



Acid Base and Respiratory Disorders:

Getting the Most Information from Blood Gas Analysis

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Outline

Acid base and Respiratory physiology

- Normal homeostatic mechanisms

Acid base and Respiratory Pathologies

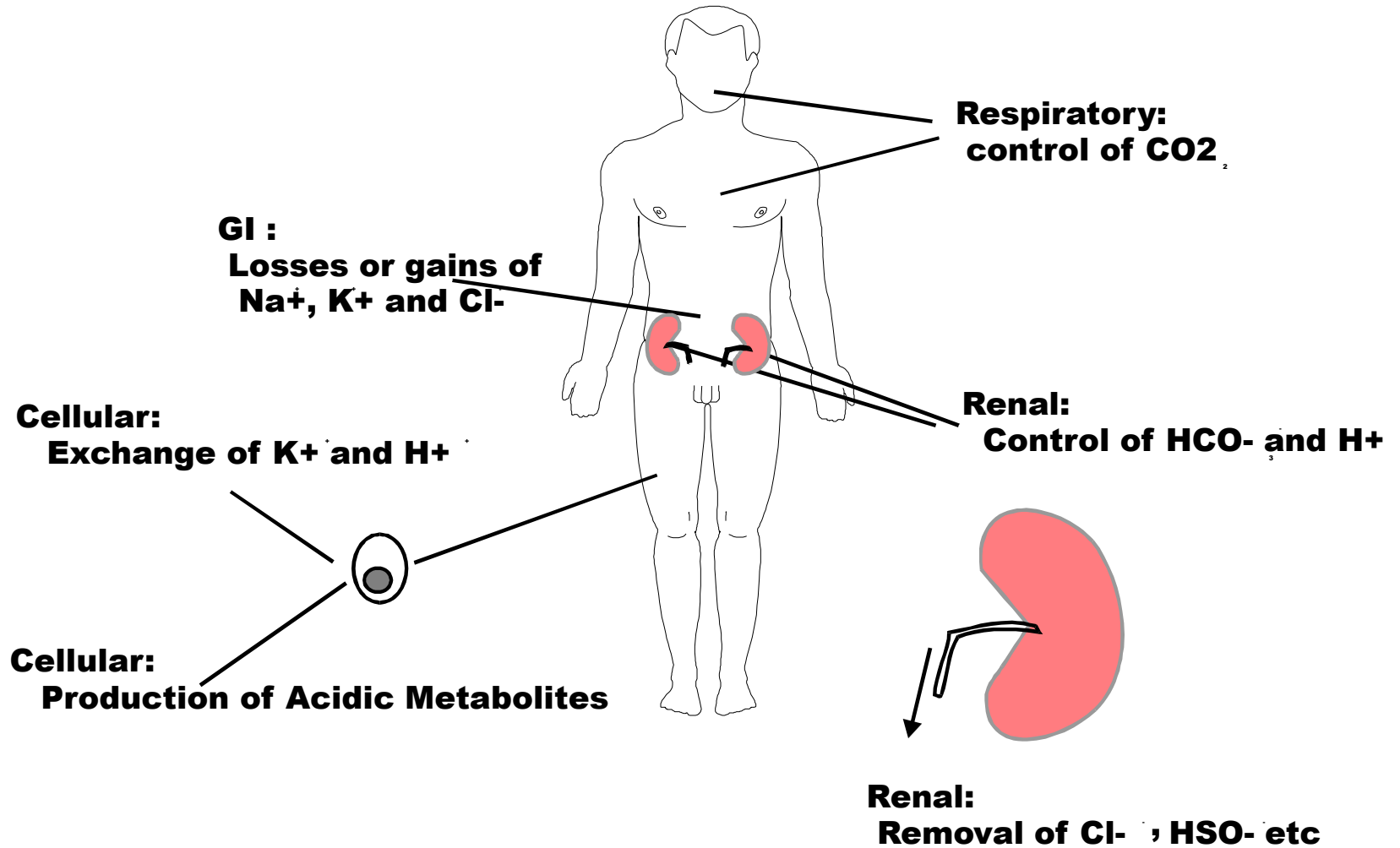
Compensation mechanisms

Cases

- Putting it all together



Acid - base Homeostatic Mechanisms



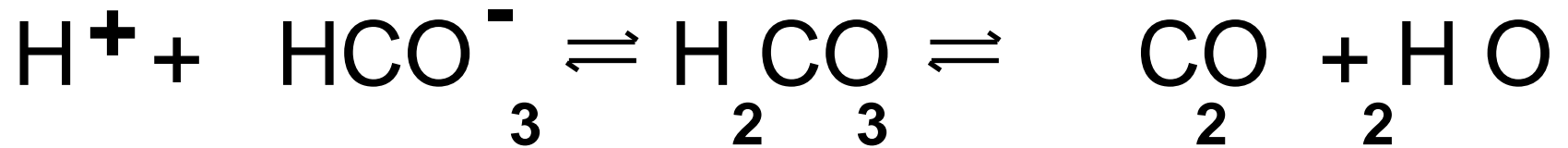


Buffering Systems

	pKA
• Haemoglobin	8.2
• Bicarbonate	6.1
• Phosphate	6.1
• Proteins	7
• Ammonia	9.2
• Misc organic acids	4 - 6



Henderson-Hasselbalch Equation



$$\text{pH} = \text{pK} + \log \left[\frac{[\text{HCO}_3^-]}{\text{pCO}_2 \times \alpha} \right]$$

[H⁺] homeostasis requires a balance between
H⁺ production and regeneration of HCO₃⁻



Acid base Homeostasis

Respiration



Properties of Oxygen & Carbon dioxide

CO₂ Buffering > O₂

CO₂ Permeability >> O₂

- Pa CO₂ ~ PA CO₂

Arterial PO₂ = 90 mmHg PCO₂ = 40 mmHg

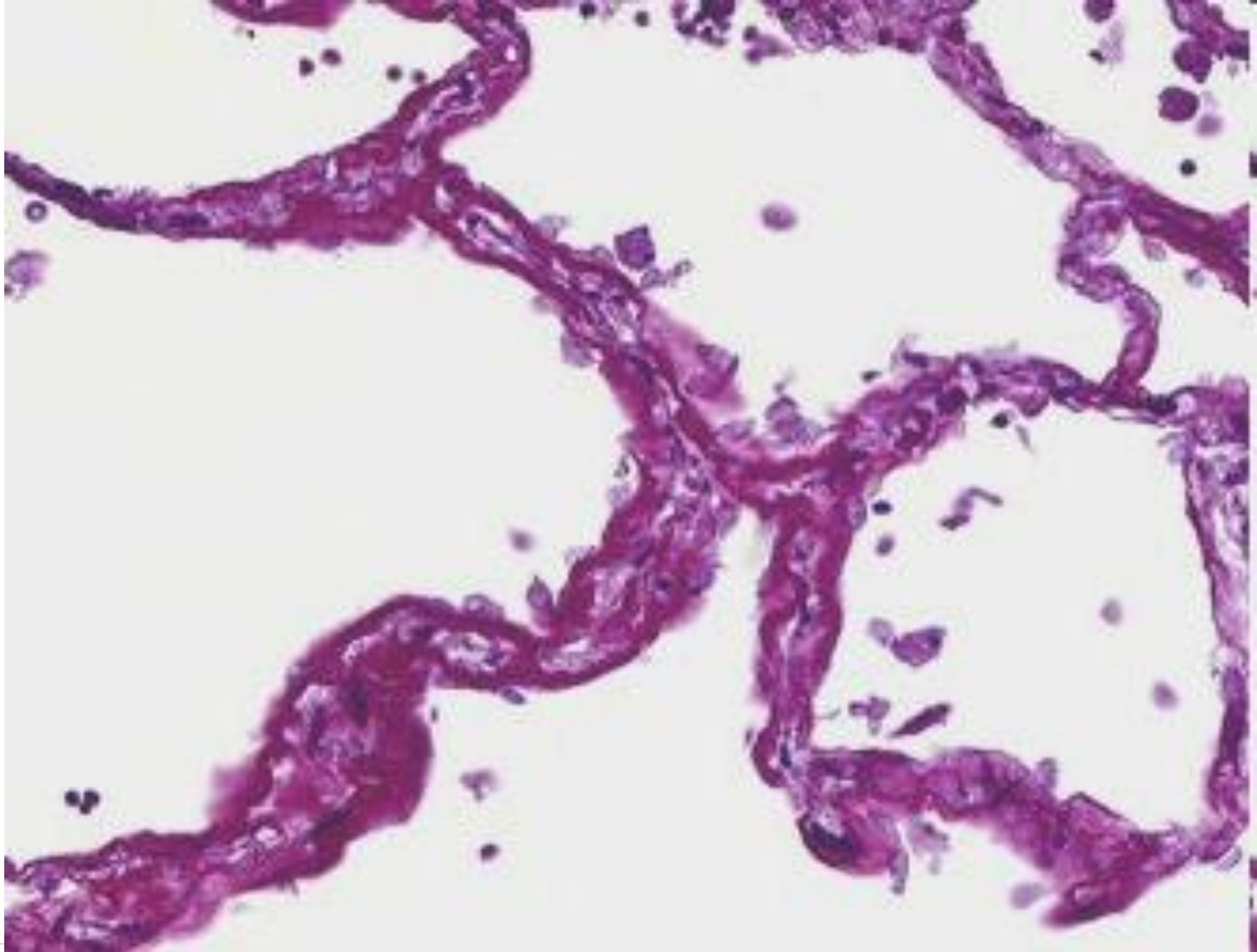
Venous PO₂ = 37 mmHg PCO₂ = 46 mmHg

Arterial O₂ content = 20 %_{v/v} CO₂ content = 49 %_{v/v}

Venous O₂ content = 15 %_{v/v} CO₂ content = 53 %_{v/v}

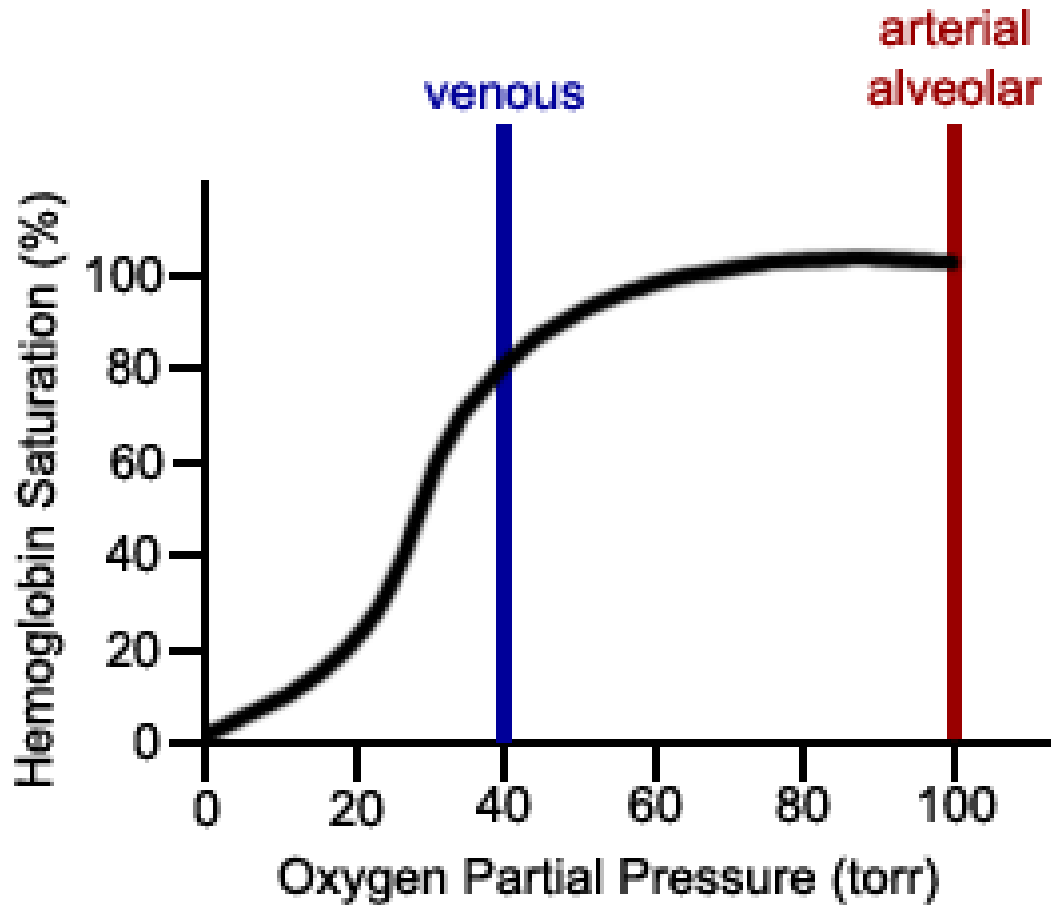


Gas Exchange





Hemoglobin Oxygen Dissociation Curve

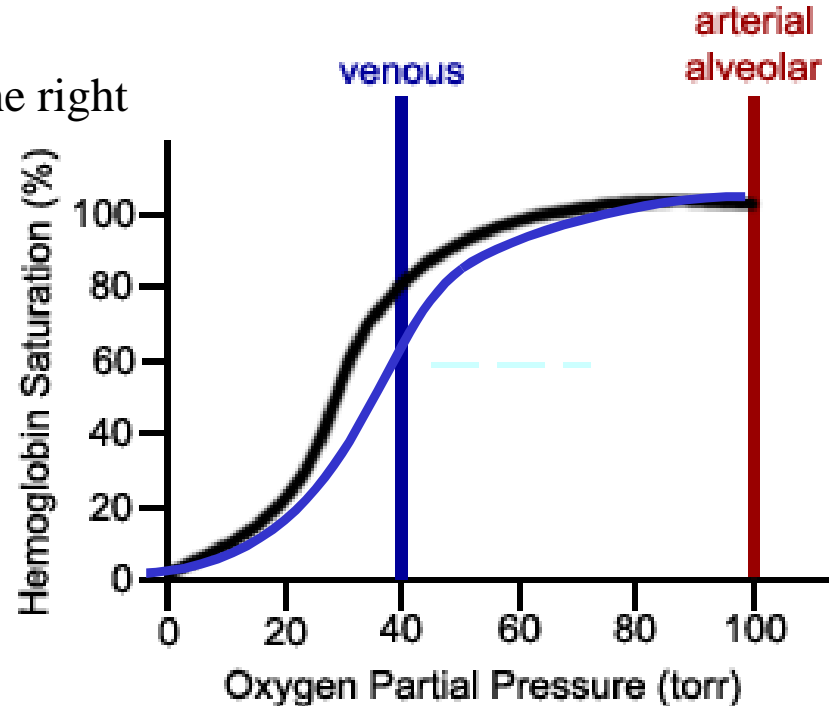




O₂ and CO₂ Interdependence

↑ PCO₂ shifts the O₂ dissociation curve to the right

the Bohr effect.



- ↑ temperature also shifts the curve to the right.

During exercise these effects increase the amount of O₂ released to the tissues while having little effect on the arterial O₂ content leaving the lungs.

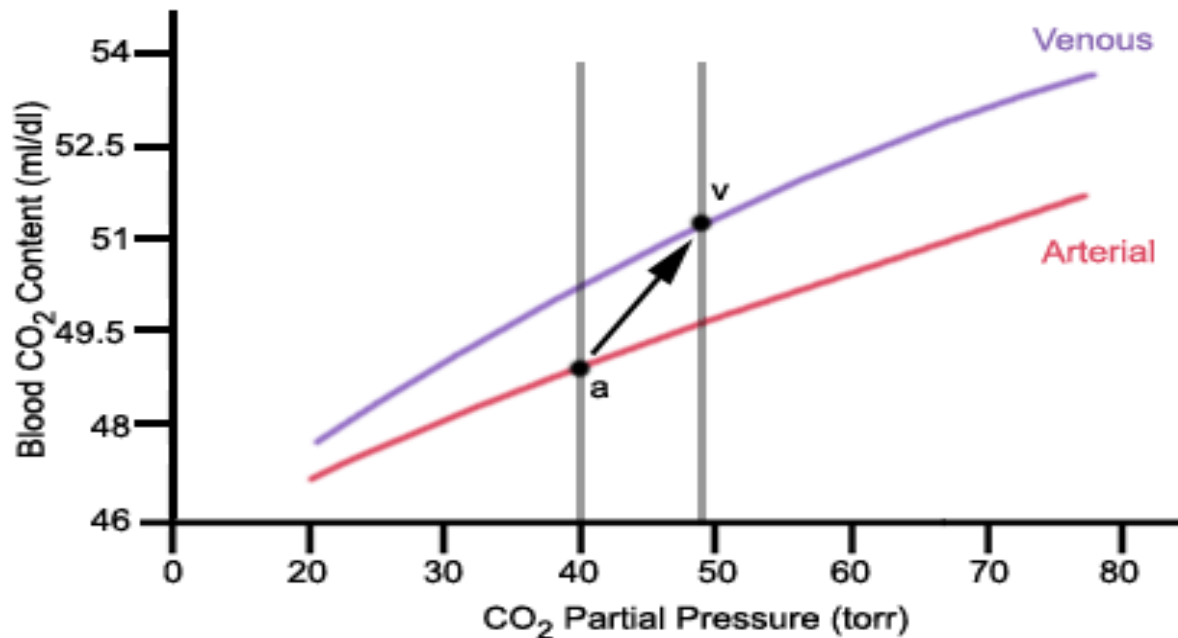


CO₂ Transport by Hemoglobin

The Haldane Effect

-Oxygen binding to HgB decreases CO₂ affinity

→ **Arterial** HgB has less capacity for CO₂ than **Venous** HgB





Calculating Cardiac output:

The rate of O₂ consumption is equal to the amount of O₂ taken up by each unit volume of blood times the rate at which these units flow through the lungs, thus

the Fick Equation $\dot{V}_{O_2} = Qt \times (C_{aO_2} - C_{vO_2})$

- where CaO₂ is the arterial O₂ content (measured in litres of O₂ at standard temperature and pressure per litre of blood), and
- CvO₂ is the mixed venous blood oxygen content.

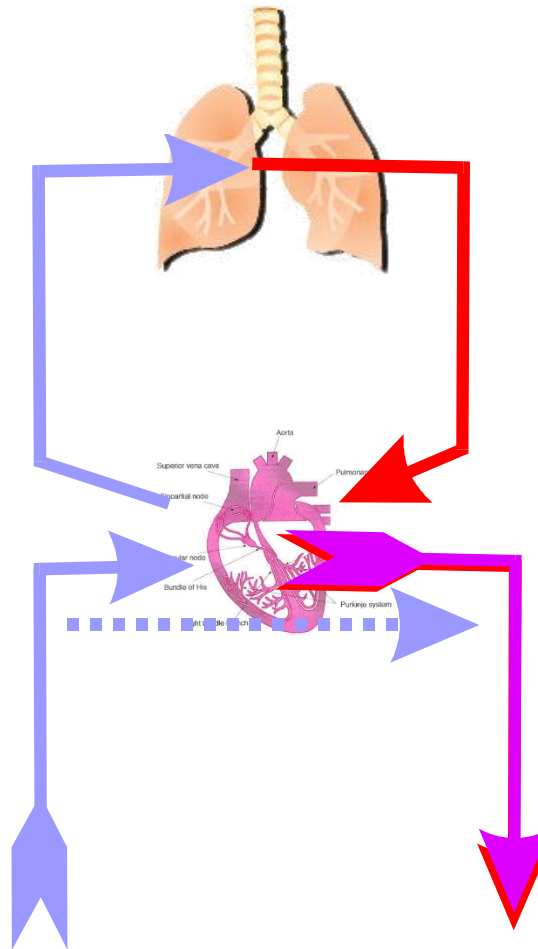


Calculating Cardiac output:

- Assumes: Cardiac Output = Pulmonary Perfusion rate
 - $Qt = V_{O_2} / (Ca_{O_2} - Cv_{O_2})$ **Fick Equation rearranged**
- Measure Arterial and Venous Blood Gases and Hemoglobin
- $O_2 \text{ Content} = \% \text{ Saturation} \times [\text{Hgb}] \times 1.34 + \text{Dissolved } O_2 (0.003 \times PO_2)$
- $V_{O_2} = \text{Ventillation rate} \times \%O_2$

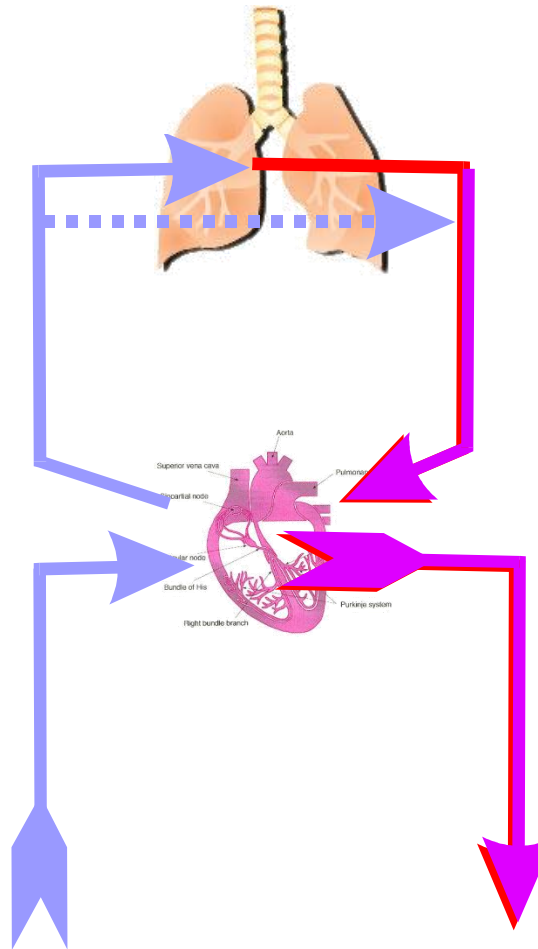


Respiration Anatomical Shunt





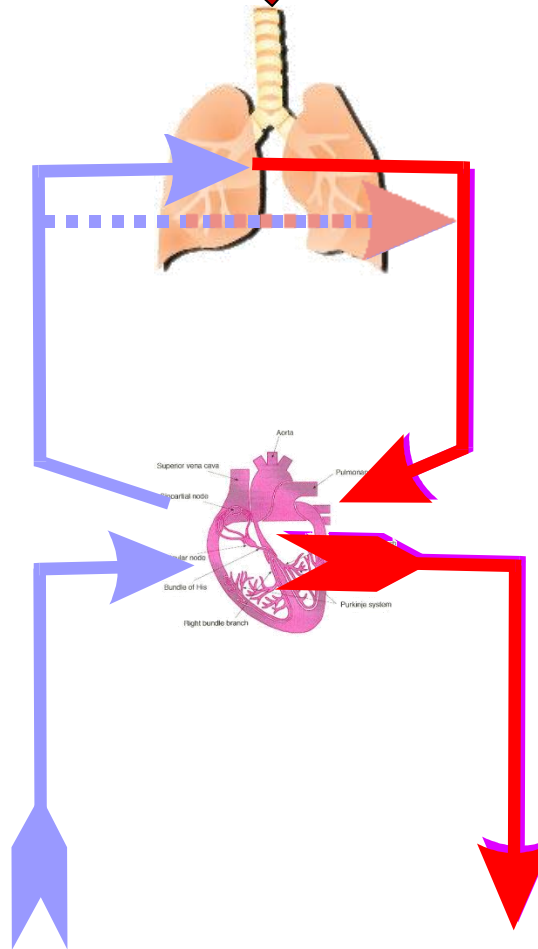
Respiration Physiological Shunt





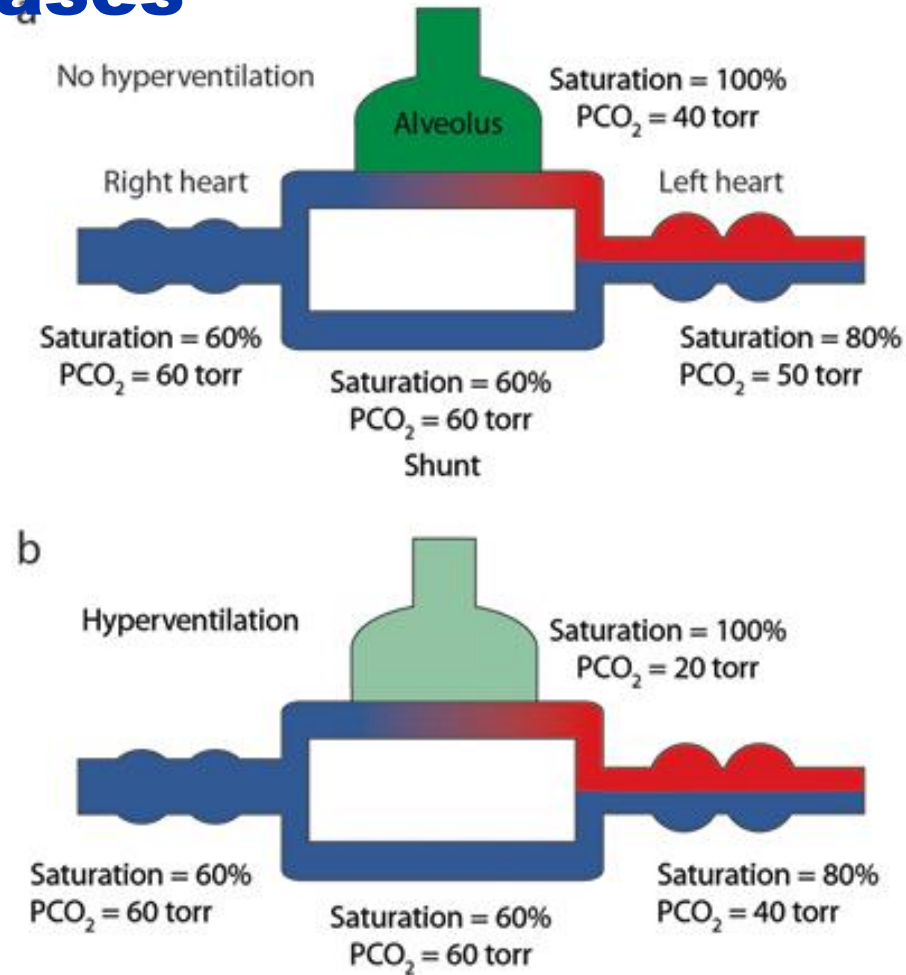
Respiration Physiological Shunt

100 % O₂





Effect Of Venous Shunting on Arterial Blood Gases





REFERENCE VALUES FOR BLOOD GASES

ANALYTE	ARTERIAL	VENOUS
p H	7.36 - 7.44	7.31 - 7.41
[H +]	44 - 36 nmol/L	49 - 39 nmol/L
p CO ₂	38 - 42 mmHg	35 - 40 mmHg
p O ₂	85 - 100 mmHg	35 - 40 mmHg

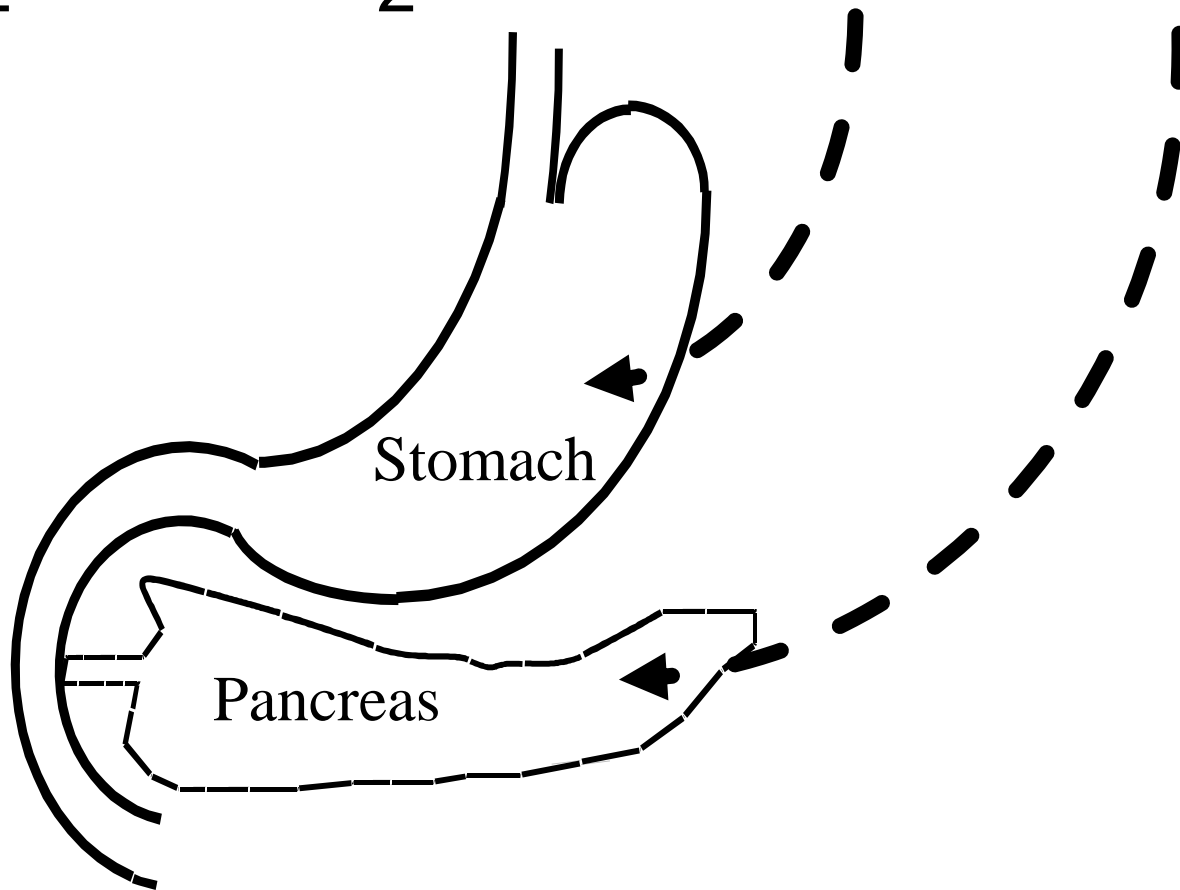


Acid base Homeostasis

Metabolic



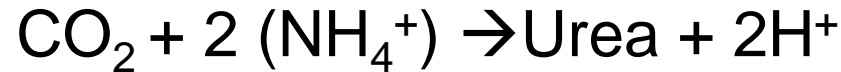
Acid-Base in the G-I Tract





Protein Metabolism and H⁺ Homeostasis

- amino-acids are catabolised to CO₂ ,H₂O ,NH₄⁺ ,HCO₃⁻



- Urea synthesis is energy dependent
- protein catabolism produces a slight excess of NH₄⁺



Miscellaneous sources of H⁺

- Extra H⁺ is derived from
 - sulphur containing a/a
 - dietary organic acids
 - organic acid metabolic products
- Excess NH₄⁺ is excreted in urine



FIXED ACIDS

Cysteine, methionine \rightarrow sulfate + H +

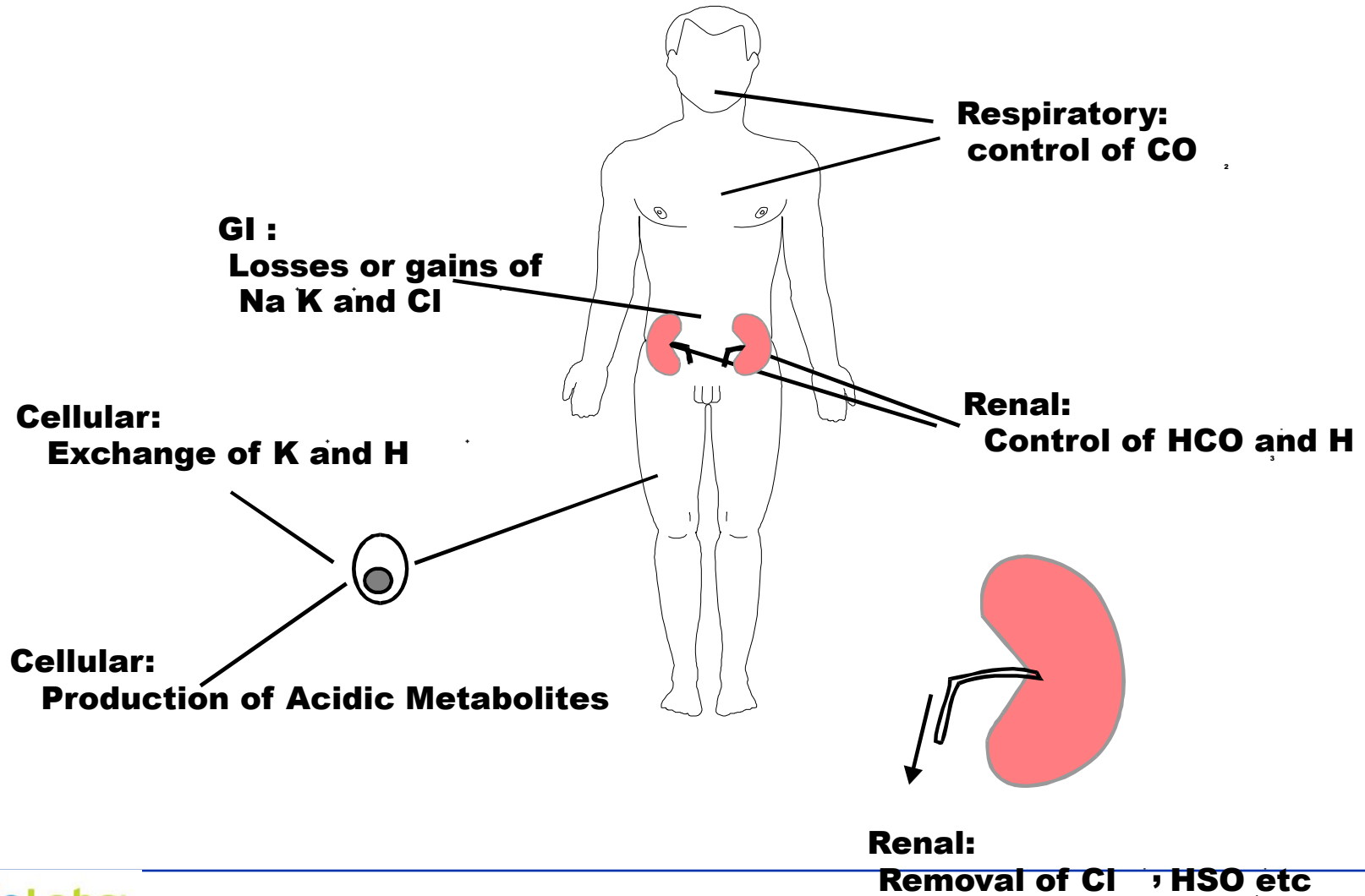
Phospholipids \rightarrow phosphate + H +

Glucose $\xrightarrow{\text{anaerobic}}$ lactate + H +

ABOUT 50 mmoles
PRODUCED AND EXCRETED DAILY

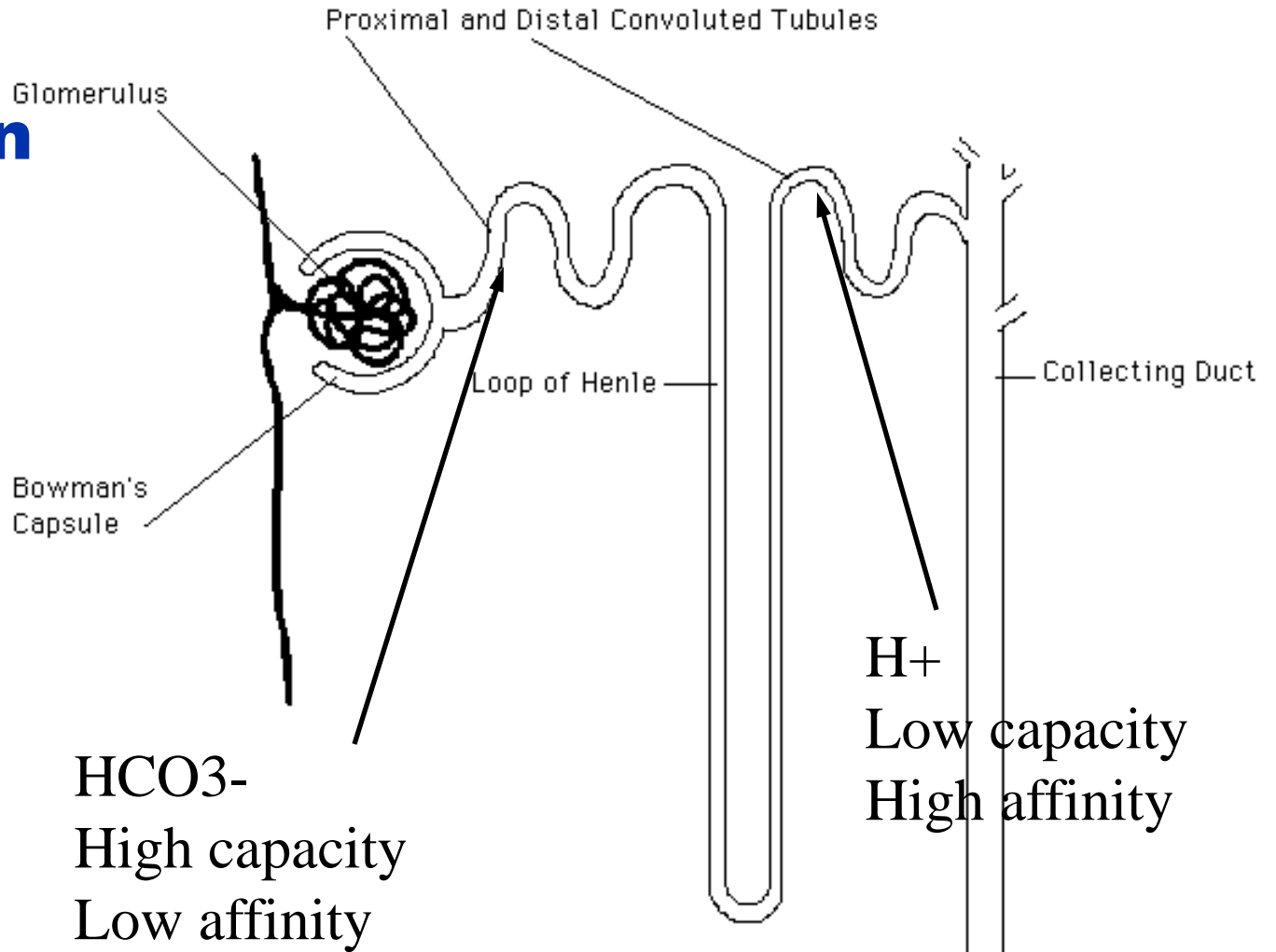


Acid - Base Homeostatic Mechanisms





Nephron

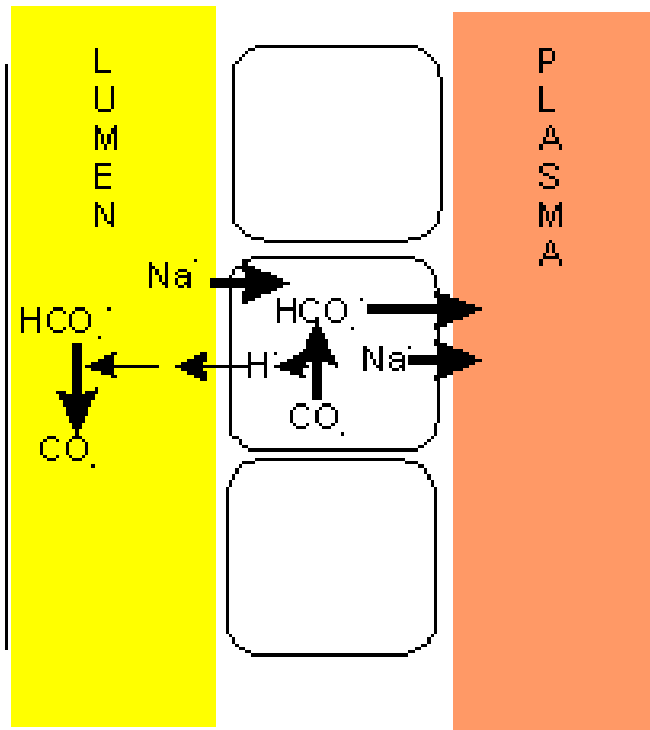




Renal Control of Acid Base Balance

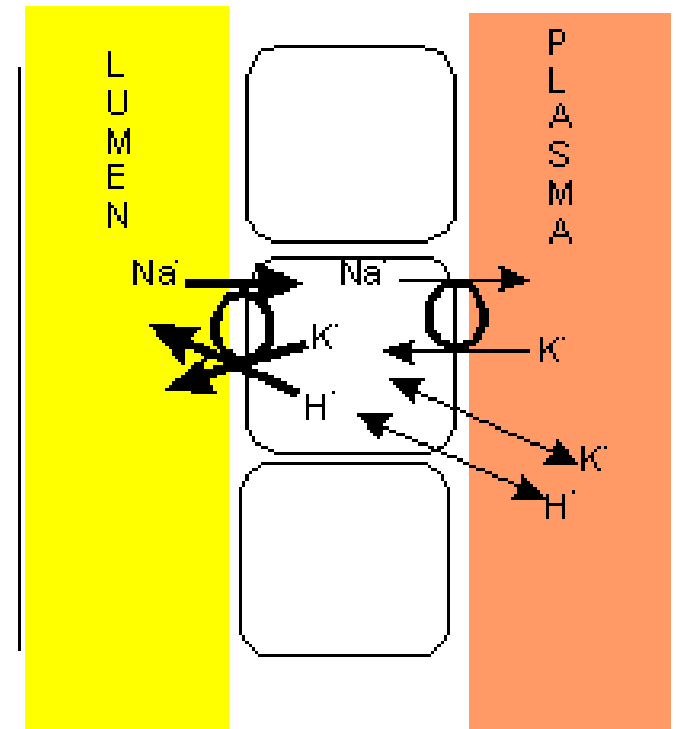
Proximal Tubule

Bicarbonate Reabsorption



Distal Tubule

Proton Excretion





Renal Ammoniogenesis

Increased NH_4^+ excretion in acidosis

glutamine \rightarrow 2 oxo-glutarate + NH_4^+

glutaminase stimulated over hours/days

ascending loop of Henle

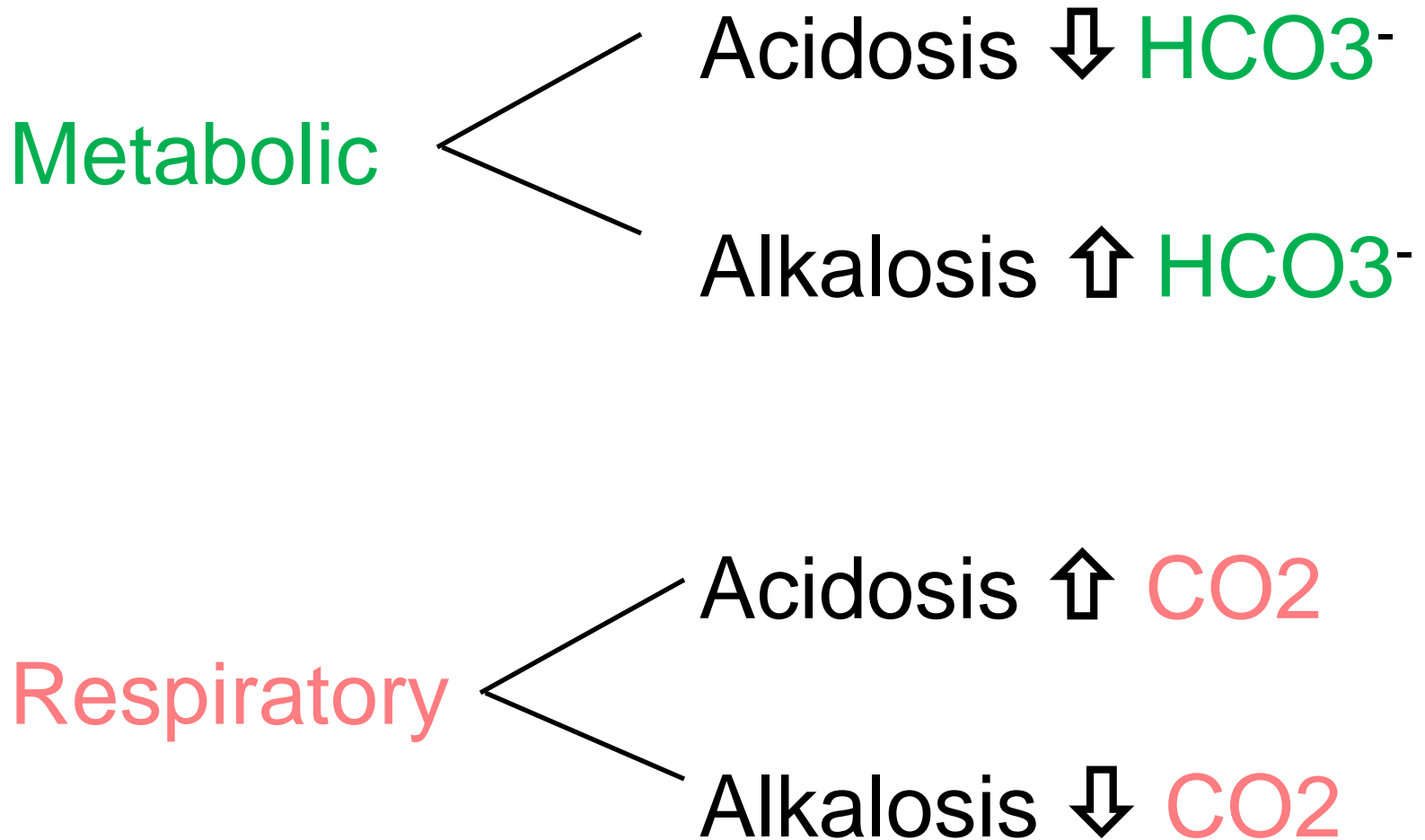
NH_4^+ absorbed and NH_3 excreted into collecting duct

H^+ secreted back into Loop of Henle and reclaims

HCO_3^-



Acid-Base Pathology





Metabolic Acidosis



- General Causes
 - Increased H^+ formation
 - Reduced renal H^+ excretion
 - Loss of bicarbonate



Metabolic Alkalosis



- General Causes

- Renal increased H⁺ excretion in Hypokalemia
- Administration of HCO₃⁻



Respiratory Acidosis

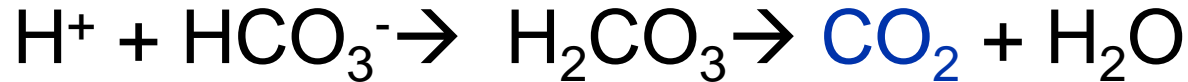


CO₂ retention due to

- inadequate ventilation
- obstructive lung disease
- inadequate perfusion



Respiratory Alkalosis



CO₂ reduction due to

-excessive ventilation

- CNS
 - Encephalopathy
 - Local Acidosis of Respiratory Center
- Pneumonia & other obstructive lung diseases
 - **Paradoxical?**



Metabolic acidosis - causes

Increased H^+ formation

Acid ingestion

Reduced renal H^+ excretion

Loss of bicarbonate



ANION GAP

Na ⁺ 140	Cl ⁻ 105
	HCO ₃ ⁻ 22
K ⁺ 4	ANION GAP

$$\text{ANION GAP} = [\text{Na}^+ + \text{K}^+] - [\text{Cl}^- + \text{HCO}_3^-] = 17$$



Diabetic keto-acidosis

- Hyperglycemia, osmotic diuresis
- Hyperketonemia, increased FFA
 - lead to acidosis



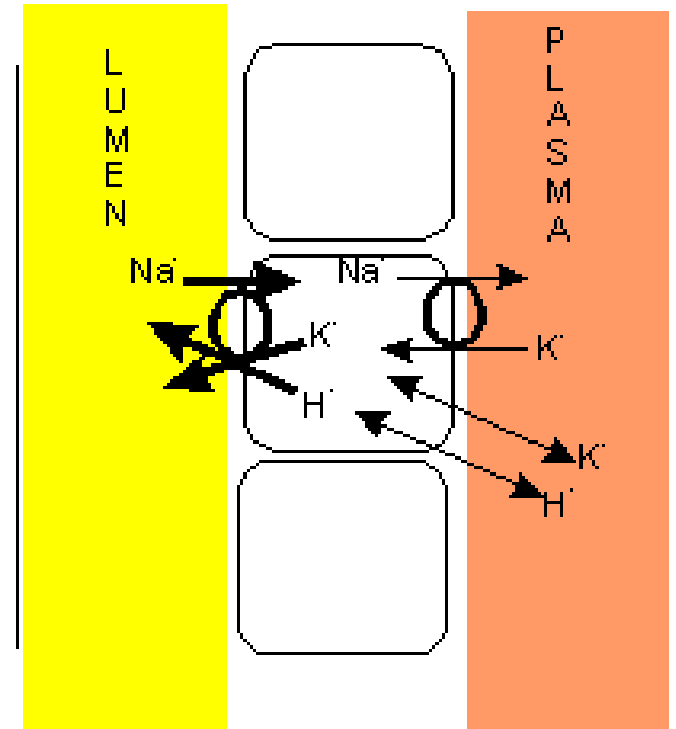
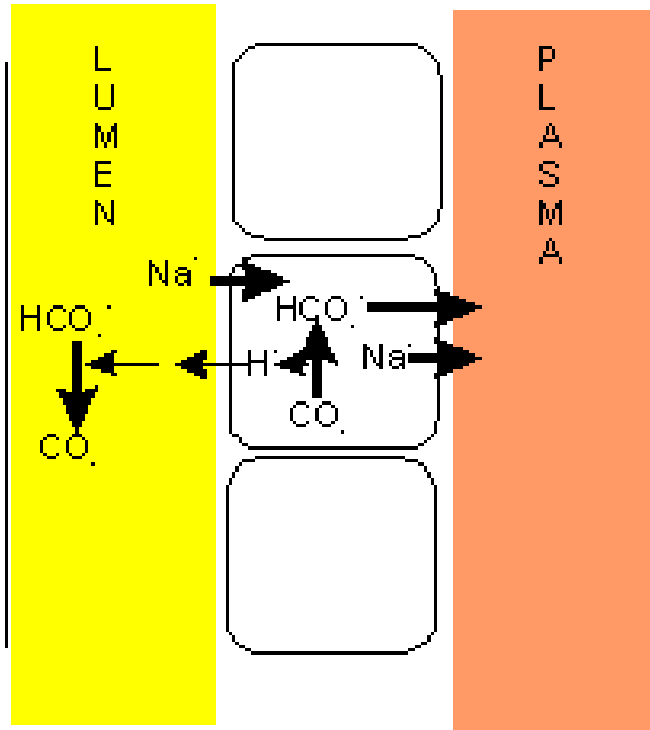
Renal Control of Acid Base Balance

Proximal Tubule

Bicarbonate Reabsorption

Distal Tubule

Proton Excretion





Normal Anion Gap Metabolic Acidosis

Normal Anion Gap (Hyperchloremic)

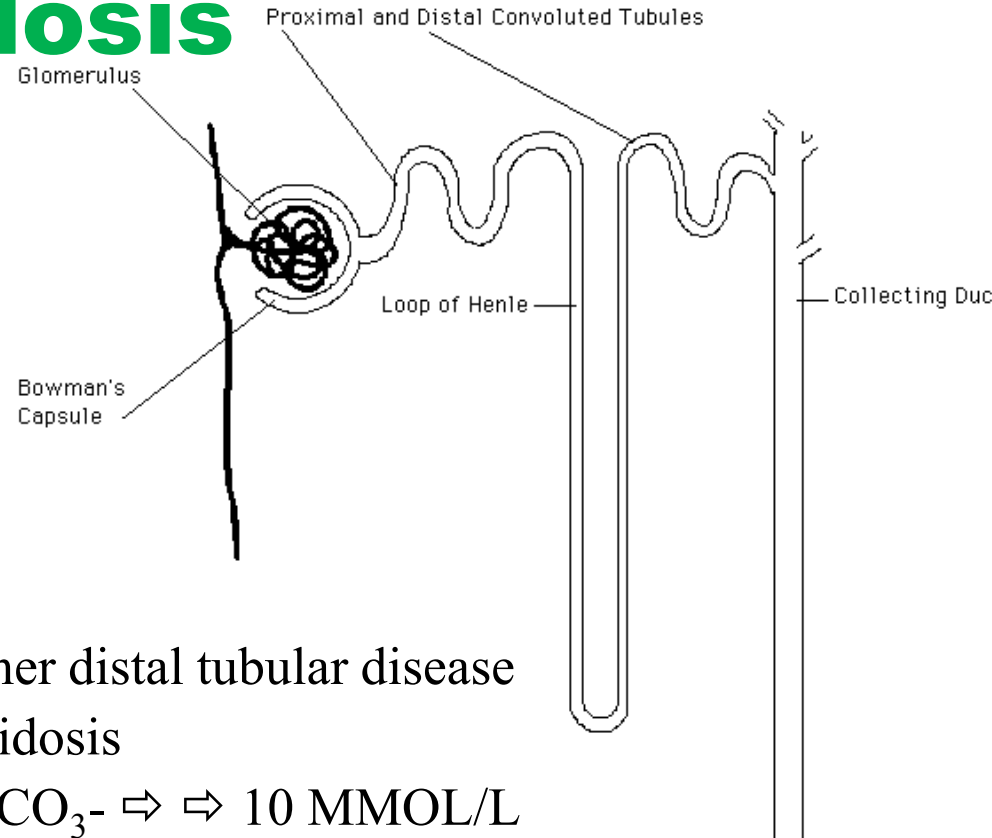
↑ Chloride Load or Retention

- Ammonium Chloride ingestion ↑ K⁺
- Obstructive Uropathy ↑ K⁺
- Ureterosigmoidostomy ↓ K⁺
- Obstructive Ileal Bladder ↓ K⁺



Normal Anion Gap

Metabolic Acidosis

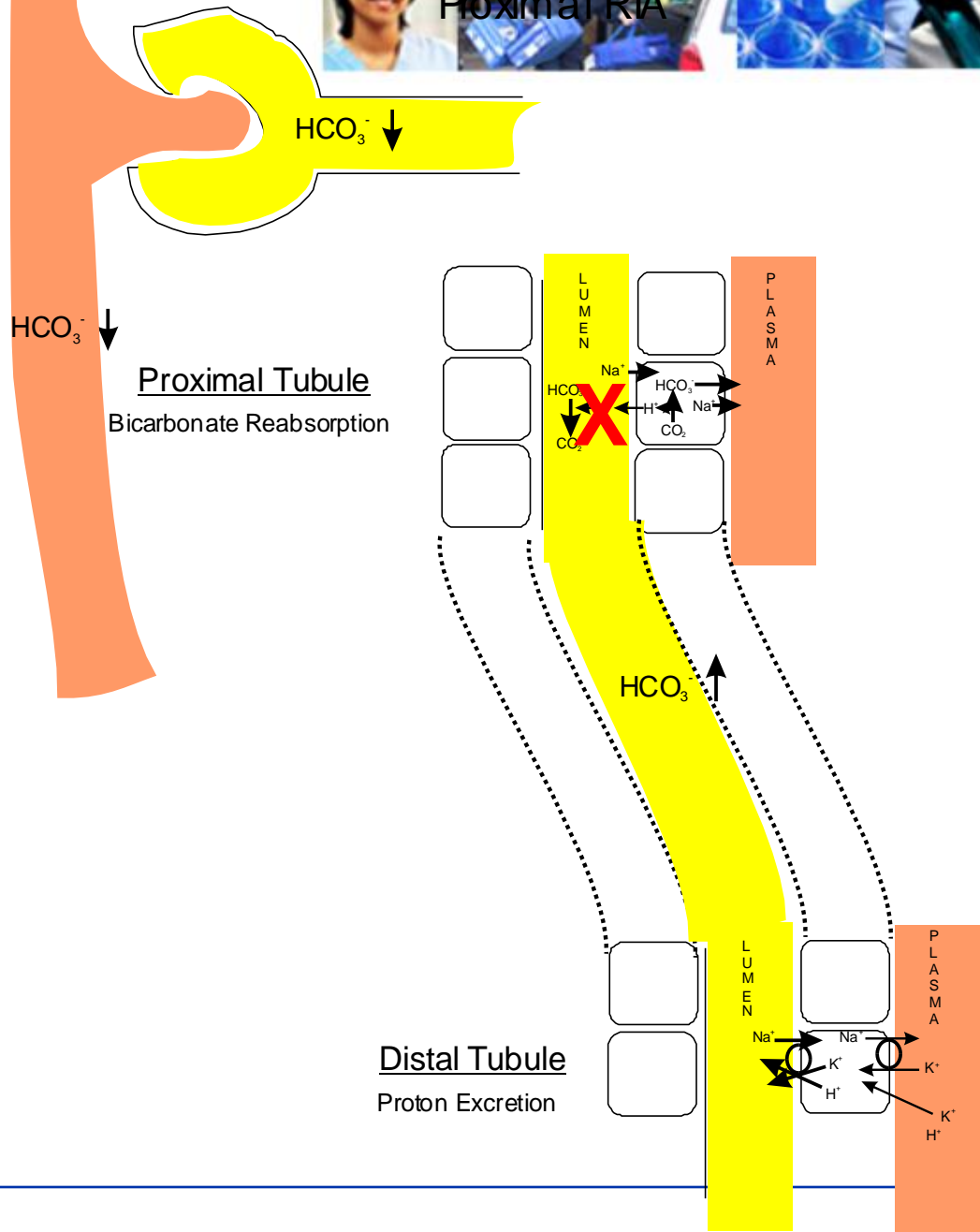


- \uparrow Bicarbonate Loss
 - \downarrow Aldosterone
 - K^+ sparing diuretics
 - interstitial nephritis or other distal tubular disease
 - proximal renal tubular acidosis
 - self limiting when $HCO_3^- \Rightarrow \Rightarrow 10 \text{ MMOL/L}$
 - distal renal tubular acidosis
 - UNLIMITED $\Rightarrow \Rightarrow$ severe acidosis

BCSCLS Blood Gases Acid Base



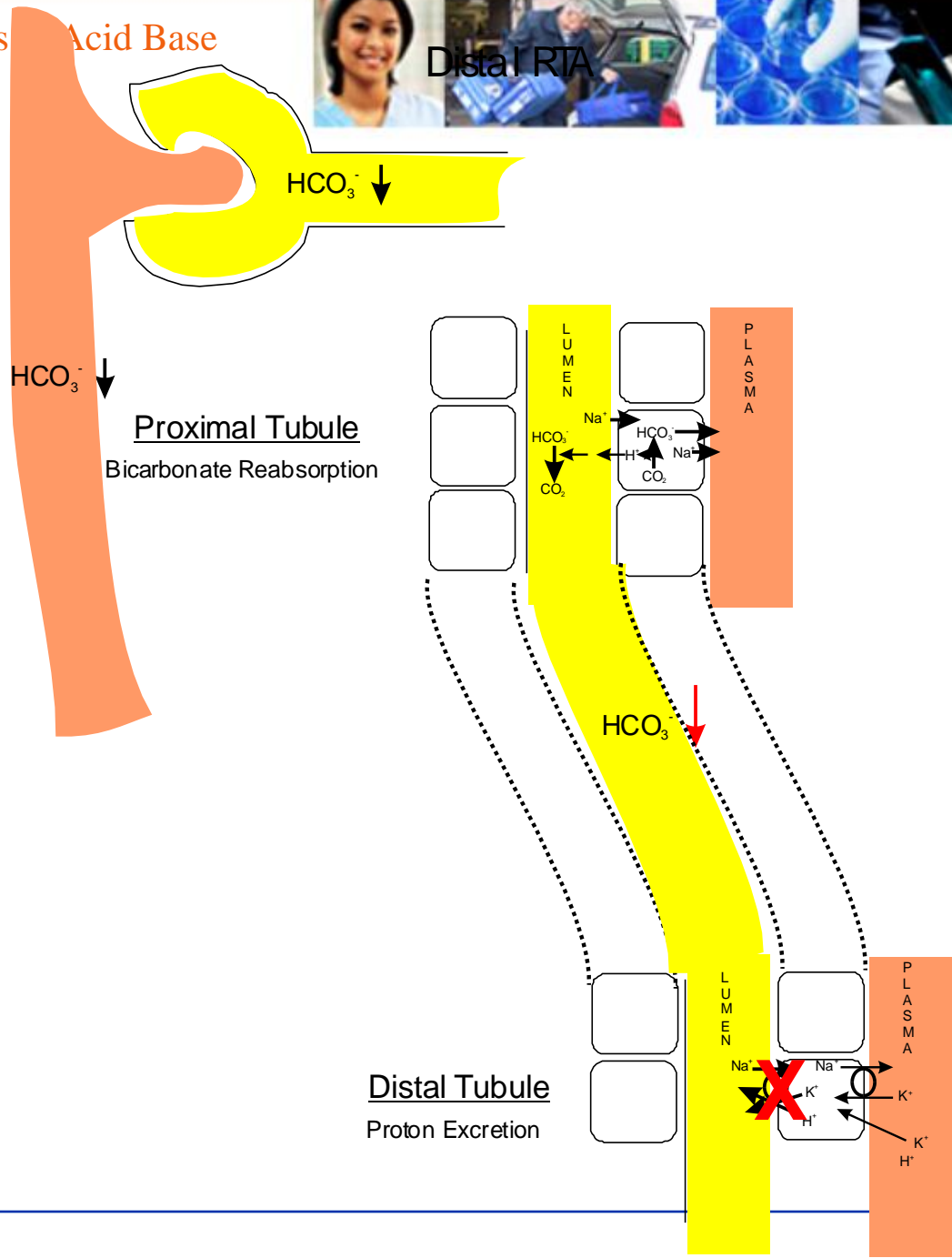
Proximal RTA



BCSCLS Blood Gases Acid Base



Distal RTA





Renal Failure

REDUCED NUMBER OF NEPHRONS

→ Increased volume load per nephron

Increased bicarbonate loss

Reduced NH_4^+ excretion

- NH_4^+ to liver for urea + H^+ synthesis
- reduced NH_4^+ derived from glutamine



Post Hypocapnic Acidosis

Hypocapnia (\downarrow CO₂) \rightarrow Alkalosis

Compensation is Metabolic Acidosis

- Loss of K⁺ in Distal Tubule

Post Hypocapnia (N – CO₂)

- residual Acidosis from compensation



Causes of Metabolic Alkalosis

Chloride Depletion

- Gastric - vomiting
 - some severe diarrhoea

Potassium Loss

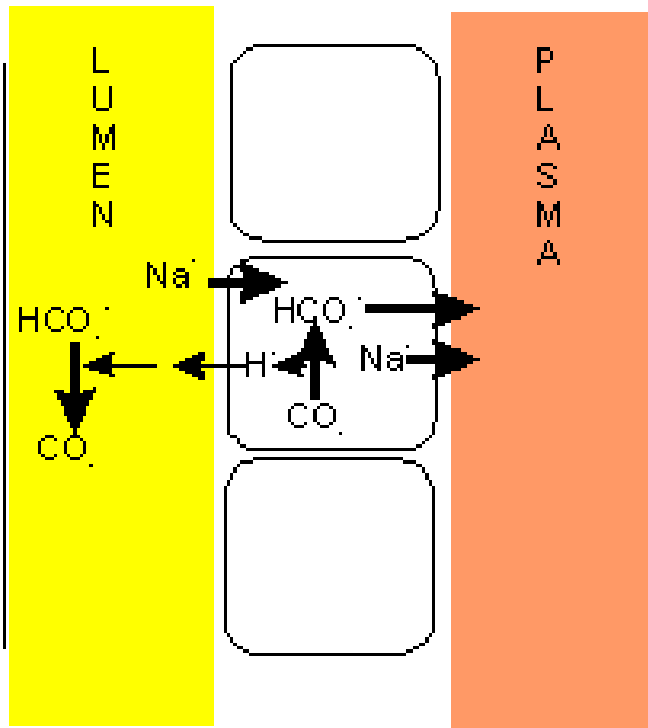
- Hyperaldosteronism
- Diuretics
- Bicarbonate administration



Renal Control of Acid Base Balance

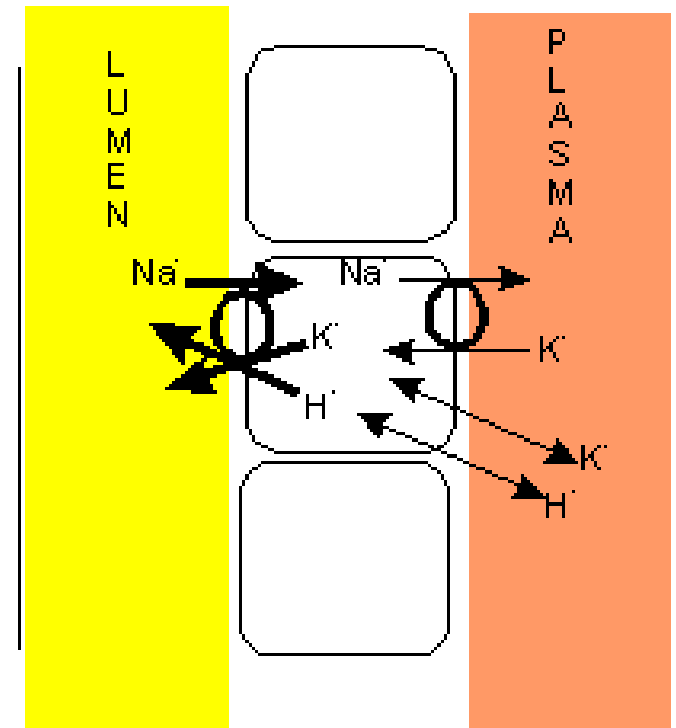
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Bicarbonate Reabsorption



Distal Tubule

Proton Excretion





Compensation

Objective pH → Normal

- Seldom achieve full compensation
- **Never** achieve over compensation
- Blood Gases identify
 - 1^o Disorder
 - Acidosis or Alkalosis
 - Respiratory or Metabolic
 - Compensation
 - Appropriate
 - Inappropriate
 - Other disorder
 - 2^o disturbance
 - or
 - **“Mixed acid-base disturbance”**



Compensation - Mechanisms

Respiration \uparrow

$\rightarrow \downarrow p\text{CO}_2 \downarrow \text{pH}$

Renal Mechanisms

- Tubule

$\uparrow \text{HCO}_3^- \text{ excretion} \rightarrow \downarrow \text{HCO}_3^- \downarrow \text{pH}$

$\uparrow \text{H}^+ \text{ excretion} \rightarrow \uparrow \text{pH}$

$\text{NH}_4^+ \text{ formation} \rightarrow \uparrow \text{pH}$



Blood Gases

Measurement of:

- pH
- PCO₂
- PO₂

Calculation of [HCO₃⁻]



Acid-Base Pathology

Metabolic

Acidosis ↓ HCO_3^-

Alkalosis ↑ HCO_3^-

Respiratory

Acidosis ↑ CO_2

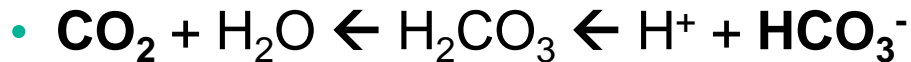
Alkalosis ↓ CO_2



Respiratory Alkalosis (**Acute**)

Compensation - **Metabolic Acidosis**

Acute (10 minutes)



↖:Buffer

$\text{HCO}_3^- \downarrow$

- Limit of compensation



- If **Measured HCO_3^-** < 18 \Rightarrow 1° Metabolic Acidosis
- If **Measured HCO_3^-** > 23 \Rightarrow 1° Metabolic Alkalosis



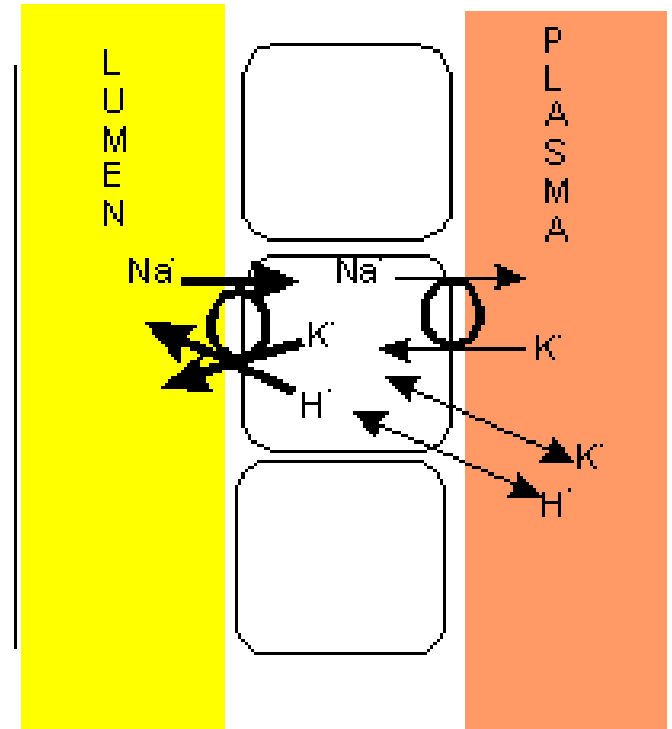
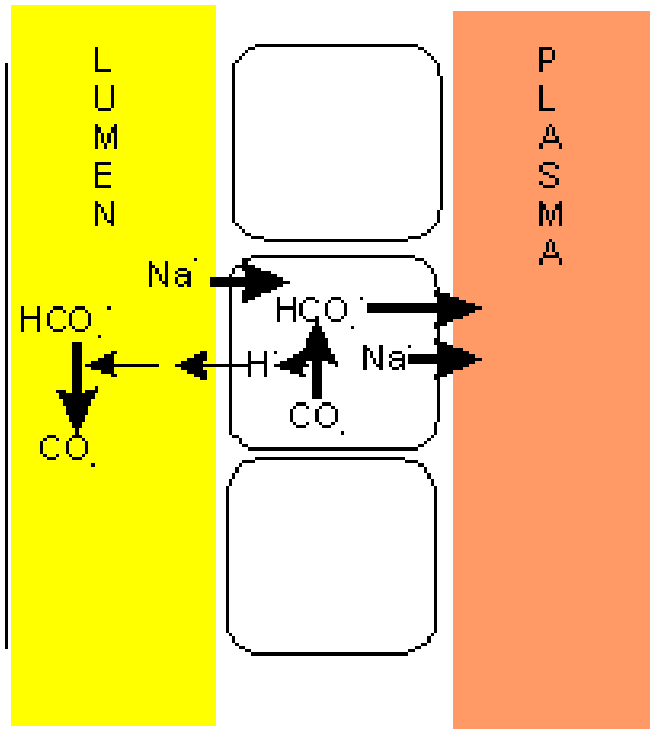
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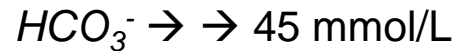


Respiratory Acidosis (Chronic)

Compensation - **Metabolic Alkalosis**

Chronic (24- 96 hr)

- Renal Reabsorption of Bicarbonate $\text{HCO}_3^- \uparrow$
- Limit of compensation



- If **Measured HCO_3^-** > 45 \Leftrightarrow 1° Metabolic Alkalosis
- Normal Compensation
 - $\text{HCO}_3^- = 0.43 \times \text{pCO}_2 + 7.6 \pm 2$



Respiratory Alkalosis: Chronic

Compensation - **Metabolic Acidosis**

Chronic (24- 96 hr can fully compensate)

- **Renal Loss of Bicarbonate**



- Limit of compensation



- If **Measured HCO_3^-** < 12 \Leftrightarrow 1^o Metabolic Acidosis
- Normal Compensation
 - $\text{HCO}_3^- = 0.5 \times \text{pCO}_2 + 5$



Metabolic Acidosis

Compensation - Respiratory Alkalosis

- (H⁺ stimulates medulla oblongata)

Chronic (12 - 24 hr)

- $p\text{CO}_2 \downarrow$
- Limit of compensation

$$p\text{CO}_2 \rightarrow \rightarrow 10 \text{ mmHg}$$

- If **Measured** $p\text{CO}_2 < 10 \Rightarrow 1^\circ$ Respiratory Alkalosis
- Normal Compensation
 - $p\text{CO}_2 = 1.54 \times \text{HCO}_3^- + 8.4 \pm 1.1$



Metabolic Alkalosis

Compensation - **Respiratory Acidosis**

Chronic Irregular (12 - 24 hr)

- $p\text{CO}_2 \uparrow$
- Limit of compensation

$p\text{CO}_2 \rightarrow \rightarrow 60 \text{ mmHg}$

- If **Measured** $p\text{CO}_2 > 60 \Rightarrow 1^\circ$ Respiratory Acidosis
- Normal Compensation
 - $p\text{CO}_2 = 0.9 \times \text{HCO}_3^- + 9$



Normal Compensation Limits for Acid-base Disturbances				
Primary Disturbance		Lower Limit	Upper Limit	Normal Compensation
Respiratory Acidosis	Acute	$\text{HCO}_3^- = 23$	$\text{HCO}_3^- = 32$	
	Chronic 24-96 h		$\text{HCO}_3^- = 45$	$\text{HCO}_3^- = 0.43 \times \text{pCO}_2 + 7.6 \pm 2$
Respiratory Alkalosis	Acute	$\text{HCO}_3^- = 18$	$\text{HCO}_3^- = 23$	
	Chronic	$\text{HCO}_3^- = 14$		$\text{HCO}_3^- = 25 - (40 - \text{pCO}_2) \times 0.5 \pm 1$
Metabolic Acidosis	Chronic 12-24 h	$\text{pCO}_2 = 10$		$\text{pCO}_2 = 1.5 \times \text{HCO}_3^- + 8.4 \pm 1$
Metabolic Alkalosis	Chronic		$\text{pCO}_2 = 60$	$\text{pCO}_2 = 0.9 \times \text{HCO}_3^- + 9$



Acid base Case Study

45 year old asthmatic male presents to the emergency department in *status asthmaticus* (profound breathing difficulty) which has worsened over the past 12 hrs.

Plasma creatinine, potassium, sodium and chloride were normal.

Blood gases were as follows:

- pH 7.21 (7.35 – 7.45)
- PCO₂ 56 (34 – 45)mmHg
- PO₂ 75 (80 – 110)mmHg
- HCO₃⁻ 21 (20 – 30) mM



Acid base Case Study

A 61 year old female came to the emergency room with a history of a vomiting for the past 36 hours. In addition to her gastrointestinal problem she was quite tachypneic which raised the possibility that she also had aspiration pneumonia. On admission, her blood gas values were:

<u>Test</u>	<u>Results</u>	<u>Reference range</u>
pH	7.36	7.35 - 7.45
PCO ₂	62	35 - 45
PO ₂	55	80 - 110
HCO ₃	40	22 - 32



Acid base Case Study

14 Year old boy passed out in his bedroom. He was taken to the emergency room .

On Admission - he was difficult to arouse
Plasma creatinine, potassium, sodium and chloride were normal.

Blood gases were as follows:

- pH 7.40 (7.35 – 7.45)
- PCO₂ 20 (34 – 45)mmHg
- PO₂ 112 (80 – 110)mmHg
- HCO₃⁻ 14 (20 – 30) mM
- Anion Gap 22 (7 – 17) mM



Acid base Case Study

A newborn boy developed cyanosis in the labour delivery room. Arterial blood gas specimens were obtained and then the baby was started on 100 percent oxygen. The blood gas results were as follows:

<u>On Room Air</u>	<u>Results</u>	<u>Reference range</u>
pH	7.29	7.35 - 7.45
PCO ₂	72	35 - 45
PO ₂	35	55 - 80
HCO ₃	34	22 - 32

<u>On 100% O₂</u>		
pH	7.28	7.35 - 7.45
PCO ₂	70	35 - 45
PO ₂	33	55 - 80
HCO ₃	32	22 - 32

What do these results tell us concerning the cause of his cyanosis.



Acid - Basics

ie The take home message

- how pH homeostasis is maintained
- the common causes of acid base disturbances
- compensation mechanisms
- Use blood gas and electrolyte results to determine...
 - the primary pathology
 - acidosis or alkalosis
 - respiratory or metabolic
 - if there is appropriate compensation or a mixed acid base disorder