



UNIVERSITY OF
CALGARY
FACULTY OF NURSING

UNIT 6

Fluid and Electrolytes

James A. Rankin RN, PhD
Associate Professor
Faculty of Nursing
University of Calgary

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UNIT 6

Fluid and Electrolytes

Despite the name of this unit, it is often looked upon by students as a very dry subject. My own theory as to why this is the case, is that students have had a hard time *understanding* fluid and electrolytes in the past. If you have difficulty *understanding* the material, then of course you will find it hard going.

This unit is specifically designed to help you! Now I did not say it was going to be easy. There is some amount of work expected from you. This will involve some reading and reflection. So let's give fluid and electrolytes another chance.

Overview

Aim

1. To familiarize you with such pathophysiological concepts as buffers, regulation of electrolytes and acid-base balance by the kidneys and the lungs.
2. To increase your understanding of:
 - Respiratory acidosis
 - Respiratory alkalosis
 - Metabolic acidosis
 - Metabolic alkalosis
3. To familiarize you with the clinical manifestations of certain electrolyte imbalances.

Objectives

As alluded to above it is expected that by the end of this unit you will feel more comfortable with terms such as “respiratory acidosis” and your overall *understanding* of fluid and electrolytes will have increased. One way of assessing your understanding is by asking yourself:

“In relation to my clinical setting:

1. Do I feel more at ease with patients who have fluid and electrolyte imbalances?
2. Do I have a greater *understanding* of laboratory reports?”

If these questions are not relevant to your current area of practice you might want to *reflect* on potential situations in which your increased knowledge of fluid and electrolytes may be of help to you.

On completion of this unit you will be able to:

1. Describe the role of the lungs and kidneys in the regulation of fluid, electrolytes and acid-base balance.
2. Describe the pathophysiology and clinical manifestations of hyper and hyponatremia.
3. Describe the pathophysiology and clinical manifestations of hyper and hypokalemia.

Resources



Requirements

Read McCance & Huether (2001). pp. 85-102.

Print Companion: Fluids and Electrolytes

Supplemental Materials

You may wish to read McCance and Huether, 2001, pp. 102 on, as they provide an overview of the content of the unit.

Orientation to the Unit

Before starting the unit, it might be worthwhile reviewing some basic information related to normal physiology. (If you feel that you have a good understanding of lung and kidney function then it is not necessary to do the review.) For example,

Van De Graaff, K. & Fox, S. (1992). *Concepts of human anatomy and physiology*. (3rd ed.) Dubuque, IA: Wm. C. Brown.

This is a useful text to review acid-base balance and the role of the kidneys and lungs, (see pp. 699-716 and 666-686), or another anatomy and physiology text which deals with the same material will do.

As previously stated the bulk of the content that you are required to know for this unit is contained in the following pages.

Take a "time out" from this unit and decide whether you need to do the review or not. If you have decided to do the review, set aside time to do it thoroughly. You will probably need about 2-3 hours. Good Luck!

OK now you have done the review. Let's get started!

There are three parts. The first part deals with buffers, the second part deals with acid-base imbalance and the third part deals with electrolyte imbalance. Each part has increasing student activity.

Web Links

All web links in this unit can be accessed through the Web CT system.

Section 1: Buffers

Basic Chemistry and pH Review

Before looking at buffers, we shall review some basic chemistry and the meaning of the term “pH”.

Basic Chemistry

Electrons are negatively charged particles that orbit the nucleus of an atom. Protons are positively charged particles found in the nucleus of an atom. When you have more electrons than protons the atom is negatively charged. Similarly when there are more protons than electrons the atom is positively charged. Particles that carry an electrical charge (whether positive or negative) are known as **ions**. Ions are formed when an atom gains or loses electrons.

For example, when a sodium (Na) atom loses an electron it has one more proton than electrons and so becomes positively charged. A positively charged ion is called a *cation* (pronounced “CAT-I-on”).

When a chlorine atom gains an electron it becomes negatively charged. A negatively charged ion is called an *anion* (pronounced “AN-I-on”).

Note: An easy way to remember that cations are the positively charged ones is to think of the top of the “t” in cation as having a plus sign (ca+ion).

pH

The “pH” symbol is taken to mean the hydrogen ion (H⁺) concentration of a solution. The scale ranges from 1 to 14. The numbers in the pH scale actually refer to the negative log of the hydrogen ion concentration in expressed in moles per litre. A mole is the amount of an element that has a mass, in grams, equal to its atomic weight. For example, Hydrogen has an atomic weight of 1.01, therefore 1.01 grams of Hydrogen would constitute one mole of Hydrogen.

A pH of 7 indicates neutrality in a solution, that is there are equal amounts of H⁺ and hydroxyl (OH⁻) ions. In one litre of pure, distilled water there is 0.0000001 moles of hydrogen ions and the same number of hydroxide ions. So we can say that the *concentration* of hydrogen ions in our 1 litre of distilled water is 0.0000001 moles per litre – this can be written as follows:

$$[\text{H}^+] = 1 \times 10^{-7} \text{ mol/L}$$

The square brackets around the name of a substance (in this case Hydrogen) means, “The concentration of...”.

You would say, $[H^+] = 1 \times 10^{-7}$ mol/L in words:

“The concentration of hydrogen ions equals one times ten to the minus seven moles per litre”.

If we take the negative log of 0.0000001 we get 7. Now we can express hydrogen ion concentration in a shorthand way rather than writing all the zeros out every time. So for distilled water instead of writing this: $[H^+] = 1 \times 10^{-7}$ mol/L we can say, the pH is 7! It is important to remember that the pH scale of hydrogen ion concentration is logarithmic, this means the concentration of H^+ changes by a factor of 10 for each unit of the scale. For example, a pH of 7 has a H^+ concentration ten times that of a pH of 8.

STUDENT ACTIVITY – You can check this out for yourself if you take the number that a pH of 8 equals i.e. 0.00000001 (this number is the same as 1×10^{-8}) and multiply it by 10 you will get 0.0000001 (or 1×10^{-7}). In other words a substance with a hydrogen ion concentration or pH of 7 has ten times more hydrogen ions (in moles per litre) than a substance with a pH of 8.

A pH of less than 7 indicates acidity, whereas a pH greater than 7 indicates alkalinity.

NB: A *decrease* in the pH number indicates an *increase* in acidity i.e. H^+ concentration and vice versa.

e.g. pH 1.7 (*low* number, *high* acidity)

pH 10.8 (*high* number, *low* acidity)

The pH of our internal environment continually fluctuates with absorption of acids and bases from foods and catabolism. Despite these substances our pH is kept remarkably constant. Apart from the extreme acidity of the stomach (pH 1 to 3), the pH of our blood, cells and certain tissue fluids tends to be around a neutral pH of 7. Table 6.1 below has the normal pH range of blood.

Table 6.1 Normal pH range of blood

Normal range of blood pH =	7.35 - 7.45 (arterial)
	7.30 - 7.41 (venous)

Average =	7.41 (arterial)
	7.36 (venous)

Table 6.2 has the pH of certain body fluids and other substances.

Table 6.2 pH of certain body fluids

Substance	PH
Hydrochloric acid in the stomach	1
Gastric juice	1 - 3
Wine	3
Tomatoes	4
Coffee	5
Urine	5 - 7
Milk	6.5
Saliva	6.3 - 7.3
Pure, distilled water at 25 ⁰ C	7
Blood	7.35 - 7.45
Household bleach	9.5
Oven cleaner	13.5
Sodium hydroxide	14

Three systems are at work to control the homeostasis of our internal pH, these are:

1. respiratory system
2. urinary system (excretion of acids and bases)
3. buffers

Buffers

A buffer is a substance “that prevents marked changes in the pH of a solution when an acid or base is added to it” (Anthony & Thibodeau, 1983, p. 687).

A buffer may bind or release H⁺ in order to maintain the stability of a solution.

Buffers consist of two substances and are referred to as “buffer pairs.” Most of the body fluid buffers consist of a weak acid and a salt of that acid, e.g.

Sodium bicarbonate

1. Bicarbonate pairs NaHCO_3 (Sodium bicarbonate is the salt)
 H_2CO_3 (Carbonic acid)

There is also KHCO_3 ; CaHCO_3 ; MgHCO_3

(i.e. potassium, calcium and magnesium bicarbonate salts)

2. Plasma protein pairs Na proteinate
 Proteins (weak acids)

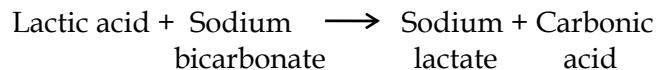
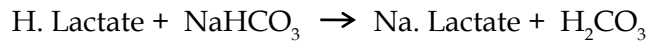
There are also hemoglobin and phosphate buffer pairs.

Buffers alone cannot control the blood pH, the respiratory and urinary systems play their part in removing H⁺ from the blood.

Let's look at some examples.

Example 1 – A build up of lactic acid (H lactate)

Lactic acid is buffered by sodium bicarbonate (NaHCO_3 , the most abundant base bicarbonate in the blood) as follows:

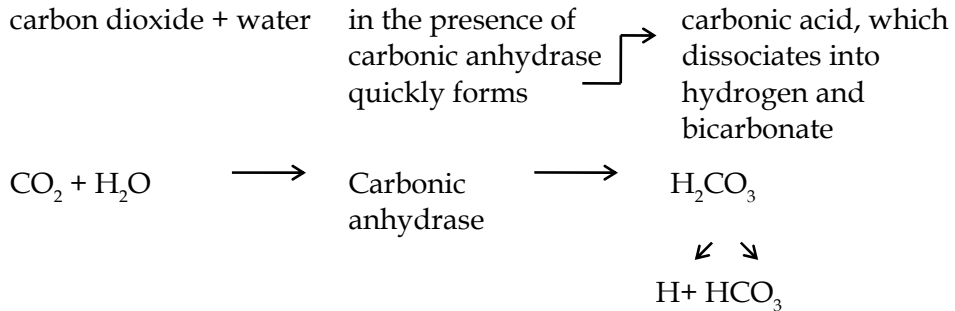


Carbonic acid is a *weaker* acid than lactic acid thus few hydrogen ions are added to the blood. (NB: Strong acids readily give up H⁺, weak acids do not.)

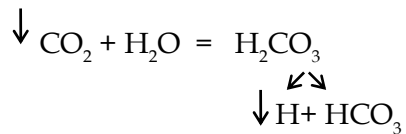
Ketone bodies (acetoacetic acid) are buffered in the same way.

Example 2—Respiratory control of blood pH

CO₂ and H₂O combine, in the presence of the enzyme, carbonic anhydrase, to form carbonic acid (H₂CO₃) as follows:



By following the above equation it may be seen that by *decreasing* the amount of CO₂ in the blood there will be a *decrease* in the amount of H⁺ in the blood (i.e. an *increase* in pH) e.g.



When there is an *increase* in H⁺ concentration in the blood e.g. in diabetic ketoacidosis the respiratory system responds as follows:

1. Diabetic ketoacidosis increase in H⁺ (decrease in pH)
2. Decreased pH stimulates respiratory centre
3. Increase in depth and rate of ventilations leads to hyperventilation
4. CO₂ is “blown off”
5. Decrease in blood CO₂ leads to a decrease in H⁺ in blood
6. Increase in blood pH toward normal

Example 3—Urinary control of pH

The kidneys can excrete both acids and bases, and therefore play a vital role in controlling pH.

Two mechanisms exist in the urinary system which allows the urine to become more acid and thus the blood to become more alkaline. These mechanisms are biochemical reactions, they are also reversible, thus the urine may become more alkaline and the blood more acid.

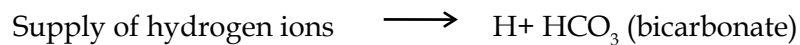
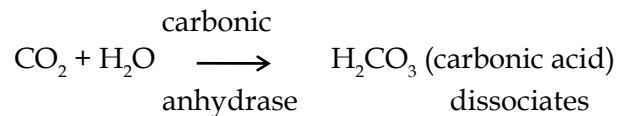
These mechanisms are:

1. The addition of H⁺ ions from the distal tubule cells to the urine in exchange for Na⁺
2. The breakdown of the amino acid glutamine in the distal tubule cell to form ammonia (NH₃). The ammonia enters the urine and is converted to ammonium (NH₄) by the addition of one H⁺

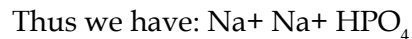
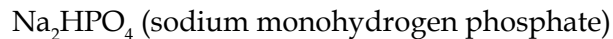
These mechanisms are illustrated below.

1. Addition of H⁺

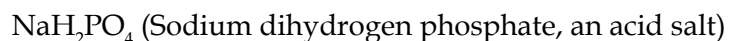
1. CO₂ from blood capillary → distal tubule cell
2. In distal tubule cell:



3. In tubular urine:



4. One of the Na⁺ is displaced by a H⁺ from the distal tubule cell to give:



5. **The urine becomes more acid**

6. The Na^+ which was displaced diffuses into the distal tubule cell and combines with the HCO_3^- (bicarbonate) to form:
 NaHCO_3 (Sodium bicarbonate, a *basic* salt).

* 7. This *basic* salt enters the capillary blood and thus the blood is made more *alkaline*.

* Steps 3 to 7 are illustrated in Figure 6.1 below.

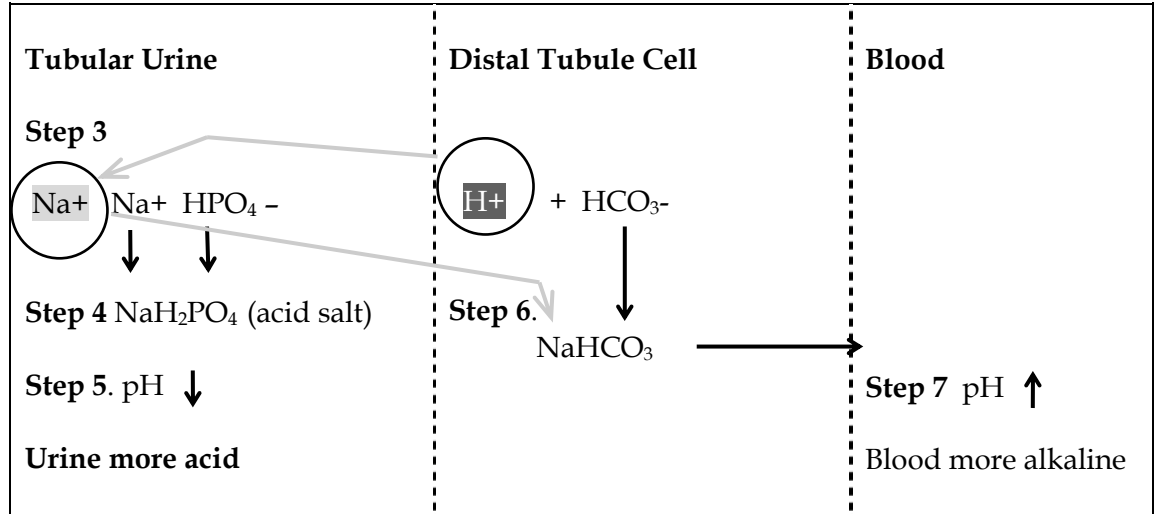


Figure 6.1 The addition of hydrogen ions to the urine



Learning Activity #1—Clinical Point

(Answers at End of Unit)

In health, renal tubules may excrete either H^+ or K^+ in exchange for Na^+ .

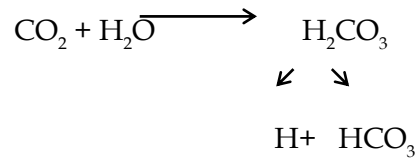
Generally speaking the more H^+ excreted the fewer K^+ are.

1. In acidosis tubule excretion of H^+ dramatically increases – why?

2. Excessive excretion of H^+ means more K^+ are retained – resulting in?

2. Ammonia/ammonium mechanism

1. The amino acid glutamine is *deaminated* in the distal tubule cell. Ammonia (NH₃) is formed and enters tubular urine
2. In the tubular urine, ammonia (NH₃) displaces Na or some other basic ion from a salt e.g. Na Cl
3. As well as displacing the Na, there are H⁺ available in the tubular urine, which have been derived from the



mechanism in the distal tubule cell.

4. These H⁺ associate freely with NH₃ to form ammonium (NH₄)
5. Thus an acid salt is formed >NH₄ Cl (ammonium chloride)
- * 6. The displaced Na⁺ enters the tubule cell and forms NaHCO₃ which diffuses into the blood, as before.

The urine is made more *acid* and the blood more *alkaline*.

*Steps 1 to 6 are illustrated in Figure 6.2.

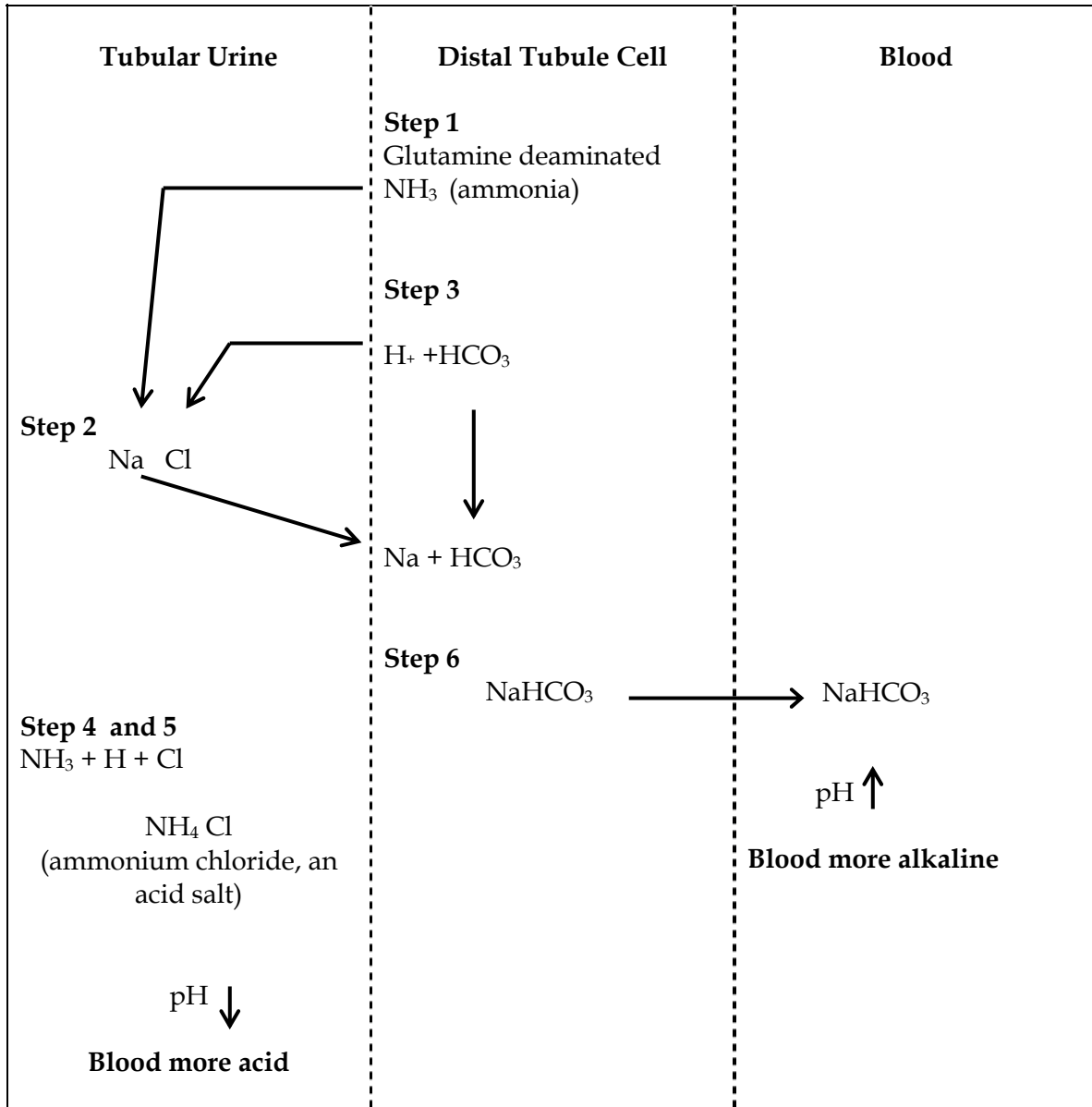


Figure 6.2 Ammonia/Ammonium Mechanism

With ammonium chloride in the urine it becomes more acid. With sodium bicarbonate in the blood it becomes more alkaline. It is important to note that the mechanisms described (addition of hydrogen ions and ammonia/ammonium mechanism) are *reversible* reactions. This means that the urine would become more alkaline and the blood more acid.

Section 2: Acid-base Imbalances

A normal acid-base balance exists when the ratio of base bicarbonate (BB) to carbonic acid (CA) is 20:1.

An *imbalance* may occur due to metabolic or respiratory disturbances. When the imbalance is in favour of BB an alkalosis occurs, whereas an imbalance in favour of CA causes an acidosis. It is the *relative* quantities of BB:CA in the extracellular fluid (ECF) which may cause an imbalance.

Before looking at some examples, remember that *bicarbonate ion* concentration is regulated by the *kidneys* and the *carbonic acid concentration by the respiratory system*.

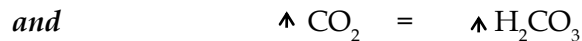
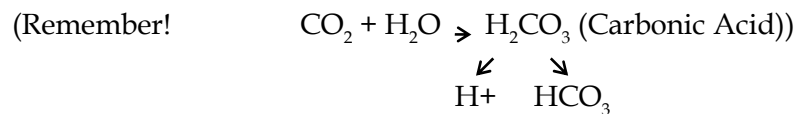
Reference values:	pH 7.35-7.45 (arterial)
[arterial blood gas	PaCO ₂ 35-45 mmHg
levels - normal]	PaO ₂ 80-100 mmHg
	HCO ₃ 24-28 mmol/L

Respiratory Disturbances

Respiratory Acidosis (Carbonic acid excess)

Cause

Accumulation of CO₂, and therefore CA.



An increase in CO₂ in the blood will mean an increase in H₂CO₃.

Accumulation is caused by retention of CO₂ therefore gas exchange must be impaired.



Learning Activity #2

Can you think of conditions which may cause this? (See Answer in at the end of this unit)

Physiological Compensation – Renal

1. Excretion of H⁺ by kidneys (acid urine)
2. Reabsorption of HCO₃
3. K⁺ exchanged, move from ICF to ECF, causing hyperkalemia

In acute respiratory acidosis renal compensation is less than effective. In chronic uncomplicated respiratory acidosis renal compensation is effective.

Clinical Features

Irritable, restless, tachycardia, ventricular ectopics leading to ventricular fibrillation.

Interventions

1. Distinguish between acute and chronic respiratory acidosis (Health history and laboratory values are necessary)
2. Airway clearance
3. Suctioning, physiotherapy, postural drainage
4. Administration of O₂
5. Mechanical ventilation



Note: Danger of O₂ administration in patients with COPD. Patients with chronic obstructive pulmonary disease (COPD) may have a PaCO₂ around 50 mmHg. Their respiratory centre is insensitive to an increase in CO₂. Therefore hypoxemia is their major stimulus for breathing. Too much O₂ “knocks out” the stimulus to breathe, coma and death can quickly ensue.

Respiratory Alkalosis (Carbonic acid deficit)**Cause**

Hyperventilation secondary to hysteria, fever or hypoxia at high altitudes, brain dysfunction. Less common than respiratory acidosis.

Physiological Compensation – Renal

1. Reduction in ammonium (NH₄) formation and reduction in excretion of H⁺
2. HCO₃ no longer conserved
3. K⁺ moves from ECF into cells in exchange for H⁺ in an attempt to add H⁺ ions to ECF
4. A compensatory acidosis develops

Clinical Features

Hyperventilation; paraesthesia fingers and toes; palpitations; Chvostek's and Trousseau's + ve.

What is Chvostek's and Trousseau's sign?

(See p. 100 Clinical Manifestations in McCance & Huether, 2001).

Interventions

1. Increase PaCO₂ by rebreathing own CO₂
2. Emotional/psychological support encourage normal breathing pattern
3. Assess for other disease process (e.g. congestive heart failure; fever; cirrhosis)

In both conditions described above electrolyte solutions play little or no part in the medical treatment. Treatment is aimed at the underlying cause.

Respiratory Disturbances Blood Gases and pH—Step by Step Guide

Step by Step Guide to help you determine the respiratory disturbance based on laboratory findings.

1. What is pH?

- pH < 7.35—acidosis
- pH > 7.45—alkalosis

2. What is the PaCO₂?

- PaCO₂ > 45—acidosis
- PaCO₂ < 35—alkalosis

3. What is the HCO₃?

- HCO₃ < 24—acidosis
- HCO₃ > 28—alkalosis

4. Match like terms

Example 1

- pH 7.34 (acidosis)*
 - PaCO₂ 50 (acidosis)*
 - HCO₃ 29 (alkalosis)
- *∴Respiratory acidosis**

Example 2

- pH 7.49 (alkalosis)*
 - PaCO₂ 30 (alkalosis)*
 - HCO₃ 22 (acidosis)
- *∴ Respiratory alkalosis**
-

Metabolic Disturbances

Metabolic Acidosis (Bicarbonate Deficit)

Cause

Vigorous exercise (lactic acid) – physiological.

- Shock -- poor tissue perfusion, accumulation of organic acids.
- Diabetic ketoacidosis.
- Renal failure.
- Depletion of HCO_3^- , chronic, acute diarrhea.

Physiological Compensation – Respiratory; Renal

Recall the clinical point example of diabetic ketoacidosis.

1. Lowering of blood pH stimulates the respiratory centre to increase the respiration rate . Hyperventilation blows-off CO_2 (carbonic acid)
2. Excretion of H^+ and NH_3 by kidneys, retention HCO_3^-
3. If these homeostatic mechanisms fail, uncompensated metabolic acidosis ensues

Clinical Features

1. Features of underlying cause
2. Kussmaul's respirations (deep and rapid)
3. Dehydration -- nausea, vomiting, diarrhea
4. Severe acidosis can produce life threatening dysrhythmias

Interventions

1. Directed at prevention e.g. in diabetics
2. Appropriate intravenous therapy administered

Medical Treatment

1. Treat underlying cause
2. Fluid and electrolyte replacement e.g. 4-6 litres of fluid; Na⁺; K⁺; NaHCO₃ as necessary

Metabolic Alkalosis (Bicarbonate Excess)**Cause**

Most commonly as a result of abnormal loss of hydrochloric acid from the stomach due to prolonged or severe vomiting. Excessive ingestion of sodium bicarbonate e.g., in the treatment of peptic ulcers. Excessive potassium loss by diarrhea, vomiting or diuretic therapy.

Physiological Compensation – Renal; Respiratory

H⁺ ions are lost in vomiting, HCO₃⁻ ions are retained, causing an imbalance in the BB:CA ratio. BB alkalosis occurs.

Kidney retain H⁺ and NH₃ formation and they no longer conserve HCO₃⁻.

Respiratory – Rate and depth of respirations are decreased, thus conserving CO₂ → PaCO₂ rises as do levels of carbonic acid.

Clinical Features

1. Underlying cause – nausea, vomiting
2. Confusion, carpopedal spasm

Interventions

1. Observation of vital signs and neurological status
2. Administration of fluid and electrolyte replacements e.g. K⁺; NaCl as necessary

Medical Treatment

1. Underlying cause
2. Fluid and electrolyte replacement. Extracellular fluid is increased by IV NaCl and K being given

Metabolic Disturbances: Blood Gases and pH Step by Step Guide

Step by Step Guide to help you determine the metabolic disturbance based on laboratory findings.

1. What is pH?

- pH < 7.35 - acidosis
- pH > 7.45 - alkalosis

2. What is the PaCO₂?

- PaCO₂ > 45 - acidosis
- PaCO₂ < 35 - alkalosis

3. What is the HCO₃?

- HCO₃ < 24 - acidosis
- HCO₃ > 28 - alkalosis

4. Match like terms

Example 1

- pH 7.48 (alkalosis)*
 - PaCO₂ 46 (acidosis)
 - HCO₃ 30 (alkalosis)*
- *∴ **Metabolic alkalosis**

Example 2

- pH 7.26 (acidosis)*
 - PaCO₂ 34 (alkalosis)
 - HCO₃ 22 (acidosis)*
- *∴ **Metabolic acidosis**

General Rule

1. When the pH tells you “acidosis” and you have -
 - matched it with PaCO₂ – the condition is *respiratory acidosis*
 - matched it with HCO₃ - the condition is *metabolic acidosis*

Similarly

2. When the pH tells you “alkalosis” and you have -
 - matched it with PaCO₂ - the condition is *respiratory alkalosis*
 - matched it with HCO₃ - the condition is *metabolic alkalosis*
-

Clinical Example: Diabetic ketoacidosis

1. What is blood pH, acid or alkaline? (Answers at the back of this unit)
2. H^+ and NH_3 secreted by distal tubule cells
3. Urine becomes more acid, by the mechanisms described above (the addition of hydrogen and the ammonia/ammonium mechanism)
4. $NaHCO_3$ is reabsorbed into capillary blood, this increases pH of blood.
5. The physiological objective is to return pH of blood to normal range.
6. What other mechanism comes in to play in an attempt to increase blood pH? (See Appendix A for answer)

Compensation

The values that you may see in clinical practice may not be quite as clear as the examples given above. This is because compensation may be taking place in an attempt to correct the abnormal values caused by the primary condition. Compensation may be absent, partial or complete.

Let's take a look at some examples.

Example 1 Respiratory Acidosis

Table 6.3 presents what is happening in the various stages of compensation in the case of respiratory acidosis. As you look down the HCO_3 column you will see that the values increase. In the complete stage of compensation the HCO_3 is abnormally high ($HCO_3 = 35$ mmol/L). Now look at the $PaCO_2$ values, as you look down the column the $PaCO_2$ is decreasing. In the complete stage of compensation the $PaCO_2$ is still abnormally high. We have the situation where both the HCO_3 and $PaCO_2$ levels are abnormal. But look at the pH column – in the complete stage of compensation the pH is normal. The *relative* changes in the HCO_3 and $PaCO_2$ have been sufficient to return the pH to normal.

Remember that the aim of compensation is to restore normal pH.

Table 6.3 Respiratory Acidosis

Stage of compensation	pH	PaCO ₂ (mmHg)	HCO ₃ (mmol/L)
Absent	7.28 Abnormal ↓	70 Abnormal ↑	28 Normal
Partial	7.31 Abnormal ↓	68 Abnormal ↑	35 Normal ↑
Complete	7.36 Normal	55 Abnormal ↑	35 Abnormal ↑

You can see from Table 6.3 that in the “absent” stage the PaCO₂ is abnormally high and the pH is abnormally low.

**Note:**

- In respiratory acidosis the *primary* problem is an *increase* in CO₂. The *aim* of compensation is to restore the pH to within the normal range. This is achieved by *increasing* HCO₃.
- The initial condition (i.e. absent compensation) shows an *abnormal* pH and PaCO₂ with a *normal* HCO₃.
- The completely compensated condition shows a *normal* pH and an *abnormal* PaCO₂ and HCO₃.
- The *aim* of compensation has been achieved – a *normal* pH.

Learning Activity #3



Using Example 1 as a reference, fill in the blanks in the tables and in the statements, in the next three examples. (See Appendix for answers.) Remember the little arrows!

Example 2 Respiratory Alkalosis

Stage of compensation	pH	PaCO ₂ (mmHg)	HCO ₃ (mmol/L)
Absent	7.49 Abnormal	30	26
Partial	7.47	30	22
Complete	7.44 Normal	32 Abnormal	18

- In respiratory alkalosis the primary problem is _____ in CO₂
- The aim of compensation is to restore the pH to _____ the _____ range
- This is achieved by _____ HCO₃
- The initial condition shows _____ pH and _____ with _____ HCO₃
- The aim of compensation has been achieved a _____ pH

Example 3 Metabolic Acidosis

Stage of compensation	pH	PaCO ₂ (mmHg)	HCO ₃ (mmol/L)
Absent	7.24	44 Normal	14
Partial	7.28	34	14
Complete	7.35	28 Abnormal	15

- In metabolic _____ the primary problem is _____ in HCO₃.
- The aim of compensation is to restore the pH to _____ the _____ range.
- This is achieved by _____ the PaCO₂.
- The initial condition shows _____ pH and _____ with _____.
- The completely compensated condition shows _____ pH and _____ and _____.
- The aim of compensation has been achieved.

Example 4

This one could be tricky. **Be very observant!**

Stage of compensation	pH	PaCO ₂ (mmHg)	HCO ₃ (mmol/L)
Absent	7.45	54	38
Partial	7.51	44	40
Complete	7.48	48	38

- In _____ the primary problem is _____ in _____.
- We know what the aim of compensation is now!
- This is achieved by _____ the _____.
- The initial condition shows _____ pH and _____ with _____.
- The completely compensated condition shows _____ pH and _____ and _____.
- Has the aim of compensation been achieved?



Learning Activity #4—A Clinical Example (just for fun!)

If you get the answers correct you are well on your way to *understanding* acid/base balance.

An 18-year-old male is admitted from Emergency after being knocked off his motorbike by a truck.

- Injuries
- Closed transverse fracture (L) femur
 - multiple (L) rib fractures
 - minor cuts

Initial arterial blood gas values were:

pH 7.35 PaCO₂ 45 HCO₃ 28

1. How would you interpret each value?

2. Complications may occur, therefore you need to be alert to changes in blood gas values. What complications might arise?

3. A few hours later, he becomes anxious. Respirations are 44/min. and he is complaining of severe pain on inspiration. What might you suspect?

Blood gas values are now:

pH 7.30 PaCO₂ 60 HCO₃ 30

4. What do these values indicate?

5. This patient would be best managed by:

Section 2: Electrolyte Imbalance

You will see from your textbook (McCance & Huether, 2001) that pp. 90-99 deals with electrolyte imbalances. A number of electrolytes are mentioned including sodium, potassium, calcium, phosphate, chloride and magnesium.

It is important to read the relevant sections in the textbook concerning these electrolytes as a variety of pathophysiological changes may occur from their imbalance.

However, you need to focus most closely on *sodium and potassium imbalance* as there will only be exam questions on sodium and potassium.

There now follows some basic information on sodium and potassium.



Read:

1. Read the basic information on sodium and potassium in the Print Companion.
2. Supplement your study notes by reading the following from McCance and Huether, 1998:
 - Hyponatremia p. 91
 - Hyponatremia p. 92
 - Hypokalemia p. 95
 - Hyperkalemia p. 98

Potassium

Potassium is the major *cation* of intracellular fluid (ICF). A cation is a positively charged electrolyte. A negatively charged electrolyte is known as an *anion*. The extracellular fluid (ECF) has *relatively* small amounts of K⁺ e.g.

Concentrations of Potassium in ICF and ECF

ICF	ECF
K ⁺ 100 mmol/L	K ⁺ 4.5 mmol/L

Serum is part of the ECF. You can see from the above table, that serum potassium concentration must be *relatively* low (i.e. *relative* to the ICF fluid concentration). Serum K⁺ concentration does not change appreciably with changes in total body water (as compared with sodium, for example).

Changes in serum K⁺ concentrations tend to reflect:

1. Changes of total body K⁺ (absolute change)
2. Shifts of potassium into or out of the large ICF pool (relative change)

Tubular Reabsorption and Secretion

By the end of the Loop of Henle approximately 90% of filtered potassium has been reabsorbed. Reabsorption must be active and requires a pump.

Almost all potassium appearing in the urine is secreted in the distal tubule by passive transport down a concentration gradient.

In the distal tubule, hydrogen is actively secreted in exchange for sodium. However, potassium competes with hydrogen to exchange with sodium. Thus:

H⁺ secretion (acidosis) = K⁺ retention (hyperkalemia)

H⁺ retention (alkalosis) = K⁺ secretion (hypokalemia)

In summary, potassium is handled in two ways in the distal tubule:

1. K⁺ is the major cation of ICF
2. Changes in serum K⁺ concentration reflect absolute potassium change or relative changes into/out of the ICF
3. Active reabsorption occurring in the proximal tubule continues to operate distally
4. K⁺ secretion can occur and depends on passive electrochemical gradients between cell and lumen

Brief notes on hypo and hyperkalemia.

Table 6.4 Predisposing Factors

Hypokalemia	Hyperkalemia
Loss of body fluids <ul style="list-style-type: none"> • diuretics • vomiting • diarrhea • sweating • lactation • gastric drainage Adrenal disorders <ul style="list-style-type: none"> • hyperaldosteronism • stress Congestive heart failure Licorice candy addiction (glyceric acid has an aldosterone-like effect) Alkalosis	Renal Failure; excessive K ⁺ intake; etc. Excessive K ⁺ intake Medications Crush injuries Addison's disease Acidosis Hemolysis

Table 6.5 The Clinical Features

Hypokalemia	Hyperkalemia
Severe weakness Tetany Cardiac arrhythmias Rapid, weak, irregular pulse Decreased reflexes Speech changes Abdominal distention, flatulence, vomiting and paralytic ileus Increased effect of digoxin	Weakness and irritable "twitchy" muscles Fearfulness Malaise Nausea, vomiting and diarrhea Flaccid paralysis ECG changes Cardiac arrest Decreased effect of digoxin

Sodium

Table 6.6 Concentrations of Sodium and Other Electrolytes in ICF and ECF

ICF	ECF
Na ⁺ 10 mmol/L Cl ⁻ 10 mmol/L HCO ₃ ⁻ 10 mmol/L	Na ⁺ 140 mmol/L Cl ⁻ 100 mmol/L HCO ₃ ⁻ 25 mmol/L

Sodium (Na⁺), chloride (Cl⁻) and bicarbonate HCO₃⁻ are the major osmotically active electrolytes in the ECF. In contrast with potassium, the concentrations of these electrolytes **are** greatly influenced by the addition or depletion of water (see McCance & Huether, pp.88-89).

Learning Activity #5—Quick Quiz!!



Answer each question true or false (See the back of this unit for answers).

1. T F Sodium is the major cation of extracellular fluid (ECF)
2. T F Chloride is an example of an anion
3. T F Bicarbonate has a higher concentration in extracellular fluid (careful with this one, it *might* be tricky!)
4. T F Bicarbonate is an example of a cation.

Brief notes on hyper and hyponatremia.

1. Predisposing factors:
See McCance & Huether, 2001, p. 90, paragraph entitled "Isotonic Alterations."
2. Clinical Features

Table 6.7 The Clinical Features

Hyponatremia	Hypernatremia
<p>CNS</p> <ul style="list-style-type: none"> • Depressed sensorium • Seizures • Lethargy • Disorientation • Agitation • Muscle cramps • Depressed reflexes • Cheyne-Stokes respirations • Hypothermia • Death <p>GI</p> <ul style="list-style-type: none"> • Anorexia • Nausea 	<p>Polyuria Polydipsia Flushed skin Dry tongue Dry mucous membranes Oliguria Anuria Elevated body temperature</p>

Final Thoughts

In this unit you have looked closely at buffers, acid-base imbalance and electrolyte imbalance. You will now have a greater understanding of all three aspects of fluid and electrolytes. It is important that you try to use this knowledge in your own clinical setting to further understand the patients/clients that you encounter.

Recommendations

This is not an easy unit! Perhaps you need to go over one or two of the sections again. Take your time in doing this. Or better yet, go over the material that you are not quite sure of with a colleague.

Another interesting way of knowing whether you *understand* the material, is to use a method known as *teachback*. *Teachback* necessarily involves having a willing victim, e.g. an unsuspecting husband or boyfriend, a bored high school student, or an enthusiastic grandmother!

What you do:

1. Obtain willing victim!
2. Tell victim that teachback is painless but requires their undivided attention for about 30 minutes.
3. Select a small amount of the information in this unit (preferably an area that you are having some difficulty with, not a **lot** of difficulty, just **some**).
4. Try and **teach** the content that you have learned **back** to your victim (hence “teachback”).
5. You will be amazed at:
 1. How much you really did learn.
 2. How much you understood.
6. Any “sticky” areas you had in doing teachback are precisely those areas you need to go over again.
7. Who said learning can't be fun! Good Luck!

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Checklist of Requirements

- q** Read Print Companion: Fluid and Electrolytes
- q** Read McCance, & Huether (2001), pp. 85-102
- q** Learning Activity #1
- q** Learning Activity #2: Alkalosis and Acidosis
- q** Learning Activity #3
- q** Learning Activity #4
- q** Learning Activity #5: Quick Quiz

Answers to Learning Activities

Learning Activity #1—Clinical Point

1. In acidosis, an increase in H⁺ excretion is necessary in an attempt to decrease the H⁺ concentration in the blood. This would result in an increase in the blood pH.
2. Hyperkalemia.

Learning Activity #2—Acid-base Imbalance: Respiratory Acidosis

Conditions which may cause respiratory acidosis include: Emphysema; asthma; narcotic/barbiturate poisoning; head injury (leading to depression of the respiratory centre). People with chronic obstructive pulmonary disease (COPD) have a moderate degree of acidosis.

Learning Activity #3—Clinical Example

Clinical Example: Diabetic ketoacidosis

1. Blood pH is abnormally low (acid)
2. Respiratory mechanism → Kussmaul respirations → blow off CO₂ in an attempt to decrease carbonic acid concentration

Acid-base Imbalance Tables

Example 2 Respiratory Alkalosis

Stage of compensation	pH	PaCO ₂ (mmHg)	HCO ₃ (mmol/L)
Absent	7.49 Abnormal ↑	30 Abnormal ↓	26 Normal
Partial	7.47 Abnormal ↑	30 Abnormal ↓	22 Abnormal ↓
Complete	7.44 Normal	32 Abnormal ↓	18 Abnormal ↓

- In respiratory alkalosis the primary problem is decrease in CO₂
- The aim of compensation is to restore the pH to within the normal range
- This is achieved by decreasing HCO₃
- The initial condition shows abnormal pH and PaCO₂ with normal HCO₃
- The aim of compensation has been achieved a normal pH

Example 3 Metabolic Acidosis

Stage of compensation	pH	PaCO ₂ (mmHg)	HCO ₃ (mmol/L)
Absent	7.24 Abnormal ↓	44 Normal	14 Abnormal ↓
Partial	7.28 Abnormal ↓	34 Abnormal ↓	14 Abnormal ↓
Complete	7.35 Normal	28 Abnormal ↓	15 Abnormal ↓

- In metabolic acidosis the primary problem is decrease in HCO₃
- The aim of compensation is to restore the pH to within the normal range
- This is achieved by decreasing the PaCO₂
- The initial condition shows abnormal pH and HCO₃ with normal PaCO₂
- The completely compensated condition shows normal pH and abnormal PaCO₂ and HCO₃
- The aim of compensation has been achieved.

Example 4

Stage of compensation	pH	PaCO ₂ (mmHg)	HCO ₃ (mmol/L)
Absent	7.45 Normal	54 Abnormal ↑	38 Abnormal ↑
Partial	7.51 Abnormal ↑	44 Normal	40 Abnormal ↑
Complete	7.45 normal ↑	48 Abnormal ↑	38 Abnormal ↑

- In metabolic alkalosis the primary problem is increase in HCO₃.
- We know what the aim of compensation is now!
- This is achieved by increasing the PaCO₂.
- The initial condition shows abnormal pH and HCO₃ with normal PaCO₂.
- The completely compensated condition shows normal pH and abnormal PaCO₂ and HCO₃.
- Not completely – only partially.

Learning Activity #4—Clinical Example

18-year-old male in motor vehicle accident

1. All values are normal
2. Fat embolism (see p. 997 in McCance & Huether, 2001).
Flail chest (see p. 1116 in McCance & Huether, 2001)
3. Patient is in acute respiratory distress. As carbon dioxide increases and oxygen decreases, the patient tries to increase the rate and depth of breathing. The patient is hyperventilating (rate 44/min.) but his lungs are not able to get rid of the high carbon dioxide levels because of his flail chest.
4. pH abnormal (acidosis)*
PaCO₂ abnormal (acidosis)*
HCO₃ abnormal (alkalosis)
 - These values indicate respiratory acidosis with partial compensation.
 - The HCO₃ has increased but not enough to return the pH to within normal range.
5. In ICU trauma on mechanical ventilation.

Learning Activity #5—Quick Quiz

1. T
2. T
3. T - I just said that it **might** be tricky!!
4. F