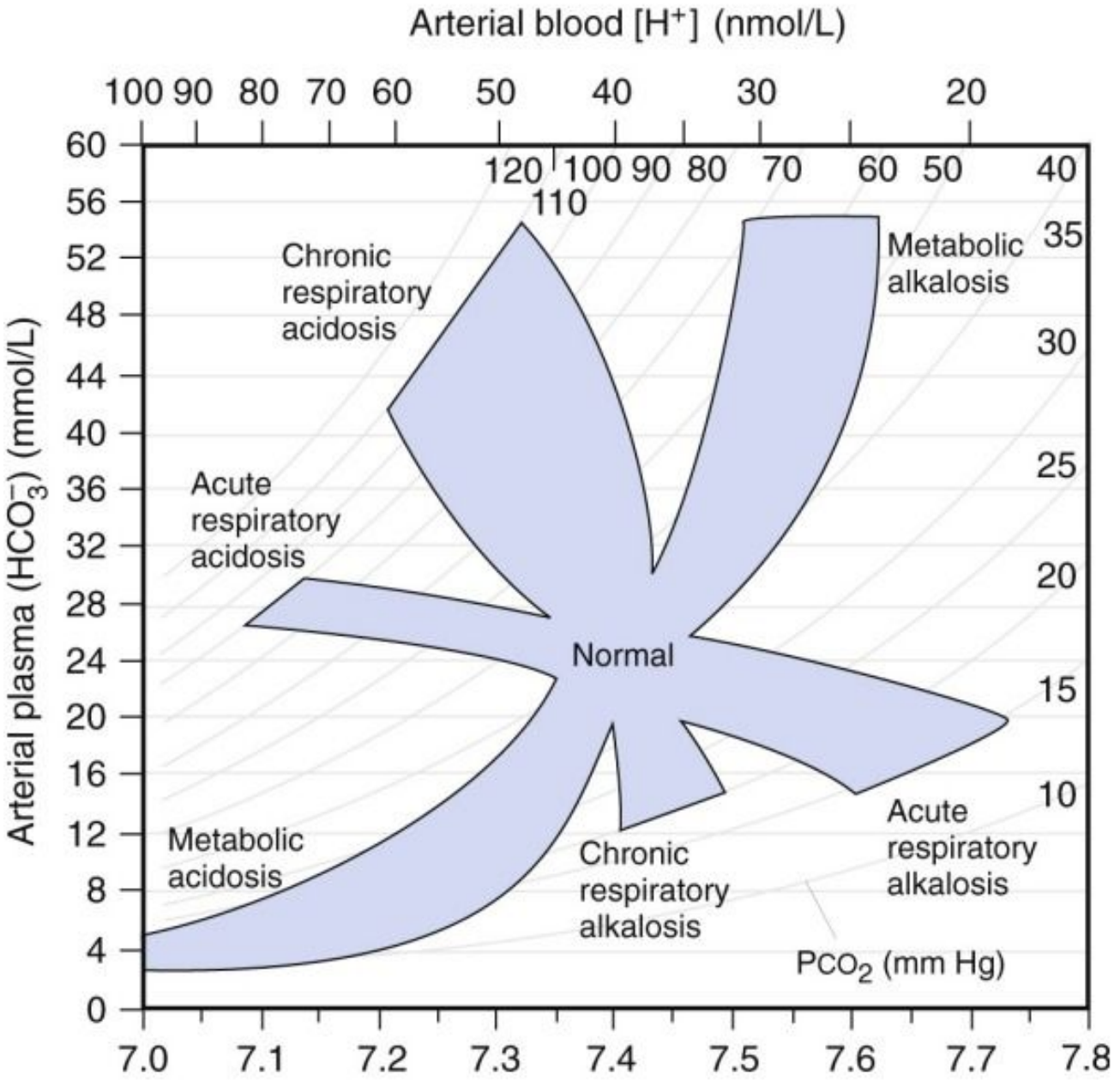

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Acid-Base Disturbances Commonly Seen Perioperatively

Disorder	Cause
Respiratory acidosis	Hypoventilation—narcosis, incomplete reversal of neuromuscular blockade
Respiratory alkalosis	Hyperventilation—anxiety, pain
Metabolic acidosis secondary to unmeasured anions (widened gap acidosis)	Hypoperfusion—lactic acidosis; diabetic ketoacidosis; renal failure
Metabolic acidosis secondary to measured anions (non-gap hyperchloremic acidosis)	Hyperchloremia—"normal" saline, hetastarch, or albumin infusions; renal tubular acidosis; bladder reconstructions
Metabolic acidosis secondary to free water excess (hyponatremia, dilution acidosis)	Hypotonic fluid administration; sodium loss—diarrhea; administration of hyperosmolar fluids—mannitol, alcohol, hyperproteinemia
Metabolic alkalosis	Hyperventilation of patient with history of carbon dioxide retention (COPD); sodium gain (sodium bicarbonate massive blood transfusion); chloride loss—nasogastric suctioning

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Causes of Respiratory Acidosis

Decreased Alveolar Ventilation

- Central nervous system depression (opioids, general anesthetics)
- Peripheral skeletal muscle weakness (neuromuscular blockers, myasthenia gravis)
- Chronic obstructive pulmonary disease
- Acute respiratory failure

Increased Carbon Dioxide Production

- Hypermetabolic states
  - Fever
  - Malignant hyperthermia
  - Sepsis
  - Multiple trauma
  - Hyperalimentation

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Causes of Respiratory Acidosis

Increased production
Malignant hyperthermia
Hyperthyroidism
Sepsis
Overfeeding
Decreased elimination
Intrinsic pulmonary disease (pneumonia, ARDS, fibrosis, edema)
Upper airway obstruction (laryngospasm, foreign body, OSA)
Lower airway obstruction (asthma, COPD)
Chest wall restriction (obesity, scoliosis, burns)
CNS depression (anesthetics, opioids, CNS lesions)
Decreased skeletal muscle strength (residual effects of neuromuscular blocking drugs, myopathy, neuropathy)
Increased rebreathing or absorption
Exhausted soda lime
Incompetent one-way valve
Laparoscopic surgery

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Causes of Respiratory Alkalosis

- Hyperventilation syndrome (diagnosis of exclusion; most often encountered in the emergency department)
- Iatrogenic hyperventilation
- Pain
- Anxiety
- Arterial hypoxemia
- Central nervous system disease
- Systemic sepsis

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Causes of Respiratory Alkalosis

Increased minute ventilation
Hypoxia (high altitude, low F_{IO_2}, severe anemia)
Iatrogenic (mechanical ventilation)
Anxiety and pain
CNS disease (tumor, infection, trauma)
Fever, sepsis
Drugs (salicylates, progesterone, doxapram)
Liver disease
Pregnancy
Restrictive lung disease
Pulmonary embolism
Decreased production
Hypothermia
Skeletal muscle paralysis

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Physiologic Effects Produced by Respiratory Alkalosis

- Hypokalemia (potentiates toxicity of digoxin)
- Hypocalcemia
- Cardiac dysrhythmias
- Bronchoconstriction
- Hypotension
- Decreased cerebral blood flow (returns to normal > 8 to 24 hrs corresponding to the return of cerebrospinal fluid pH to normal)

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Rules of Thumb for $[HCO_3^-]$ and pH Changes in Response to Acute and Chronic Changes in $PaCO_2$

Decreased $PaCO_2$

1. pH increases 0.10 for every 10 mmHg decrease in $PaCO_2$.
2. $[HCO_3^-]$ decreases 2 mEq/L for every 10 mmHg decrease in $PaCO_2$.
3. pH will nearly normalize if hypocarbia is sustained.
4. $[HCO_3^-]$ will decrease 5 to 6 mEq/L for each chronic 10 mmHg ↓ in $PaCO_2$.[a]

Increased $PaCO_2$

1. pH will decrease 0.05 for every acute $PaCO_2$ increase of 10 mmHg.
2. $[HCO_3^-]$ will increase 1.0 mEq/L for every $PaCO_2$ increase of 10 mmHg.
3. pH will return toward normal if hypercarbia is sustained.
4. $[HCO_3^-]$ will increase 4 to 5 mEq/L for each chronic 10 mmHg increase in $PaCO_2$.

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Changes in Paco2 and [HCO3-] in Response to Acute and Chronic Acid-Base Disturbances

Disturbances	$[HCO_3^-]$ versus $Paco_2$
Acute respiratory acidosis	$\Delta HCO_3^- = 0.2\ \Delta Paco_2$
Acute respiratory alkalosis	$\Delta HCO_3^- = 0.2\ \Delta Paco_2$
Chronic respiratory acidosis	$\Delta HCO_3^- = 0.5\ \Delta Paco_2$
Metabolic acidosis	$\Delta Paco_2 = 1.3\ \Delta HCO_3^-$
Metabolic alkalosis	$\Delta Paco_2 = 0.75\ \Delta HCO_3^-$

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Determining Whether Respiratory Process

Is Acute or Chronic

Acute Process

pH △ 0.08 for every 10 mm Hg △ in Pco_2 from 40 mm Hg

Chronic Process

pH △ 0.03 for every 10 mm Hg △ in Pco_2 from 40 mm Hg

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Physiologic Effects Produced by Metabolic Acidosis

- Decreased myocardial contractility
- Increased pulmonary vascular resistance
- Decreased systemic vascular resistance
- Impaired response of the cardiovascular system to endogenous and exogenous catecholamines
- Compensatory hyperventilation

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Causes of Metabolic Acidosis

Anion Gap Acidosis

M methanol, ethylene glycol
U uremia
L lactic acidosis = CHF, sepsis, cyanide toxicity
E ethanol
P paraldehyde
A aspirin, INH
K ketones = starvation, diabetic

Nongap Acidosis

Administration of 0.9% NaCl
GI losses—diarrhea, ileostomy, neobladder, pancreatic fistula
Renal losses—RTA
Drugs—acetazolamide

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Differential Diagnosis of Metabolic Acidosis

·ELEVATED ANION GAP	·NORMAL ANION GAP
Three diseases	
1. Uremia 2. Ketoacidosis 3. Lactic acidosis	1. Renal tubular acidosis 2. Diarrhea 3. Carbonic anhydrase inhibition 4. Ureteral diversions 5. Early renal failure 6. Hydronephrosis 7. HCl administration 8. Saline administration
Toxins	
1. Methanol 2. Ethylene glycol 3. Salicylates 4. Paraldehyde	

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Evaluation of a Patient with Metabolic Acidosis

1. Is acidosis being caused by measured or unmeasured anions (chloride)?
   - Look at blood chemistry

Calculate anion gap: Na + K - Cl = 10-12

If gap is normal → excess chloride , excess loss of sodium (diarrhea, ileostomy), renal tubular acidosis

If gap is wide (>16) → unmeasured anions causing acidosis

   - Check serum lactate

if >2 probably lactic acidosis ; circulatory insufficiency (shock, hypovolemia, oliguria, under-resuscitation, anemia, carbon monoxide poisoning, seizures) → "type A" lactic acidosis

If not → "type B (rare)" ; biguanides, fructose, sorbitol, nitroprusside, ethylene glycol, cancer, liver disease

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Evaluation of a Patient with Metabolic Acidosis(cont.)

– Look at creatinine & urine output

If patient is in acute renal failure → renal acids

– Look at blood glucose & urinary ketones

If patient is hyperglycemic & ketotic → diabetic ketoacidosis

If patient is ketotic (unmeasured anion) & normoglycemic → alcoholic or starvation ketosis

Check for presence of chronic alcohol abuse—high mean corpuscular volume, increased γ-glutamyl transferase on liver panel

2. If all of these tests are negative, think of intoxication

   – Send toxicology laboratory tests (particularly salicylates) and serum osmolality → calculate osmolality :

$$2(\text{Na} + \text{K}) + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{2.8}$$

   – Look for unmeasured source of osmoles

if gap between measured and calculated serum osmolality >12, think of alcohol, particularly ethylene glycol, isopropyl alcohol, and methanol

Causes of Increased Anion Gap (Organic) Metabolic Acidosis

Increased acid production

- Lactic acidosis
- Ketoacidosis ; Diabetic ketoacidosis, Starvation, Alcoholic ketoacidosis
- Toxic alcohol ingestion
- Salicylate overdose
- Pyroglutamic acidosis
- Other intoxications (e.g., toluene, isoniazid and propylene glycol)
- Inborn errors of metabolism

Failure of acid excretion

- Acute kidney injury
- Chronic kidney disease

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Anesthetic Implications of Metabolic Acidosis

- Monitor arterial blood gases and pH
- Possible exaggerated hypotensive responses to drugs and PPV of the patient's lungs (reflects hypovolemia)
- Consider monitoring with an intra-arterial catheter and pulmonary artery catheter
- Maintain previous degree of compensatory hyperventilation

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Calculation of Sodium Bicarbonate Dose

$$\text{Sodium Bicarbonate (mEq/L)} = \frac{\text{weight (kg)} \times 0.3 \times (24\ \text{mEq/L [actual bicarbonate]})}{2}$$

infants & children → initial dose = 1.0 to 2.0 mEq/kg of body weight

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Generation of Metabolic Alkalosis

·GENERATION	·EXAMPLE	·MAINTENANCE
I. Loss of acid from extracellular space		
A. Loss of gastric fluid	Vomiting; nasogastric drainage	↓ effective arterial volume (EAV)
B. Loss of acid into urine; continued Na^+ delivery to the distal tubule in presence of hyperaldosteronism	1. Primary aldosteronism 2. Diuretic administration	1. K^+ depletion + aldosterone excess 2. ↓ EAV + K^+ depletion

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·GENERATION	·EXAMPLE	·MAINTENANCE
II. Excessive HCO_3^- loads		
A. Absolute	$NaHCO_3$ administration	↓EAV
1. HCO_3^-	Lactate, acetate, citrate administration	↓EAV
2. Metabolic conversion of salts of organic acid anions to HCO_3^-	Alkali administration to patients with renal failure	Renal failure
B. Relative		
1. Alkaline loads in renal failure		
III. Posthypercapnic state	Abrupt correction of chronic hypercapnia	↓EAV

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Causes of Metabolic Alkalosis

Chloride Responsive

Renal loss—diuretic therapy

GI loss—vomiting, NG suction

Alkali administration—citrate in blood products, acetate in TPN, bicarbonate

Chloride Resistant

Hyperaldosteronism

Refeeding syndrome

Profound hypokalemia

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Rules of Thumb for Respiratory Compensation in Response to Metabolic Alkalosis and Metabolic Acidosis

Metabolic alkalosis

1. $PaCO_2$ increases approximately 0.5 to 0.6 mmHg for each 1.0 mEq/L increase in $[HCO^-_3]$
2. The last two digits of the pH should equal the $[HCO^-_3]$ + 15

Metabolic acidosis

1. $PaCO_2 = [HCO^-_3] \times 1.5 + 8$
2. $PaCO_2$ decreases 1.2 mmHg for every 1.0 mEq/L in $[HCO^-_3]$ to a minimum of 10 to 15 mmHg
3. The last two digits of the pH equal $[HCO^-_3]$ + 15

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Factors that Maintain Metabolic Alkalosis

Factor	Proposed Mechanism
Decreased GFR	Increases fractional bicarbonate reabsorption and prevents elevated plasma bicarbonate concentrations from exceeding Tm
Volume contraction	Stimulates proximal tubular bicarbonate reabsorption
Hypokalemia	Decreases GFR and increases proximal tubular bicarbonate reabsorption Stimulates sodium-independent/potassium-dependent (low) secretion in cortical collecting tubules
Hypochloremia*	Increases renin Decreases distal chloride delivery
Passive backflux of bicarbonate	Creates a favorable concentration gradient for passive bicarbonate movement from proximal tubular lumen to blood
Aldosterone	Increases sodium-dependent proton secretion in cortical collecting tubules and sodium-independent proton secretion in cortical collecting tubules and medullary collecting tubules

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Physiologic Effects Produced by Metabolic Alkalosis

- Hypokalemia (potentiates effects of digoxin; evokes ventricular cardiac dysrhythmias)
- Decreased serum ionized calcium concentration
- Compensatory hypoventilation (may be exaggerated in patients with COPD or those who have received opioids; compensatory hypoventilation rarely results in $PaCO_2$ >55 mm Hg)
- Arterial hypoxemia (reflects effect of compensatory hypoventilation)
- Increased bronchial tone (may contribute to atelectasis)
- Leftward shift of oxyhemoglobin dissociation curve (oxygen less available to tissues)
- Decreased cardiac output
- Cardiovascular depression and cardiac dysrhythmias (result of inadvertent iatrogenic respiratory alkalosis to pre-existing metabolic alkalosis during anesthetic management)

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Treatment of Metabolic Alkalosis

Etiologic Therapy

- Expand intravascular fluid volume (intraoperative fluid management with 0.9% saline; lactated Ringer's solution provides an additional substrate for generation of bicarbonate).
- Administer potassium.
- Avoid iatrogenic hyperventilation of the patient's lungs.

Nonetiologic Therapy

- Administer acetazolamide (causes renal bicarbonate wasting).
- Administer hydrogen (ammonium chloride, arginine hydrochloride, hydrochloric acid [must be injected into a central vein]).

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Determining Appropriate Compensation in Acid-Base Disorders

Metabolic Acidosis

Winter's formula:

$$P_{CO_2} = (1.5 \times HCO_3^-) + 8$$

If measured P_{CO_2} > calculated P_{CO_2}, then concurrent respiratory acidosis is present.

If measured P_{CO_2} < calculated P_{CO_2}, then concurrent respiratory alkalosis is present.

Metabolic Alkalosis

$$P_{CO_2} = (0.7 \times HCO_3^-) + 21$$

If measured P_{CO_2} > calculated P_{CO_2}, then concurrent respiratory acidosis is present.

If measured P_{CO_2} < calculated P_{CO_2}, then concurrent respiratory alkalosis is present.

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Sequential Approach to Acid-Base Interpretation

1. Is the pH life threatening, requiring immediate intervention?
2. Is the pH acidemic or alkalemic?
3. Could the entire arterial blood gas picture represent only an acute increase or decrease in $PaCO_2$?
4. If the answer to question #3 is "No," is there evidence of a chronic respiratory disturbance or of an acute metabolic disturbance?
5. Are appropriate compensatory changes present?
6. Is an anion gap present?
7. Do the clinical data fit the acid-base picture?

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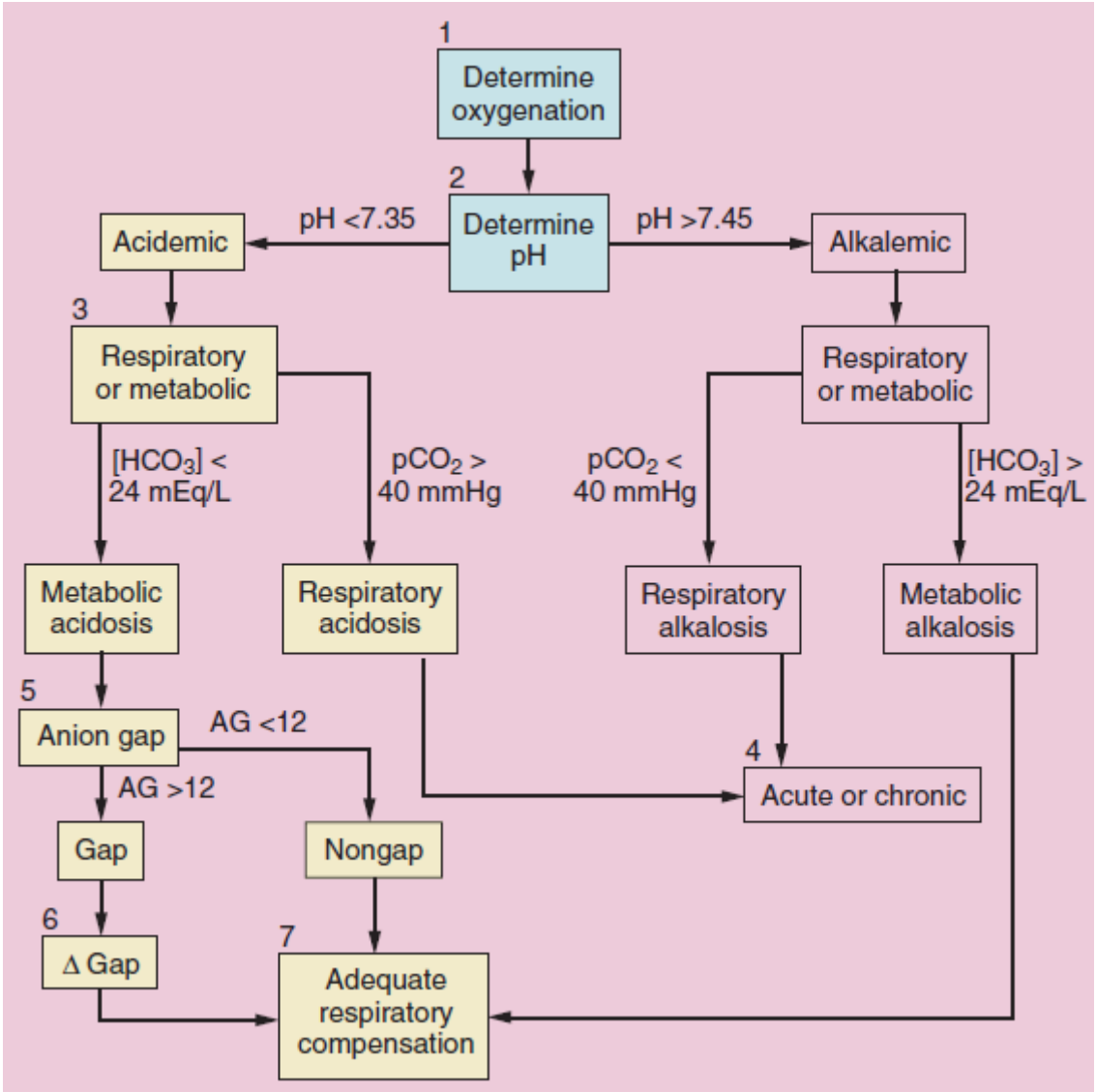
Seven steps for acid-base diagnosis



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Acid-Base Problems in Different Clinical Settings

Step 1. Look at the pH (three possibilities):

- <7.35—acidosis
- 7.35-7.45—normal or compensated acidosis
- >7.45—alkalosis

Step 2. Look for respiratory component (volatile acid = CO_2):

- Pco_2 <35 mm Hg—respiratory alkalosis or compensation for metabolic acidosis (if so, BD * > -5)
- Pco_2 35-45 mm Hg—normal range
- Pco_2 >45 mm Hg—respiratory acidosis (acute if pH <7.35, chronic if pH in normal range and BE[†]> +5)

Step 3. Look for a metabolic component (i.e., buffer base utilization):

- BD >-5—metabolic acidosis
- BE -5 to +5—normal range
- BE >5—alkalosis

Put this information together

1. Acidosis, CO_2 <35 mm Hg, ± BD >-5 → acute metabolic acidosis
2. Normal range pH CO_2 <35, BD >-5 → acute metabolic acidosis plus compensation
3. Acidosis, Pco_2 >45 mm Hg, normal range BE → acute respiratory acidosis
4. Normal range pH, Pco_2 >45 mm Hg, BE >+5 → prolonged respiratory acidosis
5. Alkalosis, Pco_2 >45 mm Hg, BE >+5 → metabolic alkalosis
6. Alkalosis, Pco_2 <35 mm Hg, BDE normal range → acute respiratory alkalosis
7. If the acid-base picture does not conform to any of these, a mixed picture is present.

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The alveolar gas equation,

calculation of alveolar-arterial (A-a) gradient

Alveolar gas equation: $P_AO_2 = (P_b\text{-}P_{H_2O})FIO_2 - PaCO_2/RQ$

P_AO_2 = alveolar partial pressure oxygen (mm Hg)
P_b = barometric pressure (760 mm Hg at sea level)
P_{H_2O} = partial pressure of water vapor (47 mm Hg at 37° C)
FIO_2 = fraction inspired oxygen concentration
RQ = respiratory quotient (0.8 for normal diet)

A-a gradient $= P_AO_2 - PaO_2$

For patient with PaO_2 of 363 mm Hg and $PaCO_2$ of 40 mm Hg breathing FIO_2 1.0

$P_AO_2 = (760 - 47)(1.0) - 40/0.8$
$= (713) - 50$
$= 663$ mm Hg

A-a gradient $= 663 - 363$
$= 300$ mm Hg

% shunt = 1% for every 20 mm Hg of A-a gradient
$= 300/20$
$= 15\%$

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Normally, the A-a gradient is

less than 15 mm Hg [breathing room air]

up to 60mm Hg [breathing FIO2 1.0]

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P/F RATIO

- The PaO2/ FIO2 (P/F) ratio is a simple alternative to the A-a gradient to communicate the degree of hypoxia for acute lung injury (ALI) versus
- ALI → P/F ratio below 300
- ARDS → P/F ratio below 200
- A ratio under 200 suggests a shunt fraction greater than 20%.

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Referrences

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