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Acid-Base Balance

Biochemistry

Acids, bases and buffers, bicarbonate buffer system, respiratory and renal regulation of pH, acidosis, alkalosis and their biochemical basis.

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CHAPTER 14

Acid-Base Balance

Buffers, respiratory and renal pH regulation, acidosis and alkalosis

1. Basic concepts

Acids donate H+; bases accept H+. Blood pH is tightly regulated. Acidaemia is a measured arterial pH below 7.35; alkalaemia is above 7.45. Acidosis and alkalosis refer to processes tending to decrease or increase pH, respectively; mixed processes can coexist with a normal pH.

2. Buffer systems

Buffer	Role
Bicarbonate / carbonic acid	Major extracellular buffer; linked to lungs and kidneys
Haemoglobin	Buffers H+ generated during CO2 transport
Protein buffers	Ionizable side chains bind or release H+
Phosphate	Important intracellular and urinary buffer

Bicarbonate equilibrium: CO2 + H2O H2CO3 H+ + HCO3 . Carbonic anhydrase accelerates interconversion. Henderson-Hasselbalch equation for arterial blood: pH = 6.1 + log10(HCO3 / (0.03 x PaCO2)), with bicarbonate in mmol/L and PaCO2 in mmHg. A ratio near 20:1 gives pH near 7.4.

3. Regulation of body pH

Chemical buffers act rapidly. Lungs regulate volatile acid by changing ventilation: faster ventilation lowers PaCO2, while inadequate ventilation raises it. Kidneys reclaim filtered bicarbonate, excrete H+ as titratable acid and ammonium, and generate new bicarbonate. Renal compensation develops more slowly (hours to days).

4. Normal adult arterial blood gas (ABG) values

Parameter	Typical reference
pH	7.35-7.45
PaCO2	35-45 mmHg
HCO3	22-26 mmol/L

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PaO2	~80-100 mmHg in healthy adults at sea level, age and context dependent

5. Primary acid-base disorders

Disorder	Primary change	Possible causes	Compensatory direction
Metabolic acidosis	HCO3	DKA, lactic acidosis, renal failure, diarrhoea	PaCO2
Metabolic alkalosis	HCO3	Vomiting, gastric suction, diuretics	PaCO2
Respiratory acidosis	PaCO2	Hypoventilation, opioid respiratory depression, lung disease	HCO3

Disorder	Primary change	Possible causes	Compensatory direction
Respiratory alkalosis	PaCO2	Hyperventilation from hypoxaemia, pain, sepsis or other causes	HCO3

Compensation limits pH disturbance and does not usually overshoot; unexpected values suggest a mixed disorder.

6. Anion gap and compensation

Anion gap (AG) = Na+ - (Cl + HCO3), using mmol/L. A typical gap without potassium is approximately 8-12 mmol/L, but analyzer-specific intervals vary. Low albumin may lower the gap. High-gap acidosis suggests unmeasured anions, e.g., lactate or ketones. Normal-gap acidosis can occur with bicarbonate loss.

For metabolic acidosis, Winter's formula estimates expected respiratory compensation: PaCO2 1.5 x HCO3 + 8 +/- 2 mmHg. A measured result outside this range suggests an additional respiratory process.

7. Worked ABG examples

pH / PaCO2 / HCO3	Interpretation
7.28 / 55 / 25	Primary respiratory acidosis; evaluate acuity and compensation
7.25 / 25 / 11	Metabolic acidosis; expected PaCO2 ~24.5 +/- 2, consistent with compensation
7.50 / 30 / 23	Primary respiratory alkalosis
7.48 / 48 / 35	Metabolic alkalosis with compensatory CO2 increase

8. Chemical basis of acid production

Normal metabolism produces carbon dioxide and nonvolatile acids. Carbon dioxide combines reversibly with water, and ventilation controls its elimination. Nonvolatile acid arises from metabolism, including sulfur-containing

amino acids; the kidney contributes to net acid excretion. Buffering, respiratory regulation and renal regulation operate together rather than independently.

Bicarbonate reclamation and new bicarbonate

In renal tubules, secreted hydrogen ions combine with filtered bicarbonate; carbonic anhydrase facilitates conversion to carbon dioxide and water. Bicarbonate is returned to blood. Net acid excretion as ammonium and titratable acid permits addition of new bicarbonate to the circulation. Reclaiming filtered bicarbonate alone does not represent net acid excretion.

9. Recognizing mixed disorders

Finding	Possible interpretation
Low pH, low bicarbonate, low PaCO2	Metabolic acidosis with possible respiratory compensation
Low pH, high PaCO2	Respiratory acidosis, with bicarbonate assessed for compensation
High pH, high bicarbonate	Metabolic alkalosis, with PaCO2 assessed for compensation
High pH, low PaCO2	Respiratory alkalosis, with bicarbonate assessed for compensation

Normal pH does not exclude significant abnormality if bicarbonate and PaCO2 are abnormal. Evaluate compensation expectations, anion gap, symptoms and timing.

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10. Nursing assessment of acid-base disturbances

- Monitor respiratory pattern, oxygenation, circulation, consciousness and fluid losses.
- Recognize deep rapid breathing as a possible compensatory response to metabolic acidosis; do not assume anxiety is the cause.
- Obtain specimens according to arterial blood gas protocol and prevent sampling errors such as air exposure or processing delay.
- Report severe or worsening abnormalities promptly; bicarbonate administration is not appropriate for every acidosis.

11. Nursing applications

Assess airway, breathing, circulation, mental status and underlying causes. Confirm specimen type (arterial vs venous), oxygen therapy and collection conditions. Promptly report severe pH disturbances, respiratory deterioration or altered consciousness. Treat the cause under the medical plan; bicarbonate or oxygen is not automatically indicated for every abnormal ABG.

12. Important Examination Questions

Long Answer Questions

1. 1. Explain buffer systems and regulation of acid-base balance.
2. 2. Describe the four primary acid-base disorders and their compensatory responses.

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3. 3. Explain the bicarbonate buffer system and the Henderson-Hasselbalch equation.

Short Answer Questions

1. 1. Write a short note on Henderson-Hasselbalch equation.
2. 2. Write a short note on anion gap.
3. 3. Write a short note on four disorders.

Very Short Answer Questions

1. 1. What is acidaemia? - Arterial pH below 7.35.
2. 2. Which organ controls carbon dioxide elimination? - Lungs.
3. 3. Name the typical change in metabolic alkalosis. - Increased bicarbonate.
4. 4. What respiratory response often compensates for metabolic acidosis? - Increased ventilation with decreased PaCO2.

Additional objective question

Primary change in respiratory acidosis? - Increased PaCO2.

13. Quick Revision

- Normal arterial pH: 7.35-7.45; PaCO2: 35-45 mmHg; bicarbonate: 22-26 mmol/L.
- Respiratory disturbances primarily change PaCO2; metabolic disturbances primarily change bicarbonate.
- The bicarbonate buffer system depends on both pulmonary and renal regulation.
- Anion gap helps distinguish several causes of metabolic acidosis.

Conclusion

Acid-base interpretation requires a systematic assessment of pH, carbon dioxide, bicarbonate, compensation and the clinical situation.

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Practice questions

Choose one best answer. The answer key and explanations follow the questions.

1. Which finding defines acidaemia?

A. Arterial pH above 7.45
B. Arterial pH below 7.35
C. PaCO2 below 35 mmHg alone
D. Bicarbonate above 26 mmol/L alone

2. Which ABG pattern indicates primary respiratory alkalosis (pH / PaCO2 in mmHg / HCO3 in mmol/L)?

A. 7.28 / 55 / 25
B. 7.25 / 25 / 11
C. 7.50 / 30 / 23
D. 7.48 / 48 / 35

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3. Which formula calculates the anion gap when potassium is excluded?

A. Na+ + Cl + HCO3

B. Cl - (Na+ + HCO3)

C. HCO3 - (Na+ + Cl)

D. Na+ - (Cl + HCO3)

4. An arterial blood gas shows pH 7.29, PaCO2 52 mmHg and bicarbonate 24 mmol/L. Which primary disturbance is most consistent with these values?

A. Respiratory alkalosis

B. Metabolic alkalosis

C. A normal acid-base state

D. Respiratory acidosis

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Answer key and explanations

1. B - Arterial pH below 7.35

Acidaemia is defined by low measured arterial pH.

2. C - 7.50 / 30 / 23

High pH with reduced PaCO2 is the listed respiratory alkalosis pattern; values are pH/PaCO2/HCO3 .

3. D - Na+ - (Cl + HCO3)

The gap is sodium minus the sum of chloride and bicarbonate.

4. D - Respiratory acidosis

The pH indicates acidaemia and the elevated PaCO2 explains the acidifying effect. The bicarbonate value does not indicate a primary metabolic alkalosis.

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