

ACID BASE WORKSHOP

Acid/Base Learning Objectives

At the completion of this module the student should be able to:

- Identify the primary acid-base disturbances from arterial blood gases and serum chemistries.
- List important causes of these acid base disturbances.
- Define metabolic and respiratory compensation and distinguish between a “normal” and “abnormal” metabolic and respiratory response.
- Calculate the anion gap.
- Describe the pathogenesis of an anion gap.
- Distinguish between anion gap and non-anion gap metabolic acidosis.
- List causes of anion gap and non-anion gap metabolic acidosis.
- Use the Henderson-Hasselbalch equation or the non-logarithmic mass-action equation to calculate pH, $p\text{CO}_2$, and bicarbonate.
- Describe how bicarbonate and $p\text{CO}_2$ interact to determine pH.
- Calculate the bicarbonate deficit.
- Distinguish between the generation and maintenance of metabolic alkalosis.
- Relate how bicarbonate is handled by the kidney and the determinants of bicarbonate reabsorption.
- Describe the causes of “chloride dependent” and “chloride independent” metabolic alkalosis.

Problems:

a.	pH: 7.57	[H ⁺]: 27 nmoles/L	pCO ₂ : 24 mmHg	HCO ₃ ⁻² : 21 mEq/L
b.	pH: 7.28	[H ⁺]; 52 nmoles/L	pCO ₂ : 24 mmHg	HCO ₃ ⁻² : 11 mEq/L
c.	pH: 7.51	[H ⁺]; 31 nmoles/L	pCO ₂ : 50 mmHg	HCO ₃ ⁻² : 39 mEq/L
d.	pH: 7.26	[H ⁺]; 55 nmoles/L	pCO ₂ :60 mmHg	HCO ₃ ⁻² : 26 mEq/L

1. What primary acid/base disorder would explain each of the above blood gas values?
2. Give examples of clinical abnormalities which could give rise to each of the above?
3. What is the compensatory response and how do we know that it is appropriate?

Case 1:

A 16 year old patient is admitted in a confused state. His respiratory rate is 35/min. He is a known juvenile diabetic being given insulin.

Chemistry values:	serum sodium	150 mEq/L
	serum potassium	4 mEq/L
	serum chloride	114 mEq/L
	serum bicarbonate	5 mEq/L
	blood glucose	700 mg/dl
Arterial blood gas values	pH	7.14
	[H ⁺] = 72 nanoEq/L	
	pCO ₂	15 mmHg
	pO ₂	89 mmHg
	bicarbonate	5 mEq/L

1. What is the primary acid/base disturbance in this patient?
2. What is meant by “unmeasured anions” or “anion gap”?
3. What acid is the cause of this patient’s acidosis?
4. What anions are the causes of this patient’s elevated anion gap?
5. What are the causes of an anion gap metabolic acidosis?
6. What are the causes of a non-anion gap metabolic acidosis?
7. Assuming this patient’s ventilation remains the same, what would his pH become if his bicarbonate fell by 1 mEq/L to 4 mEq/L?

8. What would his pH become if his $p\text{CO}_2$ rises from 15 mmHg to 20 mmHg after he is given a sedative?

9. What is the estimated bicarbonate deficit in this 70 kg man?

10. What would happen to his pH if he is given the above amount of bicarbonate?
 - a. What might be the effect on his cerebrospinal fluid pH?
 - b. What would happen to his systemic pH after he is given insulin?

Case 2:

A 45 year old man with a history of smoking 2 packs of cigarettes a day since his teens presents with several months of cough and shortness of breath.

PE: BP: 150/100 P: 90 R: 24 T: 99° F Lungs: scattered rhonchi, diffuse expiratory wheezing, poor air entry; heart: distant heart sounds; abdomen: palpable liver edge; extremities: 2+ edema

Labs: Sodium 140 mEq/L; Potassium 4.0 mEq/L; Chloride 98 mEq/L; Bicarbonate 30 mEq/L
ABG: pH 7.36; [H⁺] 44; pCO₂ 55 mmHg; HCO₃⁻² 30 mEq/L; pO₂ 60 mmHg

1. What is this patient's acid base disorder?
2. Why is his bicarbonate elevated?
3. What would be his pH if his bicarbonate was 24 mEq/L?

Despite your therapy the patient's condition worsens and he returns in severe respiratory distress.

ABG: pH: 7.22; [H⁺]: 60 nmoles/L; pCO₂: 80 mmHg; HCO₃⁻² : 32 mEq/L;
pO₂: 40 mmHg

4. Why has the patient become more acidemic?
5. What is the mechanism for the increase in bicarbonate from 30 mEq/L to 32 mEq/L?

The patient is intubated and placed on a respirator; 30 minutes later you are called because the patient is having arrhythmias.

ABG: pH: 7.63 [H⁺]: 23 nmoles/L; pCO₂: 25 mmHg; HCO₃⁻² : 26 mEq/L: pO₂: 180 mmHg

6. What is the patient's acid/base disorder?
7. What is the cause of his arrhythmias?

Case 3:

A 44 year old woman was admitted to the hospital complaining of recurrent episodes of severe right upper abdominal pain and protracted vomiting for one week. The patient appeared as an obese middle-aged woman in no acute distress. BP was 100/60. Pulse was 100. Respiratory rate was 20. Temperature was 37.5C orally. Her weight was 90kg. She had tenderness to palpation in the right upper quadrant, but no rebound tenderness. The liver was not enlarged or tender. Bowel sounds were normal. Jaundice was not apparent. An ultrasound of the abdomen revealed a dilated common bile duct. The gallbladder contained stones. The patient continued to have pain and to vomit during the initial 24 hours. She was therefore placed on continuous nasogastric suction.

Laboratory data:

	<u>Normal</u>	<u>Admission</u>	<u>Fourth Day</u>
BUN	6-18 mg%	10	20
Creatinine	0.6 - 1.2 mg %	0.8	1.2
Na	135 - 145 mEq/L	140	137
K	3.5 - 5.5 mEq/L	3.3	3.3
Cl	98 - 106 mEq/L	98	85
HCO ₃	24 - 26 mEq/L	30	40
pH Art	7.36 - 7.44	7.46	7.53
pCO ₂	38 - 46 mmHg	44	51
H+	nanomole/L	35	30

1. What is the patient's acid/base disturbance?
2. What is the electrolyte content of gastric secretions?
3. What factors generated the acid/base disturbance?
4. What factor(s) are contributing to the maintenance of this disorder?
 - a. What would happen to the pH of a normal person if given sodium bicarbonate?

5. What would you expect the patient's urine chloride to be?
 - a. How does urine chloride level impact therapy?
 - b. What would happen to this patient's urine bicarbonate excretion if he was given saline?

6. Name the causes of "chloride-dependent" metabolic alkalosis.

7. Name the causes of "chloride-independent" metabolic alkalosis.

8. Why did the nasogastric suction worsen the acid/base disturbance and how could this complication have been prevented?