A microscopic image showing a dense network of neurons with green-stained cell bodies and processes, and some red-stained structures.

PowerPoint® Lecture  
Presentation

PRINCIPLES OF  
**HUMAN  
PHYSIOLOGY**

SIXTH EDITION

CINDY L. STANFIELD

CHAPTER **19c**

The Urinary  
System: Fluid  
and Electrolyte  
Balance

# Chapter Outline

19.1 The Concept of Balance

19.2 Water Balance

19.3 Sodium Balance

19.4 Potassium Balance

19.5 Calcium Balance

19.6 Interactions Between Fluid and Electrolyte  
Regulation

19.7 Acid-Base Balance

# 19.6 Interactions Between Fluid and Electrolyte Balance

- Increase in solute reabsorption increases osmotic gradient for water reabsorption
- ADH increases the number of sodium channels in the apical membrane of principal cells
- Angiotensin II increases ADH secretion
- ANP decreases ADH secretion

# Interactions Between Fluid and Electrolyte Balance

- Physiological response to hemorrhage
  - Decrease in blood volume → decrease in MAP
  - Neural control of heart and vasculature
    - Baroreceptor reflex
  - Hormonal control of blood volume
    - ADH
    - Renin-angiotensin-aldosterone system

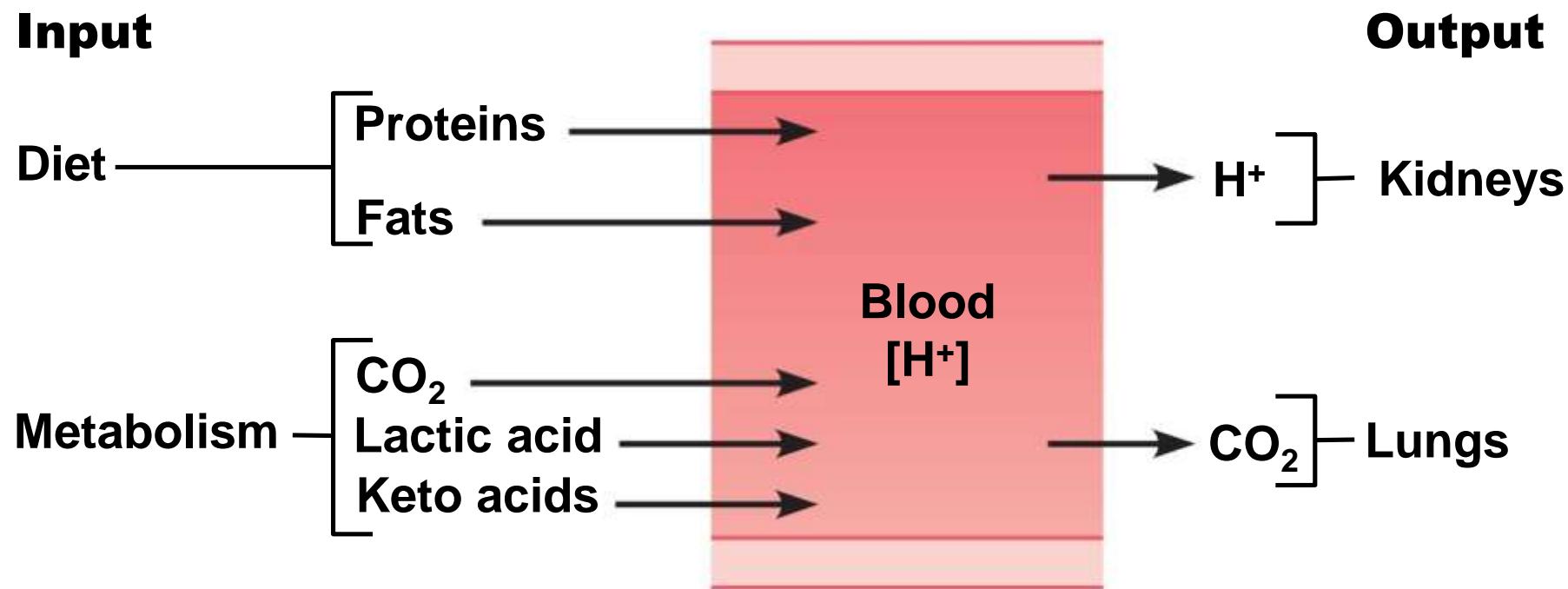
## 19.7 Acid-Base Balance

- Normal pH of arterial blood: 7.35–7.45
  - $\text{pH} < 7.35$  = acidosis
  - $\text{pH} > 7.45$  = alkalosis

# Acid-Base Balance

- Complications with acid-base disturbance
  - Conformation change in protein structure
  - Changes in excitability of neurons
  - Changes in potassium balance
  - Cardiac arrhythmias
  - Vasodilation

Figure 19.24 Inputs and outputs of acid to the blood.



# Sources of Acid-Base Disturbances

- Respiratory disturbances
  - Carbon dioxide is a source of acid

CA



- Normal  $P_{\text{CO}_2}$  arterial blood = 40 mm Hg
- Sources of  $\text{CO}_2$ : metabolism
- Output of  $\text{CO}_2$ : through respiratory system
- Increased plasma  $[\text{CO}_2]$  → respiratory acidosis
- Decreased plasma  $[\text{CO}_2]$  → respiratory alkalosis

- Respiratory acidosis can arise from a break in any one of these links:
- For example, it can be caused from depression of the respiratory center through drugs or metabolic disease, or from limitations in chest wall expansion due to neuromuscular disorders or trauma.
- It can also arise from pulmonary disease, cardiogenic pulmonary edema, aspiration of a foreign body or vomitus, pneumothorax and pleural space disease, or through mechanical hypoventilation.

- Respiratory alkalosis can be due to either direct or reflex hypoxic stimulation of the respiratory center, to pulmonary disease, or to excessive mechanical ventilation.

# Sources of Acid-Base Disturbances

- Metabolic acidosis
  - Decreased pH through something other than CO<sub>2</sub>
    - High-protein diet
    - High-fat diet
    - Heavy exercise
    - Severe diarrhea (loss of bicarbonate)
    - Renal dysfunction

# Sources of Acid-Base Disturbances

- Metabolic alkalosis
  - Increased pH through something other than CO<sub>2</sub>
    - Excessive vomiting (loss of hydrogen ions)
    - Consumption of alkaline products (baking soda)
    - Renal dysfunction

# Defense Mechanisms Against Acid-Base Disturbances

- Three lines of defense
  - Buffering of hydrogen ions
  - Respiratory compensation
  - Renal compensation

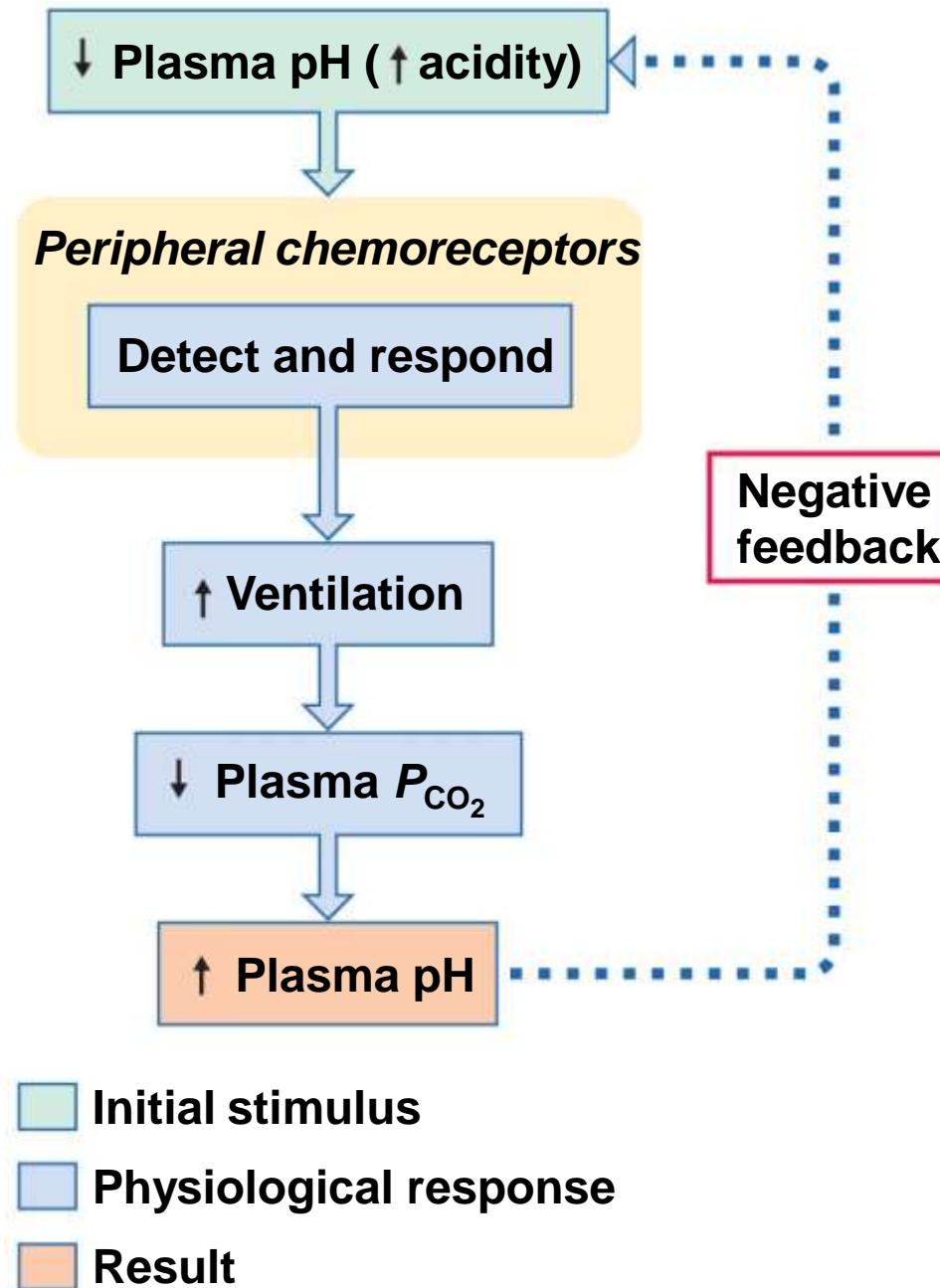
# Defense Mechanisms Against Acid-Base Disturbances

- Buffering
  - Quickest defense against changes in pH
  - Most important ECF buffer = bicarbonate
    - $\text{HCO}_3^- + \text{H}^+ \rightleftharpoons \text{H}_2\text{CO}_3$
  - ICF buffers
    - Proteins:  $\text{Protein}^- + \text{H}_+ \rightleftharpoons \text{H}\bullet\text{Protein}$
    - Phosphates:  $\text{HPO}_4^{2-} + \text{H}^+ \rightleftharpoons \text{H}_2\text{PO}_4^-$

# Defense Mechanisms Against Acid-Base Disturbances

- Respiratory compensation
  - Second line of defense
    - Takes minutes to have an effect
  - Regulates pH by varying ventilation
    - Increased ventilation → decreased CO<sub>2</sub>
    - Decreased ventilation → increased CO<sub>2</sub>

Figure 19.25 The mechanism by which decreases in plasma pH increase ventilation.



# Defense Mechanisms Against Acid-Base Disturbances

- Renal compensation
  - Third line of defense
  - Takes hours to days
  - Regulates excretion of hydrogen ions and bicarbonate in urine
  - Regulates synthesis of new bicarbonate in renal tubules
- Effects of increased acidity
  - Increased secretion of hydrogen ions
  - Increased reabsorption of bicarbonate
  - Increased synthesis of new bicarbonate

# Defense Mechanisms Against Acid-Base Disturbances

- Renal handling of hydrogen and bicarbonate ions
  - Proximal tubule
    - Bicarbonate reabsorption coupled to hydrogen ion secretion
  - Distal tubule and collecting duct
    - Secretion of hydrogen ions coupled to synthesis of new bicarbonate ions

Figure 19.26 Bicarbonate reabsorption and hydrogen ion secretion in the proximal tubule.

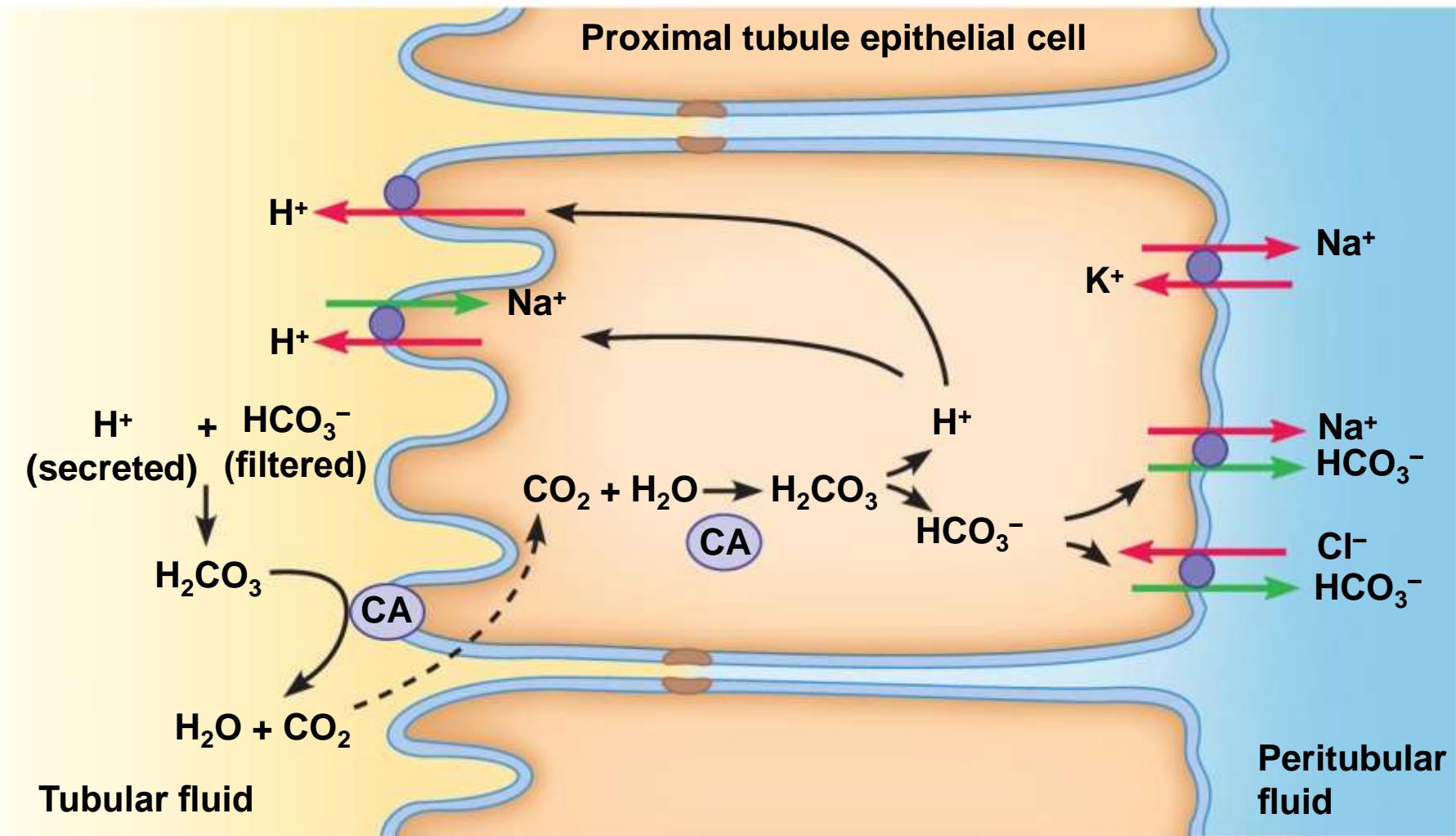
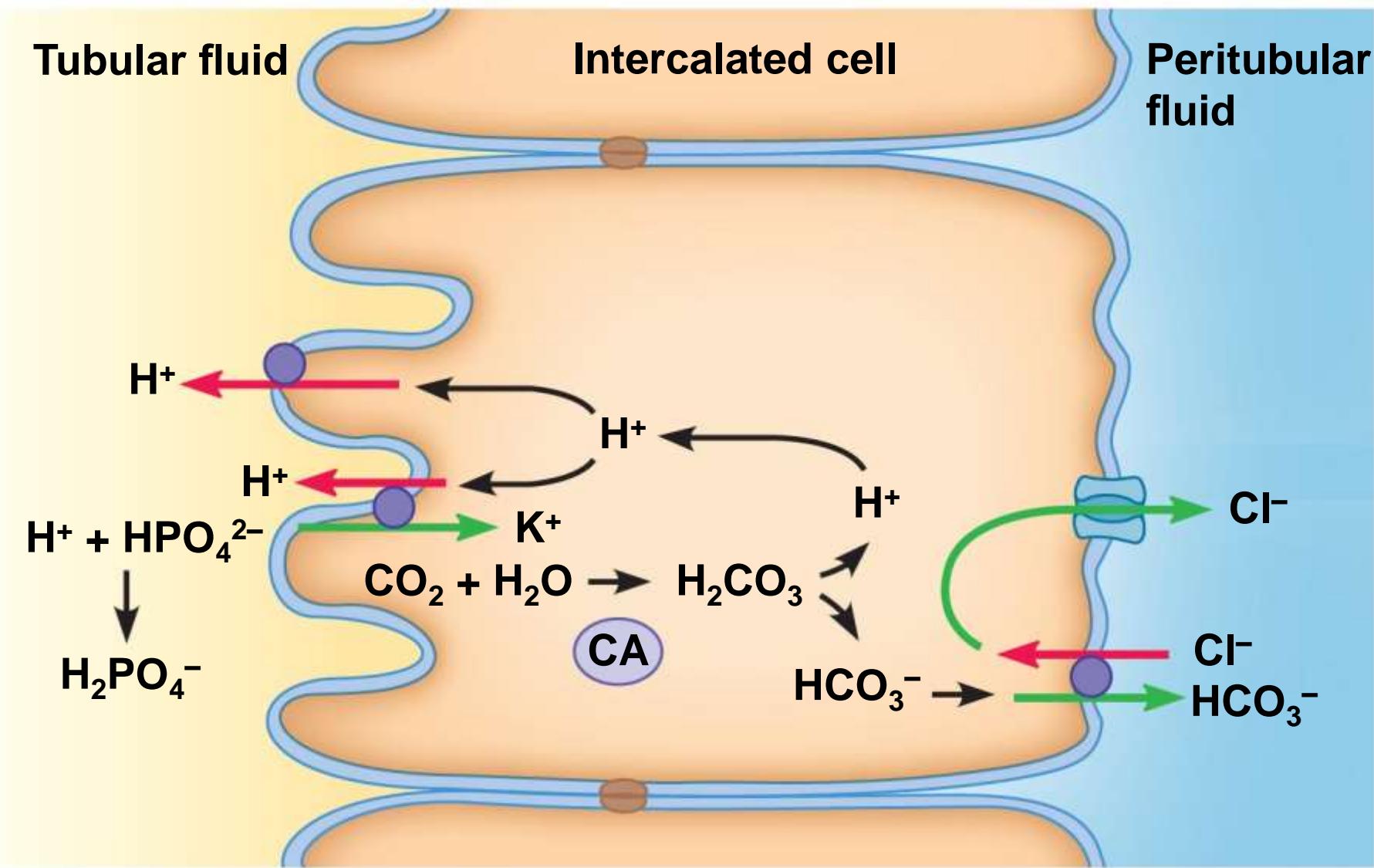


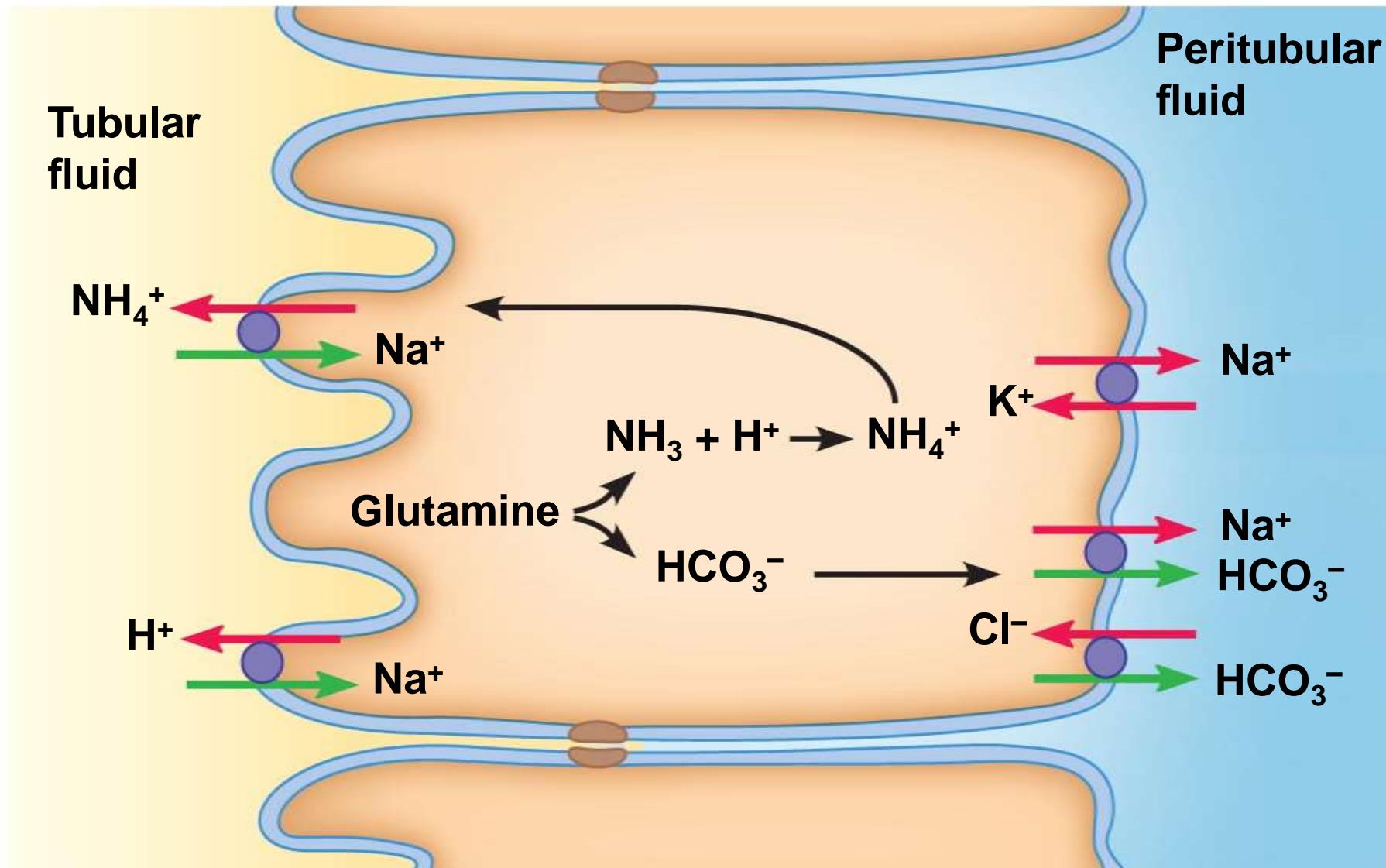
Figure 19.27 Bicarbonate synthesis and hydrogen ion secretion by intercalated cells of the distal tubule and collecting duct.



# Defense Mechanisms Against Acid-Base Disturbances

- Glutamine in renal compensation
  - Regulation of hydrogen ion secretion, bicarbonate reabsorption, and bicarbonate synthesis by kidneys is usually sufficient
  - Severe acidosis
    - Glutamine metabolism produces new bicarbonate and secretes hydrogen in the form of ammonium

Figure 19.28 Bicarbonate production and hydrogen secretion by glutamine metabolism in the proximal tubule.



# Compensation for Acid-Base Disturbances

- Henderson-Hasselbalch equation

$$\text{pH} = 6.1 + \log \frac{[\text{HCO}_3^-]}{[\text{CO}_2]}$$

- For pH = 7.4,  $[\text{HCO}_3^-]/[\text{CO}_2] = 20:1$
- Acidosis:  $[\text{HCO}_3^-]/[\text{CO}_2] < 20:1$
- Alkalosis:  $[\text{HCO}_3^-]/[\text{CO}_2] > 20:1$
- Kidneys regulate  $\text{HCO}_3^-$
- Lungs regulate  $\text{CO}_2$

# Compensation for Acid-Base Disturbances

- Respiratory acidosis
  - Cause: hypoventilation
  - Increased  $\text{CO}_2$  → increased  $\text{H}^+$
  - Compensation: renal
    - Increased  $\text{H}^+$  secretion
    - Increased  $\text{HCO}_3^-$  reabsorption

# Compensation for Acid-Base Disturbances

- Respiratory alkalosis
  - Cause: hyperventilation
  - Decreased  $\text{CO}_2 \rightarrow$  decreased  $\text{H}^+$
  - Compensation: renal
    - Decreased  $\text{H}^+$  secretion
    - Decreased  $\text{HCO}_3^-$  reabsorption

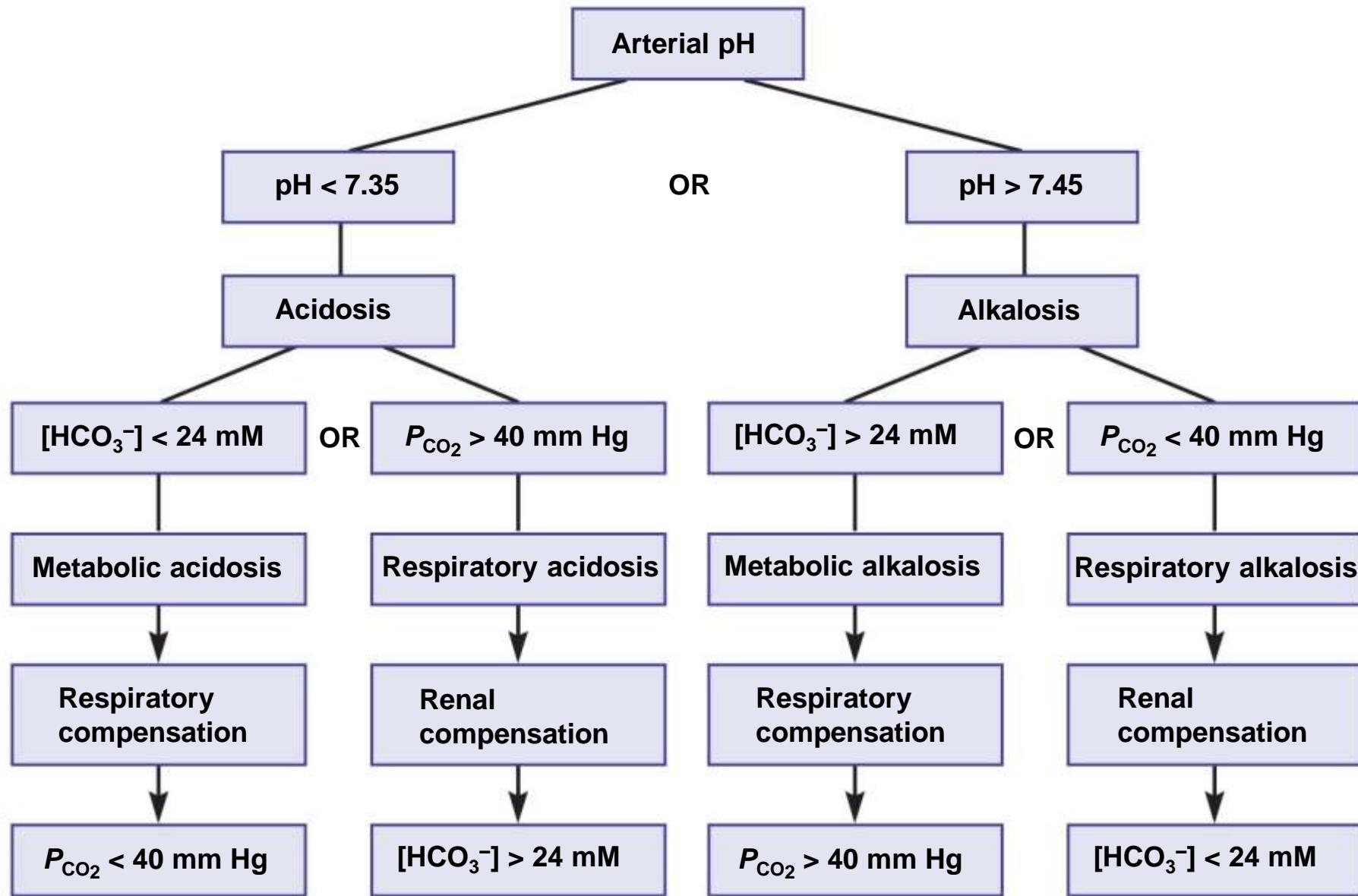
# Compensation for Acid-Base Disturbances

- Metabolic acidosis
  - Cause: increased  $H^+$  independent of  $CO_2$
  - Compensation: respiratory and renal (unless renal problem)
  - Respiratory compensation
    - Increased ventilation → decreased  $CO_2$
  - Renal compensation
    - Increased  $H^+$  secretion
    - Increased  $HCO_3^-$  reabsorption
    - Increased synthesis of new bicarbonate

# Compensation for Acid-Base Disturbances

- Metabolic alkalosis
  - Cause: decreased H<sup>+</sup> independent of CO<sub>2</sub>
  - Compensation: respiratory and renal (unless renal problem)
  - Respiratory compensation
    - Decreased ventilation → increased CO<sub>2</sub>
  - Renal compensation
    - Decreased H<sup>+</sup> secretion
    - Decreased HCO<sub>3</sub><sup>-</sup> reabsorption
    - Decreased synthesis of new bicarbonate

Figure 19.29 Summary of acid-base disturbances and compensation.



- The H-H equation defines the relationship between pH and the ratio of salt (bicarbonate) and weak acid (carbonic acid):
  - $\text{HCO}_3^-(\text{bicarbonate}) + \text{H}^+ \rightleftharpoons \text{H}_2\text{CO}_3(\text{carbonic acid}) \rightleftharpoons \text{CO}_2 + \text{H}_2\text{O}$

# Henderson Hasselbalch Equation

$$\text{pH} = \text{pK}_a + \log \frac{[\text{conjugate base}]}{[\text{weak acid}]} \text{ (for weak acid)}$$

$$\text{pOH} = \text{pK}_b + \log \frac{[\text{conjugate acid}]}{[\text{weak base}]} \text{ (for weak base)}$$

(A) H-H Equation

$$\text{pH} = \text{pK}_a + \log \frac{[\text{Salt}]}{[\text{Acid}]}$$

(B) H-H Equation for Carbonic Acid

$$\text{pH of blood plasma} = 6.1 + \log \frac{[\text{HCO}_3^-]}{[\text{H}_2\text{CO}_3]}$$

$$\text{pH} = 6.1 + \log \frac{20}{1}$$

$$7.4 = 6.1 + 1.3$$

## Normal ABG Values

pH: 7.4

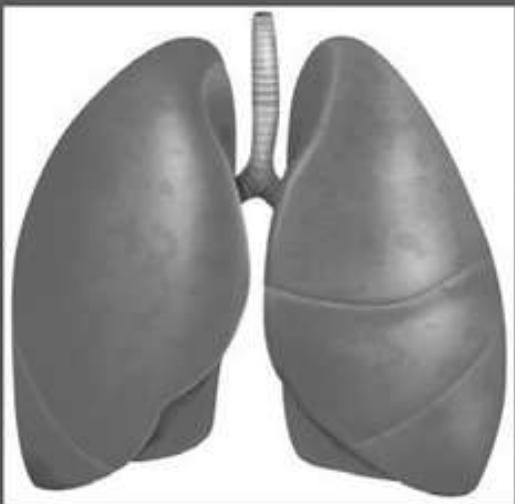
pCO<sub>2</sub>: 40 mm Hg

[H<sub>2</sub>CO<sub>3</sub>]: 1.2 mEq/L

[HCO<sub>3</sub><sup>-</sup>]: 24 mEq/L

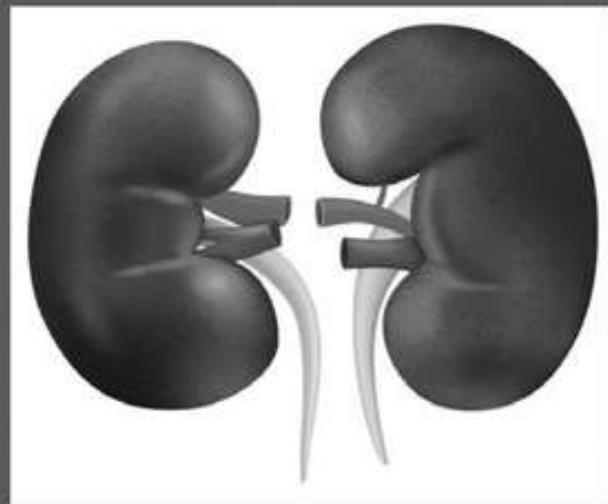


Carbonic acid-bicarbonate equilibrium illustrated as a level seesaw. The component on the lefthand side of the seesaw is [H<sub>2</sub>CO<sub>3</sub>] obtained by converting from the ABG PCO<sub>2</sub> value. The component on the righthand side of the seesaw is [HCO<sub>3</sub><sup>-</sup>] and is obtained directly from the ABG results. Note how the fulcrum is off-center toward [HCO<sub>3</sub><sup>-</sup>] because the ratio of bicarbonate to carbonic acid is 20:1.



Hyperventilation removes  $\text{H}^+$  ions, thereby increasing pH.

Hypoventilation increases  $\text{H}^+$  ions, thereby decreasing pH.



Kidneys can excrete bicarbonate ions or retain  $\text{H}^+$  ions to effectively decrease pH.

Kidneys can retain bicarbonate ions or excrete  $\text{H}^+$  ions to effectively increase pH.

## *Normal ABG Results*

<i>pH</i>	<7.35 (acidic) <b>7.35-7.45 (normal)</b> >7.45 (alkaline)
<i>PCO<sub>2</sub></i> <i>Respiratory Component</i>	<b>35-45 mm Hg</b>
<i>[HCO<sub>3</sub><sup>-</sup>]</i> <i>Metabolic Component</i>	<b>22-26 mEq/L</b>

(A) Normal ABG Values

$$\text{pH} = 6.1 + \log \frac{[24 \text{ mEq/L}]}{[.03(40 \text{ mm Hg})]}$$

$$\text{pH} = 6.1 + \log \frac{[24 \text{ mEq/L}]}{[1.2 \text{ mEq/L}]}$$

$$\text{pH} = 6.1 + \log \frac{[20]}{[1]}$$

$$7.4 = 6.1 + \log (20)$$

(B) Metabolic Acidosis ABG Values

$$\text{pH} = 6.1 + \log \frac{[17 \text{ mEq/L}]}{[.03(40 \text{ mm Hg})]}$$

$$\text{pH} = 6.1 + \log \frac{[17 \text{ mEq/L}]}{[1.2 \text{ mEq/L}]}$$

$$\text{pH} = 6.1 + \log \frac{[17]}{[1.2]}$$

$$7.25 = 6.1 + \log (14)$$

(A) Median normal values for  $[\text{HCO}_3^-]$  and  $\text{PCO}_2$  are used to demonstrate the calculation of pH using the Henderson-Hasselbalch equation. (B) Example of  $[\text{HCO}_3^-]$  and  $\text{PCO}_2$  values from a patient with an acid-base disorder. Since  $[\text{HCO}_3^-]$  is <22,  $\text{PCO}_2$  is normal, and pH is <7.35, this is a case of metabolic acidosis.

### (A) Normal ABG Values

pH: 7.4

pCO<sub>2</sub>: 40 mm Hg

[H<sub>2</sub>CO<sub>3</sub>]: 1.2 mEq/L

[HCO<sub>3</sub>]: 24 mEq/L



### (B) Metabolic Alkalosis

pH: 7.6

pCO<sub>2</sub>: 40 mm Hg

[H<sub>2</sub>CO<sub>3</sub>]: 1.2 mEq/L

[HCO<sub>3</sub>]: 37.9 mEq/L



### (D) Metabolic Acidosis

pH: 7.2

pCO<sub>2</sub>: 40 mm Hg

[H<sub>2</sub>CO<sub>3</sub>]: 1.2 mEq/L

[HCO<sub>3</sub>]: 15.1 mEq/L



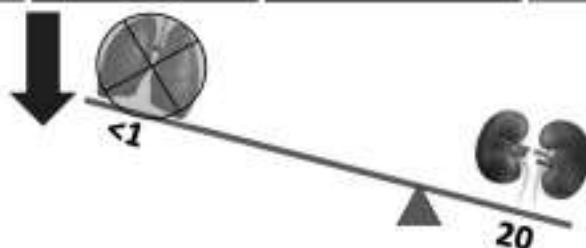
### (C) Respiratory Alkalosis

pH: 7.6

pCO<sub>2</sub>: 25.3 mm Hg

[H<sub>2</sub>CO<sub>3</sub>]: 0.759 mEq/L

[HCO<sub>3</sub>]: 24 mEq/L



### (E) Respiratory Acidosis

pH: 7.2

pCO<sub>2</sub>: 63.5 mm Hg

[H<sub>2</sub>CO<sub>3</sub>]: 1.9 mEq/L

[HCO<sub>3</sub>]: 24 mEq/L



(A) Normal ABG Values

pH: 7.4       $\text{pCO}_2$ : 40 mm Hg       $[\text{H}_2\text{CO}_3]$ : 1.2 mEq/L       $[\text{HCO}_3^-]$ : 24 mEq/L



(B) Compensated Metabolic Alkalosis

pH: 7.6  
-  
pH: 7.4

$\text{pCO}_2$ : 63.3 mm Hg       $[\text{H}_2\text{CO}_3]$ : 1.9 mEq/L       $[\text{HCO}_3^-]$ : 37.9 mEq/L



(D) Compensated Metabolic Acidosis

pH: 7.3  
-  
pH: 7.4

$\text{pCO}_2$ : 25.2 mm Hg       $[\text{H}_2\text{CO}_3]$ : 0.76 mEq/L       $[\text{HCO}_3^-]$ : 15.1 mEq/L



(c) Compensated Respiratory Alkalosis

pH: 7.6  
-  
pH: 7.4

$\text{pCO}_2$ : 25.3 mm Hg       $[\text{H}_2\text{CO}_3]$ : 0.759 mEq/L       $[\text{HCO}_3^-]$ : 15.1 mEq/L



(E) Compensated Respiratory Acidosis

pH: 7.4  
-  
pH: 7.6

$\text{pCO}_2$ : 63.5 mm Hg       $[\text{H}_2\text{CO}_3]$ : 1.9 mEq/L       $[\text{HCO}_3^-]$ : 38 mEq/L

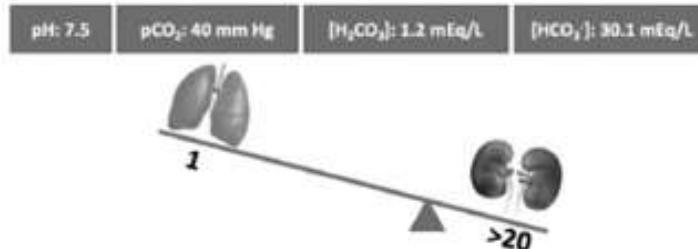


A 72-year-old male has been suffering with acute acid reflux and took half a bottle of antacid tablets to help alleviate his heartburn. A few hours later, he fell ill and was rushed to the hospital. The ABG indicated that the patients' blood pH was 7.5, his  $\text{PCO}_2$  was 38, and his  $\text{HCO}_3^-$  concentration was 29. Use this information to answer the following questions.

- a) **Which blood gas values are out of the normal range based on the ABG report? Explain what this could indicate.**

His pH is 7.5 which indicates that he is in a state of alkalosis likely caused by the abundance of antacid tablets ingested. His  $\text{pCO}_2$ , and therefore  $[\text{H}_2\text{CO}_3]$  are well within the normal range (38 mm Hg, 1.14 mEq/L). However, his  $[\text{HCO}_3^-]$  is too high indicating a **metabolic cause** of the disorder.

- b) Illustrate this scenario by drawing a seesaw to visualize where the imbalance lies. (See Figure 8 for reference).

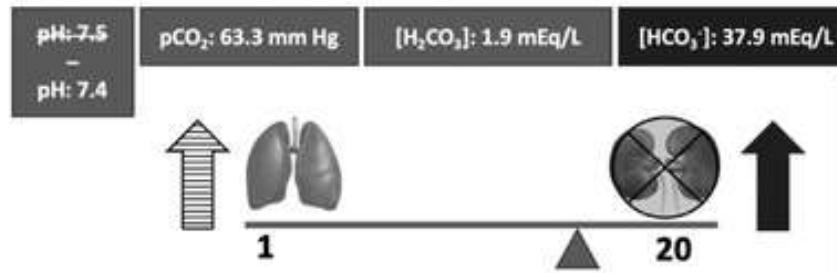


As shown in the figure,  $[\text{HCO}_3^-] = 30.1 \text{ mEq/L}$  is greater than the normal range of values (22-26 mEq/L).  $\text{HCO}_3^-$  is the metabolic component which is regulated by the kidneys which suggests renal dysfunction. Since  $[\text{HCO}_3^-]$  is high and  $\text{pCO}_2$  is normal, the scale will tip towards the 'heavy' renal component.

c) What is the appropriate diagnosis for this patient based on the ABG test results and the known symptoms?

### Metabolic Alkalosis

d) Based on what you know about the buffer system, which organ will be responsible for compensating to re-establish a physiological pH? (See Figure 9 for reference)



In this case, the kidneys are not functioning normally. Therefore, the [HCO<sub>3</sub><sup>-</sup>] value cannot change, and the compensatory response must be initiated by a respiratory pathway.

Since the kidneys are not functioning normally, the renal component [HCO<sub>3</sub><sup>-</sup>] cannot initiate the compensatory response. Instead, the lungs can compensate by slowing the respiratory rate which increases pCO<sub>2</sub>. While neither pCO<sub>2</sub> nor [HCO<sub>3</sub><sup>-</sup>] are normal, the temporary compensation by the respiratory component allows for pH = 7.4 while the metabolic issue is addressed.







Figure 19.29 Summary of acid-base disturbances and compensation.

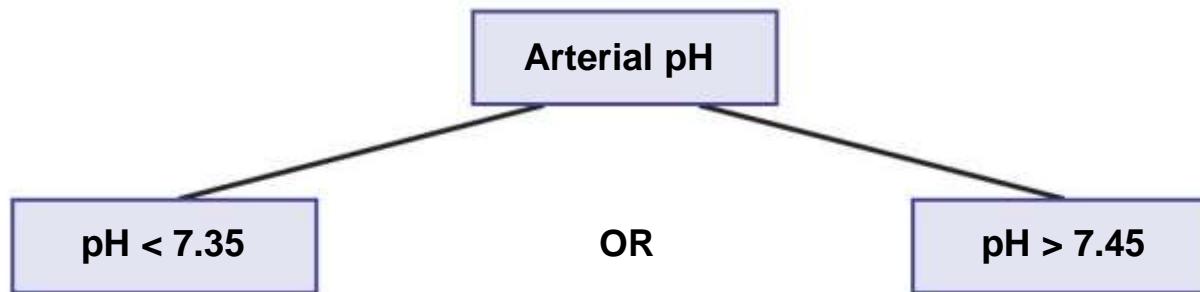


Figure 19.29 Summary of acid-base disturbances and compensation.

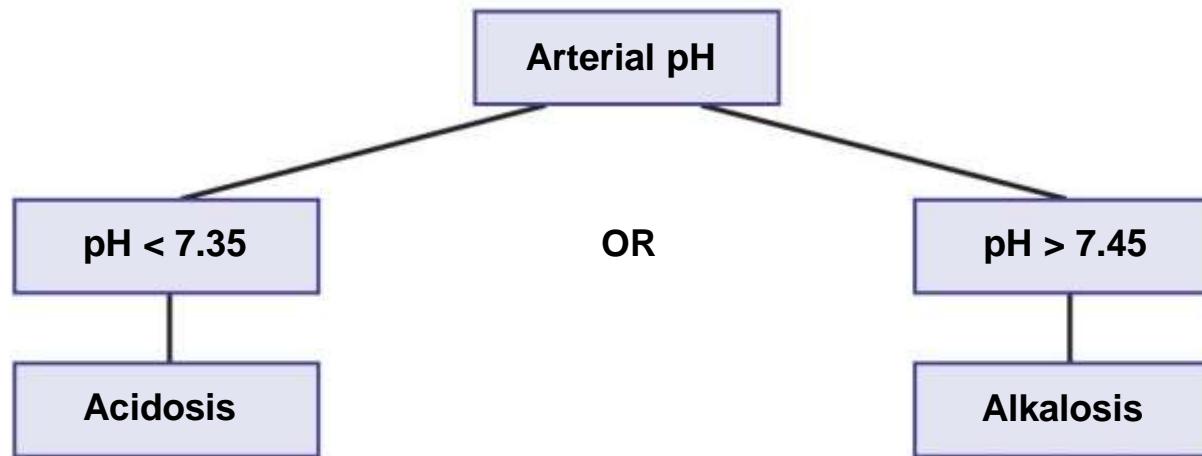


Figure 19.29 Summary of acid-base disturbances and compensation.

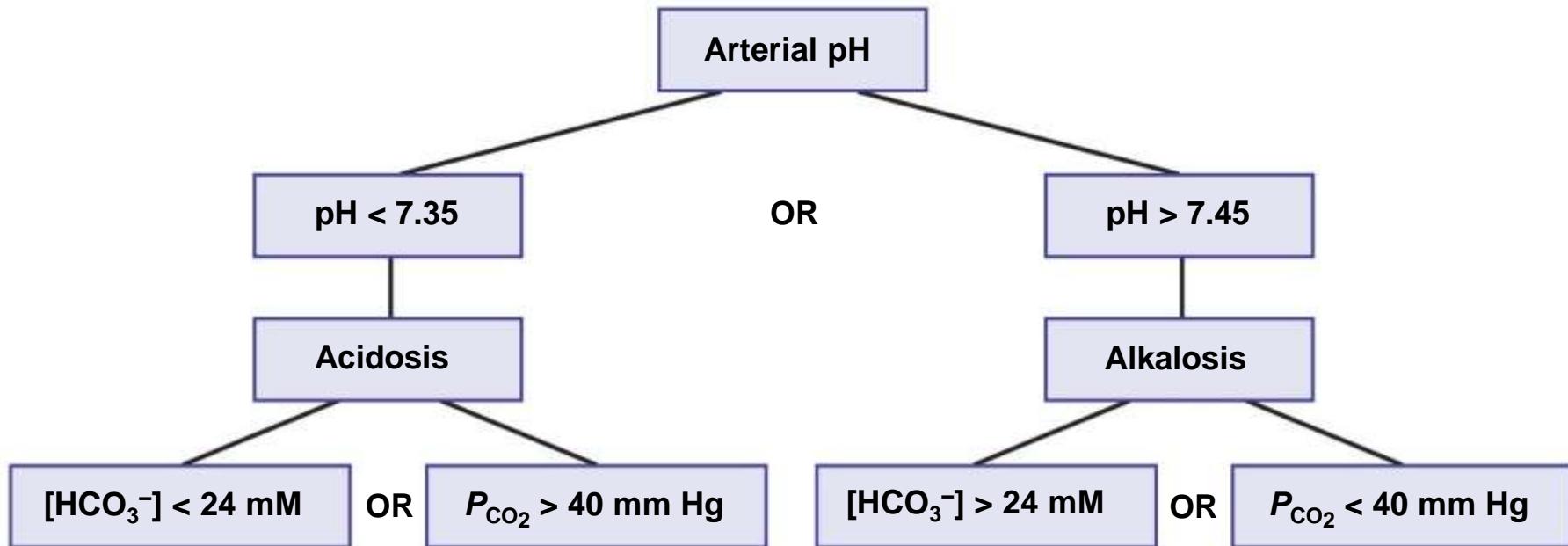


Figure 19.29 Summary of acid-base disturbances and compensation.

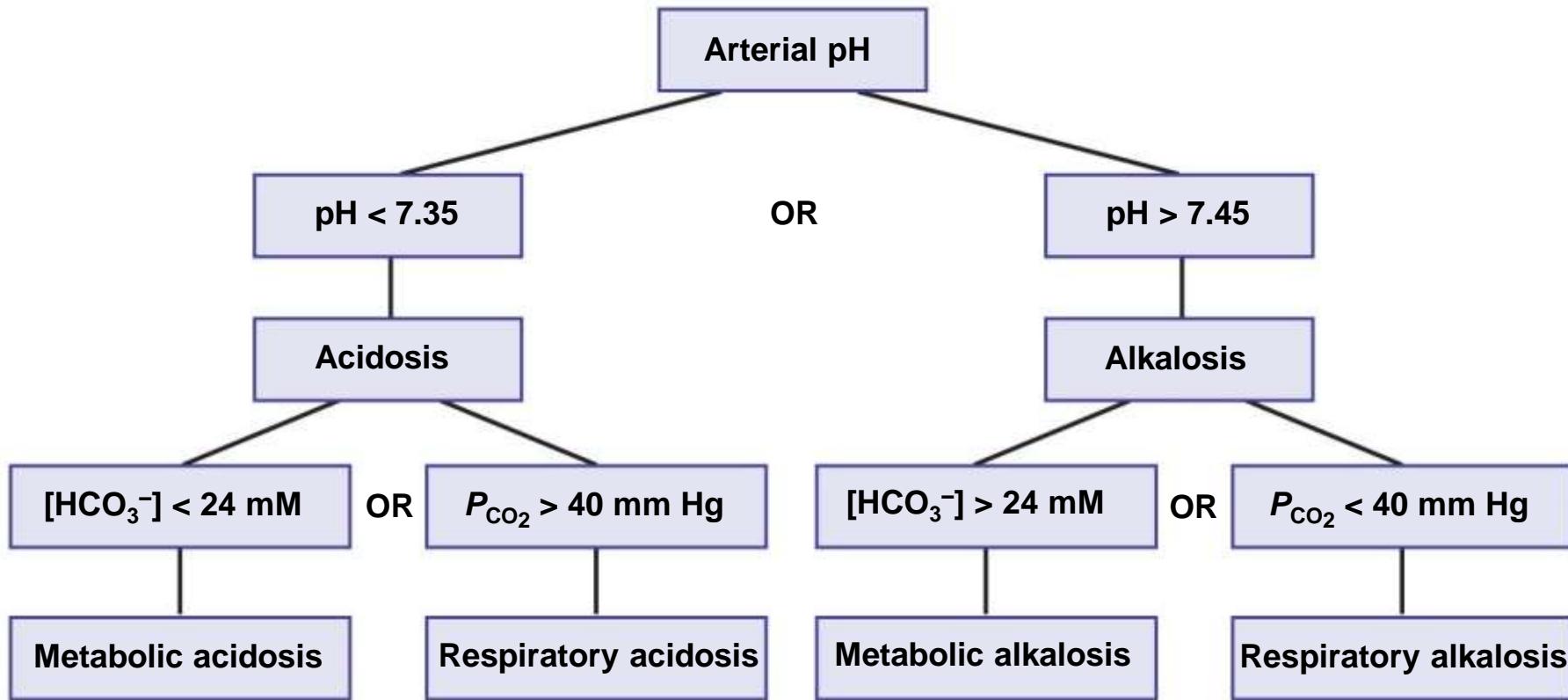


Figure 19.29 Summary of acid-base disturbances and compensation.

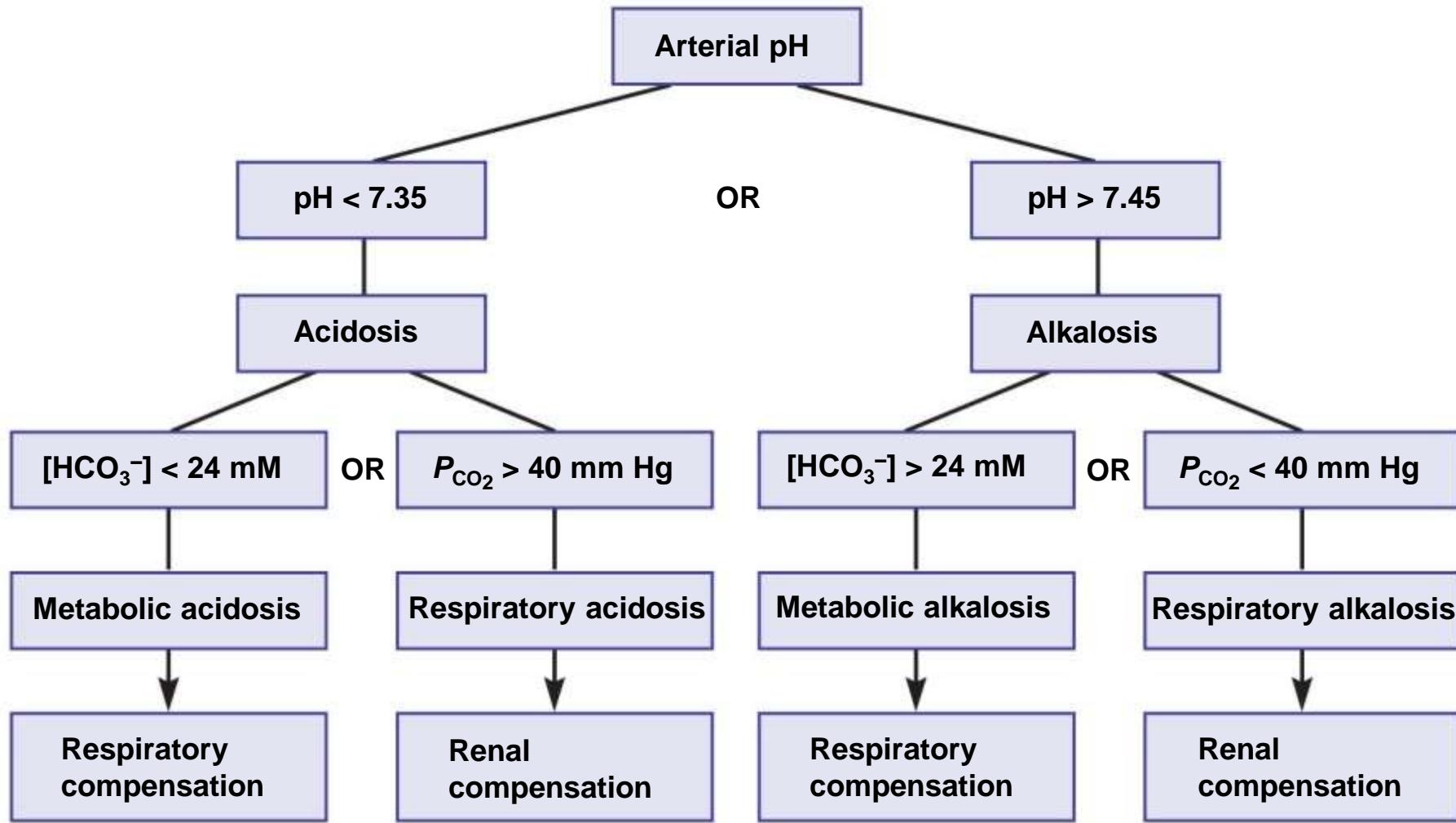


Figure 19.29 Summary of acid-base disturbances and compensation.

