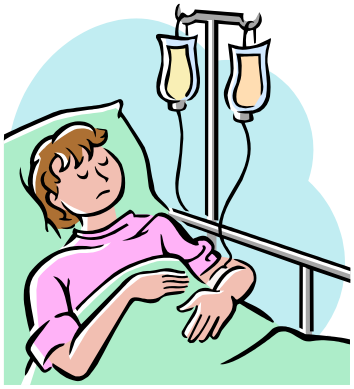
GIPPSLAND HEALTH SERVICES CONSORTIUM

FLUIDS & ELECTROLYTES LEARNING PACKAGE (Revised)



NAME_____

HEALTH SERVICE / DEPARTMENT_____

GRCE Points 4

Revised March 2009, November 2011 & June 2021

Original developed by Paul Osborn 2007
Approved by the Gippsland Region Nurse Educators Group May 2009 & November 2011. Revised by Kate Roberts Graduate Nurse Coordinator Bairnsdale Regional Health Service 2021

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ACKNOWLEDGEMENTS

This package was originally developed by Mr. Paul Osborn, West Gippsland Healthcare Group since then the package has been revised by Educators from the Gippsland Health Services Consortium. Extensive revision by Kate Roberts Graduate Nurse Coordinator Bairnsdale Regional Health Service 2021.

All references used in the development of this package are listed at the end of the SDLP.

INTENDED LEARNING OUTCOMES (ILOS)

On completion of this program, it is expected that participants will be able to:

- **MODULE 1:**
  - Describe the distribution of body water in terms of anatomical compartments
  - Define electrolytes, state the major electrolytes occurring in the body and describe their distribution

- **MODULE 2:**
  - Explain the mechanisms by which fluids and electrolytes move between the anatomical compartments

- **MODULE 2 & 3:**
  - State the mechanisms involved in the regulation of fluids and electrolytes

- **MODULE 4:**
  - Define and state the causes, signs and symptoms and treatment of the following fluid imbalances: dehydration; hypovolaemia; hypervolaemia; and water intoxication

- **MODULE 5 - 10:**
  - Define and state the causes, signs and symptoms and treatment of the following electrolyte imbalances: hyponatraemia and hypernatraemia; hypokalaemia and hyperkalaemia; hypomagnesaemia and hypermagnesaemia; hypocalcaemia and hypercalcaemia; hypophosphataemia and hyperphosphataemia; hypochloraemia and hyperchloraemia

- **MODULE 11 - 12:**
  - Explain how the balance of acids and bases is maintained in the body
  - Define the concepts acidosis and alkalosis
  - Define and state the causes, signs and symptoms and treatment of the following acid-base imbalances: metabolic acidosis; respiratory acidosis; metabolic alkalosis; respiratory alkalosis.
  - Briefly discuss the concept of anion gap

INTRODUCTION

The body's water content and the solutes (electrolytes and other substances) contained in it are referred to as the 'internal environment' and are critical to the physiological processes that sustain life. The amount and distribution of water and the types and concentrates of electrolytes and other dissolved substances must be kept in balance if health is to be maintained.

Where imbalances occur, physiological functioning may be adversely affected and the health of the individual compromised. This state of balance in the body's internal environment is so significant that normal values have been established for all of its constituents. Comparison of the patient's pathology against these normal values is used to detect imbalances where they arise during illness so that corrective measures may be instituted. Reference ranges provided in this program are based on those set out by the Gippsland Pathology Service but, where variations occur those employed by the individual health service should be used.

The assessment and maintenance of a patient's fluid and electrolyte balance is a major nursing responsibility. This self-directed learning package is separated out into **12 modules** to allow you to move at your own pace through each section. The Learning package provides an overview of the anatomy and physiology associated with fluids and electrolytes, examines the causes of selected disorders produced by imbalances in the internal environment, and outlines strategies to manage them.

Module 1: The Body's Internal Environment

Section 1.1 Body Water

Anatomical Considerations

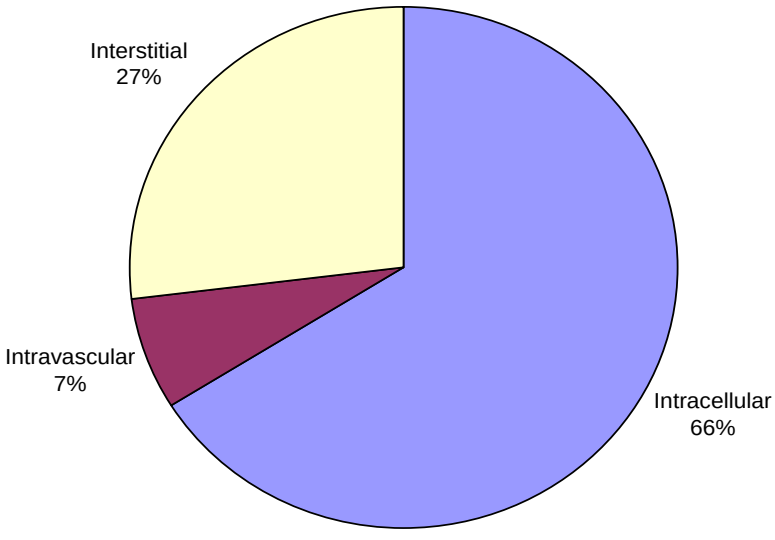
A large proportion of body weight is made up of water and dissolved substances. The actual percentage of water to total weight varies with age, gender and level of body fat. Approximately 75% of the neonate's mass is made up of water, while that of a young adult male is 60% and that of a young adult female is about 50%. As age increases the water to body weight ratio gradually decreases. Also, individuals with higher body fat have proportionately less total body water as, due to the incompatibility of fat and water, very little water is contained in adipose cells.

Body fluids, which play a crucial transport role in cell and tissue functioning, are distributed throughout the body within three types of compartments. The intracellular compartment (i.e. cell interiors) contains about two thirds of the total body water in the form of intracellular fluid or ICF. The other one third occurs outside the cells and is termed extracellular fluid (ECF). It is made up by the interstitial and the intravascular compartments. The interstitial compartment refers to the spaces between cells outside of the blood vessels which contain interstitial fluid, and the intravascular compartment refers to the blood vessels that contain intravascular fluid or plasma. There are also two smaller extracellular compartments, one containing lymph and the other transcellular fluids such as synovial, gastrointestinal, cerebrospinal, pleural, peritoneal, pericardial and intraocular fluids, urine and sweat.

Table 1: Distribution of Body Water

Compartment	% Body Weight
Intracellular fluid (ICF)	**40**
Extracellular fluid (ECF)	**20**
Interstitial	(15)
Intravascular	(5)
Total body water	**60**

Figure 1: Volumes of body fluids in each fluid compartment



Fluid Balance

Water continually leaves and enters the body. It leaves via the kidneys, skin, lungs and large bowel, and is replenished from ingested liquids, water in food, absorbed fluids and water resulting from catabolism. Under normal circumstances intake of fluids approximately equals output, so that the body maintains a constant volume and is in fluid balance.

Table 2: Approximate normal fluid intake and loss in an adult eating 2500 calories per day

INTAKE		**OUTPUT**	
Route	**Amount of gain (mL)**	**Route**	**Amount of loss (mL)**
Water in food	1000	Skin	500
Water from oxidation	300	Lungs	350
Water as liquid	1200	Faeces	150
		Kidney	1500
Total	**2500**	**Total**	**2500**

Internal Movement of Fluid

Water moves relatively freely between the compartments due to the actions of a number of mechanisms, but the volume in each remains relatively stable in the healthy individual.

Fluid interchange between the intracellular and extracellular compartments is regulated primarily by the process of osmosis, while that between the plasma and interstitial spaces is controlled by the interaction of hydrostatic and osmotic forces that operate in the capillaries. These are examined in more detail in 'Fluid and Electrolyte Movement Mechanisms' within Module 2.

Tonicity

Body fluids, because of the varying amounts of dissolved substances they contain, also vary in tonicity. This is a term used to compare the osmolality (i.e. number of dissolved particles per unit of water) of a solution to the normal body fluid osmolality. Fluids may be; isotonic, hypotonic or hypertonic.

Types of body fluids

Isotonic

An isotonic solution has the *same solute concentration* as the surrounding fluid; i.e. each unit of fluid has the same number of dissolved particles. In this solution there is no passive movement of fluid from compartment to compartment through the semipermeable membrane because the osmolality is the same in each. Normal saline solution (0.9% sodium) is considered isotonic because its sodium concentration is very similar to that of blood.

IV fluid type: Isotonic	Osmolality
0.9% Normal saline	300 mOsm/kg
5% Dextrose in water	252 mOsm/kg
4% Dextrose & 1/5 (0.18%) NaCl (saline)	284 mOsm/kg
Compound sodium lactate (CSL) / Hartmann's solution	254 mOsm/kg

Hypotonic

A hypotonic solution is one in which the *concentration of solutes is lower* than that of the surrounding fluid. If, for instance, one solution contains markedly less sodium than the fluid in the adjoining compartment, it is said to be hypotonic. In this situation, fluid would move from the compartment where the concentration is lower into the compartment where it is higher until there is an equal sodium concentration (number of particles per unit of water) in each.

IV fluid type: Hypotonic	Osmolality
0.45% Saline (0.45% NaCl)	154 mOsm/kg

Hypertonic

A hypertonic solution has a *higher solute concentration* than the surrounding fluid. For instance, if a body fluid contains more dissolved sodium than another fluid, is considered hypertonic in comparison. As a result, fluid from the compartment with the lower concentration would move through the semipermeable membrane and mix with the fluid with the higher osmolality until each has the same number of dissolved particles per unit of fluid and equilibrium is established.

IV fluid type: Hypertonic	Osmolality
10% Dextrose	505 mOsm/kg
5% Dextrose & 0.45% saline	432 mOsm/kg
3% Saline	1,026 mOsm/kg
20% Mannitol	1,098 mOsm/kg
Albumin	
Parenteral Nutrition	

Section 1.2 Body electrolytes

The body fluids contain a variety of dissolved chemicals or solutes, which may be classified as either electrolytes or non-electrolytes.

Dissolved chemical or solutes

Electrolytes

Electrolytes constitute about 95% of the solutes in body fluids. They are electrically charged particles which dissociate into ions when dissolved in body water. The ions obtain their electric charge by virtue of having gained or lost an electron when dissolved. Electrolytes occur in body fluid as acids, bases and salts. Most are inorganic compounds but a few, such as some proteins are organic.

Non-electrolytes

Non-electrolytes, on the other hand, are substances that remain molecularly intact; when dissolved in water. Their structure remains essentially unchanged and they do not dissociate, ionise or carry an electric charge in solution. Non-electrolytes are primarily made up of organic compounds like as glucose, urea and creatinine.

Types of Electrolytes

Because they are electrically charged, electrolytes exhibit a positive or negative polarity.

Cations

Ions with a positive charge are called cations and occur where one or more electrons have been lost in a chemical reaction with body fluid, so that the number of protons exceeds the number of electrons in their atomic structure. Important cations in the body include sodium (Na^{+}), potassium (K^{+}), calcium (Ca^{++}) and magnesium (Mg^{++}).

Anions

Negatively charged ions (anions) are formed where atoms gain electrons so that, as a result, there are more electrons than protons. Important anions in body fluid include chloride (Cl^{-}), bicarbonate (HCO_3^{-}), phosphate (HPO_4^{-}) and sulphate ($SO_4^{=}$).
The major body electrolytes according to *polarity* or *electrical charge* are:

Cations	Anions
Sodium	Bicarbonate
Potassium	Chloride
Calcium	Phosphate
Magnesium	Proteins

Electrolyte Functions

Electrolytes are essential to the maintenance of good health for several reasons. They include many of the essential minerals; they control the osmotic movement of water between the three body compartments; they help control body water volume; and assist in maintaining the acid-base balance necessary for normal cellular activities.

An overview of the functions of the major body electrolytes and the mechanisms by which their levels are regulated is provided below under ‘Regulation of Fluids and Electrolytes’ in Module 3.

Distribution of Electrolytes

While similar types of electrolytes are found in each of the body's fluid compartments, and while they move from compartment to compartment, they are not evenly distributed, with concentrations varying considerably, as shown in Table 3.

Table 3: Normal electrolyte content of body fluids*

		Extracellular		Intracellular
Electrolyte	**Type of body electrolyte**	**Intravascular (Vessels)**	**Interstitial (Tissues)**	**Intracellular (Cells)**
Sodium (Na^+)	Cation	142 mmol/L	146 mmol/L	15 mmol/L
Potassium (K^+)	Cation	5 mmol/L	5 mmol/L	140 mmol/L
Calcium (Ca^{++})	Cation	2.5 mmol/L	1.5 mmol/L	1 mmol/L
Magnesium (Mg^{++})	Cation	1 mmol/L	0.5 mmol/L	13.5 mmol/L
Chloride (Cl^-)	Anion	102 mmol/L	114 mmol/L	1 mmol/L
Bicarbonate (HCO_3^-)	Anion	27 mmol/L	30 mmol/L	10 mmol/L
Phosphate (HPO_4^-)	Anion	0.65 mmol/L	0.65 mmol/L	32.3 mmol/L

- Note that the electrolyte level of the intravascular and interstitial fluids (extracellular) is approximately the same and that sodium and chloride contents are markedly higher in these fluids, whereas potassium, phosphate and protein contents are markedly higher in intracellular fluid.

Differences in individual ion concentrations also occur between various types of extracellular fluids. Gastric secretion, for instance, is acid and consequently has a high concentration of hydrogen ions. Pancreatic secretion is more alkaline and contains a high concentration of bicarbonate. Sodium ions are also highly concentrated in both of these fluids, as well as in bile.

Electrolyte Balance

In the healthy individual the ratio of cations to anions in each of the body fluids is relatively constant, as is the concentration of the various types of ions in them. This applies despite the fact that electrolytes are lost from the body in various ways.

Loss occurs mainly from the kidneys, but smaller numbers of electrolytes leave the body via the skin, lungs and bowel. The kidneys, as a result of hormonal influences, selectively excrete electrolytes, retaining those that are required to maintain normal body fluid composition. Replacement electrolytes are normally absorbed from dietary intake, although alternative routes such as IV infusions may be required where diet is inadequate or where nutrition cannot be taken orally.

Summary: Key Learning Points

A rudimentary understanding of chemistry is necessary to understand fluid movement in the body's internal movement. This module discusses bodily water in relation to anatomical considerations and fluid distribution. Fluid balance concepts were discussed. The three (3) types of body fluids were covered; isotonic, hypotonic and hypertonic fluids. Examples of common IV fluids with various tonicity were outlined. Body electrolytes were mentioned; including electrolytes and non-electrolytes as were the types of electrolytes; cations and anions. Electrolyte functions and their distribution in body compartments were elaborated on.

QUESTIONS & ANSWERS

Module 1: The Body's Internal Environment

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. The most abundant anion in the body's extracellular fluid is:

a) Phosphate
b) Sodium
c) Chloride
d) Bicarbonate

2. In which of the following groups of people is the percentage of water in the total body mass highest?

a) Young males
b) Young females
c) Obese individuals
d) Neonates

3. Which of the following groups is made up solely of cations?

a) Sodium, magnesium, chloride
b) Potassium, calcium, magnesium
c) Bicarbonate, phosphorus, chloride
d) Sodium, calcium, chloride

4. The largest proportion of total body water occurs in the:

a) Intracellular compartment
b) Interstitial space
c) Intravascular space
d) Extracellular compartment

5. The most abundant cation in the body's intracellular fluid is:

a) Potassium
b) Sodium
c) Calcium
d) Phosphate

6. A solution in which the concentration of solutes is <u>higher</u> than that in the surrounding fluid is:

a) An isotonic solution
b) A hypertonic solution
c) A hypotonic solution
d) A non-electrolyte solution

7. A solution in which the concentration of solutes is <u>lower</u> than that in the surrounding fluid is:

a) A hypotonic solution
b) A non-electrolyte solution
c) An isotonic solution
d) A hypertonic solution

8. A solution in which has the <u>same</u> solute concentration as the surrounding fluid is:

a) A non-electrolyte solution
b) A hypertonic solution
c) A hypotonic solution
d) An isotonic solution

9. When dissolved in water, which of the following substances displays the properties of a 'non-electrolyte' or a substance that remains molecularly intact?

a) Sodium
b) Phosphorus
c) Glucose
d) Calcium

10. In normal healthy individuals, most <u>electrolyte excretion</u> occurs via the:

a) Lungs
b) Skin
c) Bowel
d) Kidneys

Module 2: Fluid and Electrolyte - Movement Mechanisms

Body water and the substances dissolved in it are able to pass through the semipermeable membranes that separate the **three compartments**. Such movement is continual and occurs as oxygen and nutrients are transported to the cells, and wastes removed from them by the blood. The volume of water and the concentration of solutes in each compartment, however remain relatively unchanged.

Passive, mediated and active transport mechanisms are responsible for the movement of body water and its solutes from compartment to compartment.

Section 2.1 Transport mechanisms

'Passive' transport mechanisms

Diffusion, *osmosis* and *filtration*, are all examples of passive transport, where water and small charged particles or *ions* move through the membrane without any expenditure of cellular energy.

Diffusion

Diffusion is the movement of a solute (dissolved particles) from an area of greater concentration to an area of lesser concentration (Figure 1). It includes dispersion of the solute throughout the fluid contained within a compartment wall as well as solute movement through the membrane separating the two compartments until its concentration is equal on both sides of the membrane.

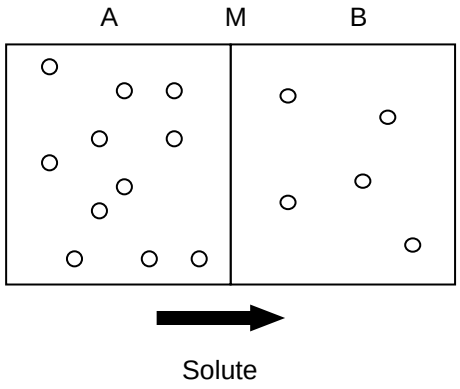


Figure 1: Diffusion: Solute moves through membrane (M) from area of greater concentration (A) to area of lesser concentration (B) until concentration on both sides is equal.

The semipermeable walls of blood vessels and cells contain tiny pores through which small molecules and electrolytes diffuse freely. Large molecules such as glucose are too large to pass through membrane pores and are assisted in crossing the membrane by a carrier substance in a process called facilitated diffusion.

Osmosis

Osmosis is the movement of water across a semipermeable membrane from an area of lower concentration of solute to an area of higher solute concentration (Figure 2). The water moves to dilute the more highly concentrated solution until equilibrium is reached on both sides of the membrane.

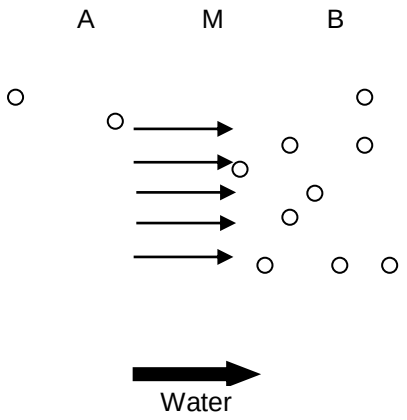


Figure 2:
Osmosis: water moves from area of less solute concentration (A) through a membrane (M) to area of great solute concentrations (B) until concentration of solute on both sides of the membrane is equal.
Compartment B will have to expand (as shown by dotted lines) to accept additional water.

Osmosis is the main mechanism involved in the movement of fluid between the cells and the extracellular compartment. In this process, water moves to the area with the greater number of dissolved particles. Particles may be whole molecules or ions. Electrolytes exert a much greater effect on osmosis than nonelectrolytes because, when they are dissolved in water, electrolytes break down into two (2) or more particles, while nonelectrolytes remain unchanged.

For example, sodium chloride (NaCl) breaks down into two ions (Na^+ and Cl^-) when dissolved in water, thus producing two particles. Calcium chloride ($CaCl_2$) contributes even more particles as it forms three ions (Ca^{2+}, Cl^- andCl^-) when it dissolves. Calcium chloride produces a three-time stronger solute concentration and therefore a much stronger osmotic effect than the nonelectrolyte glucose, which remains molecularly unchanged and thus only contributes one particle to the solution.

Furthermore, once an electrolyte has broken down, its ions can attract ions of the opposite charge, so that if equal amounts of Ca^{2+} and Na^+ are dissolved, the calcium ion will attract twice as many oppositely charged chloride ions than the sodium.

On the other hand, protein molecules, because of their large size, normally have little movement between compartments. Their presence, especially in the intravascular fluid, creates a pressure called colloid osmotic or oncotic pressure, which functions to hold water within the blood vessel.

Filtration

Filtration in the context of body fluids and electrolytes refers to the process by which fluid is forced through a semipermeable membrane due to pressure differences on either side of it. Higher pressure on one side of the membrane causes some the fluid on that side to move to the other where the pressure is lower to equalise the difference. In the process, some of the solutes (particles) are separated from the water as they cannot pass through the membrane.

Filtration is important in the movement of fluid and electrolytes between the intravascular (plasma) and the interstitial compartments. It occurs in the capillaries, specifically at the junction of the arteriole and the venule. At the arteriole end, hydrostatic fluid pressure caused by heart contractions is significantly higher than that in the surrounding interstitial space, so fluid is filtered (forced) out of the capillary. At the venous end of the capillary, the oncotic (or osmotic) pressure exerted by plasma proteins, which normally do not cross the capillary membrane and so remain within the vessel, exceeds the hydrostatic force, so that fluid is drawn back into the circulation from the extravascular spaces. A balance between the fluid filtered from the arteriole and that reabsorbed in the venule is maintained, with the hydrostatic or 'push' force being balanced by the oncotic or 'pull' force.

'Mediated' transport mechanisms

Mediated transport involves *transport proteins* that either bind with a specific type of solute molecules to transfer them across the membrane, or form a water-filled channel through the membrane which permits the passage of specific ions.

Passive mediated Transport

In passive mediated transport, the *protein transporter* moves the solute particles through the cellular membrane *without the expenditure of cellular energy*.

'Active' transport mechanisms

Active mediated transport systems or pumps, on the other hand, involve the *protein transporter* moving the solute particle against the concentration gradient which requires an *energy source*. Many pumps, like the the *sodium and potassium pump*, use ATP to facilitate movement.

Summary: Key Learning Points

A number of transport mechanisms are involved in the movement of fluid and electrolytes in the body's three (3) compartments. These transport mechanisms are 'passive' requiring no expenditure of cellular energy, 'mediated' involving transport proteins and solute particles movement without expenditure of cellular energy and 'active' mechanisms involve the protein transporter using ATP to facilitate movement.

QUESTIONS & ANSWERS

Module 2: Fluid and Electrolyte - Movement Mechanism

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. The process that is primarily responsible for the movement of fluid between the intracellular and extracellular compartments is:

a) Diffusion
b) Active transport
c) Osmosis
d) Filtration

2. Fluid and electrolytes are moved from the capillaries to the interstitial space and back again by the actions of both;

a) Active and passive mediated transport
b) Osmosis and diffusion
c) Diffusion and mediated transport
d) Hydrostatic and oncotic forces

3. Diffusion is a 'passive' transport mechanism that is defined as:

a) The movement of dissolved particles (a solute) from an area of greater concentration to an area of a lesser concentration
b) The movement of a solute from an area of lesser concentration to an area of a greater concentration
c) The movement of a solute between compartments resulting in an unequal concentration on both sides of the compartments
d) The movement of water across a semi-permeable membrane

4. The sodium and potassium pump is an example of which type of transport mechanism?

a) 'Mediated' transport mechanism
b) 'Active' transport mechanism
c) 'Passive' transport mechanism
d) 'Passive mediated' transport mechanism

Module 3 Fluids and Electrolytes - Regulation

In addition to the passive and active regulating mechanisms outlined in Module 2, fluid and electrolyte balance is also strongly influenced by the actions of hormones, particularly antidiuretic hormone (ADH), aldosterone and parathyroid hormone. The common electrolyte, both anions and cations listed below also regulate fluid and electrolyte movement.

Section 3.1 Hormonal regulation

Antidiuretic hormone (ADH)

ADH is *produced in the hypothalamus* and stored and released from the posterior pituitary gland. It acts on the renal tubules to retain water and decrease urinary output.

Aldosterone

Aldosterone is *secreted by the adrenal cortex* and acts on the renal tubules causing them to reabsorb sodium and excrete potassium. It also increases circulatory volume by reabsorbing water along with sodium.

Parathyroid hormone

Parathyroid hormone as the name suggests is *produced by the parathyroid glands*. It promotes absorption of calcium from the intestine and the release of calcium from bone. It also increases the excretion of phosphate ions by the kidneys.

Section 3.2 Common electrolytes effect on regulation

Sodium (Cation)

Sodium (Na^+) is the most abundant extracellular ion, compromising about 90% of all extracellular cations. Along with its constituent anions, chloride and bicarbonate, sodium levels regulate osmosis and therefore control water balance. Other important functions include involvement in the maintenance of interstitial and intravascular fluid volumes, neuromuscular function in conjunction with potassium and calcium, regulation of acid-base balance, cellular chemical reactions and membrane transport.

The kidneys maintain sodium within the serum range of **135 to 148 mmol/L**, primarily through reabsorption in the renal tubules which is, in turn, influenced by hormonal and neural mediators. Blood sodium levels are controlled primarily by the hormone aldosterone which is produced by the adrenal cortex. Aldosterone is secreted in response to reduced blood volume, diminished cardiac output, decreased extracellular sodium, raised extracellular potassium or physical stress. It acts on the distal convoluted tubules and collecting ducts of the kidneys, causing them to increase their reabsorption of sodium, so that sodium moves from the filtrate back into the blood.

Potassium (Cation)

The normal range for the potassium concentration in **serum is 3.8 – 4.9 mmol/L** and in plasma 3.4 – 4.5 mmol/L. Potassium (K^+) is an electrolyte that occurs primarily in the intracellular compartment. As the predominant cation in intracellular fluid, it is

important in regulating intracellular osmolality and fluid balance. It is also essential for the deposition of glycogen and glucose in the cells of the liver and skeletal muscles.

Potassium plays an important part in the transmission and conduction of nerve impulses, the maintenance of normal cardiac rhythm and the contraction of both smooth and skeletal muscle.

Potassium is filtered in the glomerulus, but normally 90% is reabsorbed in the proximal tubule and loop of Henle. The distal tubule secretes potassium and also determines the amount to be excreted. When potassium concentrations as indicated by levels in the peritubular capillaries are high, potassium is secreted by passive transport into the urine by the distal tubules. Decreased plasma concentrations, on the other hand, result in decreased secretion by the distal tubule, although small amounts are still lost. Changes in the rate of urine flow through the distal tubule also affect potassium secretion. When the urine flow rate is high, the concentration of potassium in the urine within the distal tubule is low. This causes the movement of potassium from the peritubular capillaries into the urine.

The hormone aldosterone is also involved in potassium regulation. Increased plasma potassium concentrations cause the release of aldosterone, which stimulates the secretion of potassium into the urine via the distal renal tubules. Aldosterone also increases the secretion of potassium by the sweat glands. Insulin plays a part in potassium regulation in that it facilitates its movement out of the blood and into liver and muscle cells.

Calcium (Cation)

Calcium (Ca^{2+}) occurs mainly in the extracellular compartment. It is an essential component of bones and teeth and is also necessary for blood clotting, muscle contraction, normal heart beat and chemical transmitter function.

Blood calcium levels are regulated by the actions of two hormones to maintain them within the **serum range 2.10 to 2.60 mmol/L**. When they are low, parathyroid hormone (or parathormone) is released. This stimulates osteoclasts to break down bone matrix and release calcium into the blood. It also increases the absorption of calcium from the gastrointestinal tract and stimulates reabsorption from the urinary filtrate in the kidney. Where levels are elevated, the hormone calcitonin from the thyroid stimulates osteoblast action which causes the removal of calcium from the blood for deposition in the bones, and inhibits osteoclast activity which reduces calcium absorption from bony structures. These two (2) hormones therefore interact through negative feedback to control blood calcium levels.

Magnesium (Cation)

Magnesium (Mg^{2+}), which is primarily an intracellular electrolyte, activates the enzymes required for the production of cellular energy from the breakdown of ATP into ADP. It also causes neuromuscular excitability and plays an important role in smooth muscle contraction and relaxation.

Serum magnesium levels in the range **0.8 to 1.0 mmol/L** are regulated by a balance between gastrointestinal absorption and renal excretion. Absorption occurs in the small intestine and is vitamin D dependent. Excretion occurs in the glomeruli, but the

rate varies depending on serum levels. Most is reabsorbed, mainly in the loop of Henle and the proximal tubule. It is also thought that the endocrine system is involved in magnesium regulation in that aldosterone secretion may affect rates of reabsorption or excretion.

Chloride (Anion)

Chloride (Cl^-) is the major anion occurring in the extracellular fluid, with normal serum levels ranging from **95 to 110 mmol/L**. It moves in and out of cells with sodium and potassium, and combines with cations to form such important compounds as sodium chloride, hydrochloric acid, potassium chloride and calcium chloride. It is important because:

- Due to its relationship with sodium, it helps maintain serum osmolality and water balance;
- It provides electroneutrality, particularly in relation to sodium;
- It combines with hydrogen in the glands of the gastric mucosa to form hydrochloric acid;
- It is a component of cerebrospinal fluid.

Chloride levels are determined by intake and excretion and reabsorption in the kidneys. Most is ingested in the form of salt (NaCl) and absorbed in the intestines. Only a small amount is excreted in the faeces. It is also produced in the stomach in the form of hydrochloric acid.

Chloride moves readily between the extracellular fluid and the intracellular fluid and as a result is involved in regulating osmotic pressure differences between these two compartments. Its movement occurs mainly as a result of passive transport and generally follows the active transport of sodium. Thus, like sodium, regulation of chloride is under the control of aldosterone, although indirectly, as aldosterone controls sodium reabsorption in the kidneys and chloride passively follows sodium.

Consequently, increases or decreases in chloride are proportional to changes in sodium levels.

Phosphate (Anion)

Phosphate (HPO_4^{2-}) is the principal anion in the intracellular fluid. Regulation of serum phosphate levels between **0.80 and 1.5 mmol/L** occurs primarily in the kidneys through which about 90% is excreted. The remaining 10% is excreted via the GI tract. If serum levels rise, the kidneys increase the rate at which phosphorus is excreted; if they fall, the rate of tubular reabsorption increases. This process is influenced by hormonal control, specifically parathyroid hormone which is secreted in response to changes in calcium levels.

About 85% of the body's phosphate exists in the bones and teeth in combination with calcium; 14% is found in soft tissues and less than 1% occurs in extracellular fluid. It is an important structural component of bones and teeth, and, in addition plays an important role in:

- maintaining membrane integrity (cell membranes are made up of phospholipids);
- the formation of nucleic acids (DNA and RNA) and the key compounds ATP and creatine phosphate that provide energy for cellular functions;
- acid-base buffering reactions;
- muscle function;
- neurological activity; and
- carbohydrate, protein and fat metabolism.

It is also vital to the formation of the compound in red blood cells responsible for delivering oxygen to the tissues and is involved in platelet function and white cell phagocytosis.

Total body phosphates are determined by dietary intake, kidney excretion, hormonal regulation and shifts between fluid compartments. Phosphorus is absorbed from the GI tract, mainly in the jejunum, and the amount absorbed is proportional to the amount ingested.

Calcium levels and phosphate levels have a close but inverse relationship related, so that if one is low the other is elevated. If calcium is low, parathyroid hormone secretion is increased to promote reabsorption from bones and additional absorption from the jejunum. While this increases calcium levels, it also increases phosphates which were already high. To address raised phosphate levels, parathyroid hormone then acts on the kidneys causing them to increase the excretion of phosphates. A reduction in parathyroid hormone levels, on the other hand, promotes phosphorus reabsorption by the kidneys which raises serum concentrations.

Summary: Key Learning Points

Module 3 discussed the effect of three (3) main hormones; ADH, aldosterone and parathyroid hormone. It also discussed the effects common electrolytes have on fluid and electrolyte movement. Four (4) main cations; sodium, potassium, calcium and magnesium and two (2) main anions were mentioned; chloride and phosphate and their effect on fluid regulation within the body. The normal ranges of these electrolytes were discussed.

QUESTIONS & ANSWERS

Module 3: Fluid and Electrolyte - Regulation

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. The substance that promotes absorption of calcium from the intestine and the release of calcium from the bones is known as:

a) Aldosterone
b) Parathyroid
c) Calcitonin
d) Antidiuretic hormone (ADH)

2. The hormonal influence exerted by aldosterone is an important factor in the excretion of which electrolyte:

a) Sodium
b) Chloride
c) Calcium
d) Potassium

3. The substance produced in the hypothalamus and closely involved in the regulation of fluids and electrolytes due to its action on the renal tubules is:

a) Parathyroid hormone
b) Antidiuretic hormone
c) Aldosterone
d) Calcitonin

4. Which two (2) electrolytes have a close but inverse relationship to each other:

a) Sodium and potassium
b) Calcium and magnesium
c) Chloride and phosphate
d) Calcium and phosphate

Module 4: Fluid and Electrolyte Imbalances

Almost all medical-surgical conditions threaten fluid and electrolyte balance. There may be deficits or excesses of water, particular electrolytes or acids or bases which occur individually or in combination. Common conditions involving such imbalances are discussed below.

Section 4.1 Fluid imbalances

While the body is able to correct minor variations in fluid volumes, sometimes imbalances occur that cannot be readily redressed by normal regulatory mechanisms, and problems involving fluid deficits or excesses arise. These include dehydration, hypovolaemia, hypervolaemia and water intoxication.

Section 4.1.1. Dehydration

As previously noted, the body replenishes water lost via the kidneys, skin, lungs and bowel with water derived from ingested fluids, absorption and catabolism. However, if water replacement does not keep place with loss, fluid can be lost from the cells in a process called dehydration.

This condition occurs because the loss of extracellular fluid causes increased solute concentrations and a resultant rise in sodium levels in the blood. This causes water molecules to move out of the cells and into the blood to re-establish balance between the fluids in the extracellular and intracellular spaces. In association with increased fluid intake and increased water retention in the kidneys, this process is usually effective in maintaining balance.

However, if the loss of extracellular fluid is excessive or ongoing and is not adequately replaced, water will continue to move out of the cells and into this compartment. Continual shift causes the cells to shrink and loss of water interferes with their metabolic processes. Unless intracellular fluids are replaced, cell damage will occur.

Dehydration – Causes

Dehydration can be caused by inadequate fluid intake and/or increased rates of fluid loss. Examples of the latter include prolonged fever (diaphoresis), renal failure (inability to reabsorb water), protracted diarrhoea or vomiting, diabetes insipidus (excessive diuresis due to failure to secrete antidiuretic hormone) and hypoglycaemia (production of large volumes of dilute urine). Groups at greatest risk of dehydration are confused, immobile or comatose patients who cannot respond appropriately to the sensation of thirst, older patients because of their lower body water content, and infants who cannot drink independently and cannot concentrate urine effectively.

Dehydration – Signs & Symptoms

Signs and symptoms of dehydration include:

- dizziness, weakness
- complaints of extreme thirst
- dry skin and mucous membranes
- poor skin turgour
- elevated temperature because less water is available for sweat
- concentrated urine (unless the patient has diabetes insipidus)
- changes in mental functioning
- tachycardia
- decreased urine output
- elevated haemotocrit
- elevated serum osmolality
- raised serum sodium
- high urine specific gravity (unless the patient has diabetes insipidus)

Dehydration – Treatment

Dehydration is treated by rectifying the cause of the fluid deficit and replacing lost fluids. However, because dehydration produces high blood solute concentrations, hypertonic replacement fluids must be avoided. Oral fluids, if employed, should be salt-free. Where the IV route is used, hypotonic, low sodium solutions (e.g. 5% glucose) are generally prescribed and administered gradually to avoid cerebral oedema which may occur if brain cells swell as a result of rapid absorption of fluid from the vascular system.

Section 4.1.2. Hypovolaemia

Hypovolaemia is a condition in which there is isotonic fluid loss from the extracellular space. If it is not detected early and treated appropriately it may progress to hypovolaemia shock.

Hypovoleamia – Causes

It may occur as a result of excessive extracellular fluid loss due to such conditions as:

- haemorrhage
- increased urination associated with diabetes mellitus
- diarrhoea and/or vomiting
- excessive diuretic therapy
- fever/excessive sweating
- renal failure with increased micturition
- fistula.

Hypovolaemia may also be associated with *third-space fluid shift* which occurs when fluid moves out of intravascular spaces but not into the cells. Instead, it may move into bodily spaces like the abdominal, pleural or pericardial cavities. This fluid shift occurs as a result of increased capillary membrane permeability or changes in osmotic pressure. It may be due to such complications as:

- burns
- intestinal obstruction
- peritonitis
- heart failure
- liver failure
- pleural effusion
- crush injuries

Where volume depletion is less than about 15% of total circulating blood, the body attempts to compensate by increasing the heart rate. This may also be accompanied by orthostatic hypotension, restlessness, anxiety, decreased urine output but still more than 30 mL/hour, delayed capillary refill and pallor of the extremities.

Hypovolemia – Signs & Symptoms

Further reduction of circulating volume produces symptoms of hypovolaemia and impending hypovolaemic shock. These include:

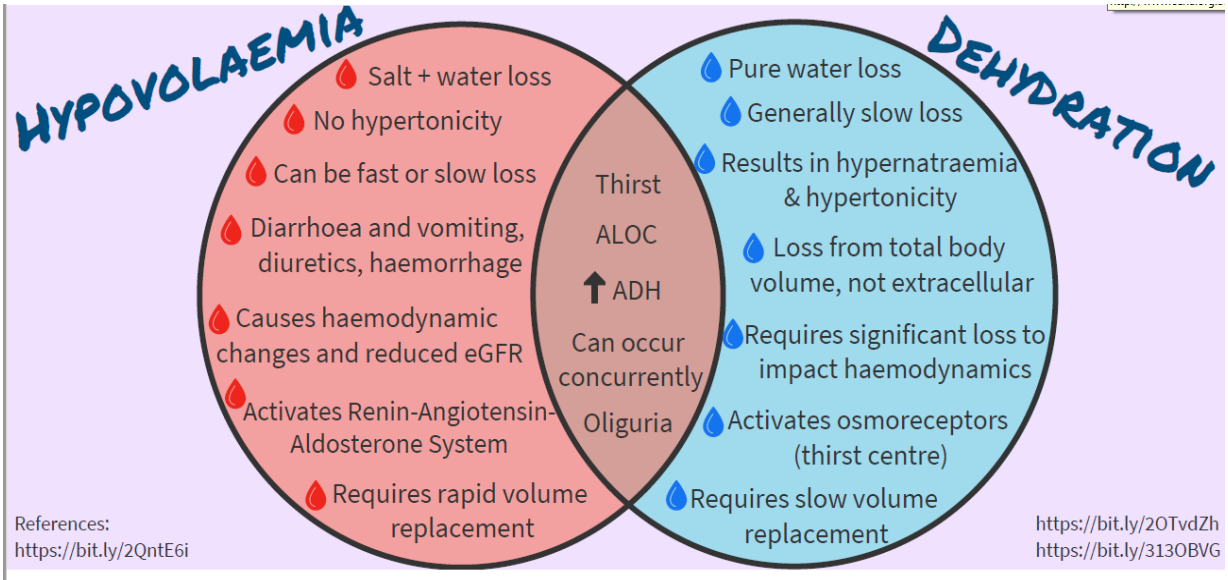
- deteriorating mental status, with increasing restlessness, irritability and anxiety progressing to unconsciousness
- thirst
- tachycardia
- delayed capillary refill
- orthostatic hypotension progressing to marked hypotension
- falling urine output to below 10 mL/hour
- cool, pale skin over the extremities
- flattened jugular veins
- decreased central venous pressure
- weak or absent peripheral pulses
- weight loss.

Moderate intravascular volume depletion (about 25%) is accompanied by increasing confusion and irritability and extreme thirst. The skin is cool and clammy, the pulse rapid and thready and the blood pressure drops. Urine output decreases to between 10 and 30 mL/hour.

Severe, hypovolaemia occurs with a decrease of 40% or more of circulating blood volume and may lead to hypovolaemic shock. The patient becomes tachycardic and hypotensive. Peripheral pulses are weak or absent. The skin is cool and may be mottled or cyanotic. Urine output drops to below 10 mL/hour and unconsciousness may occur.

Hypovolemia – Treatment

Treatment includes replacing lost fluids with fluids of the same concentration to help restore blood volume and normalise blood pressure. Because oral fluids are unlikely to be adequate, isotonic fluids such as normal saline or Compound Sodium Lactate (Hartmann's solution) are administered IV to expand the circulating volume. Administration is rapid, with large volumes of fluid given over short periods, usually via a large bore cannula. This is followed by an infusion of plasma protein and, if the cause of hypovolaemia is haemorrhage, a blood transfusion. A vasopressor may be ordered to help maintain blood pressure until the underlying cause is treated. Oxygen therapy is also initiated to maintain tissue perfusion.



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Section 4.1.3. Hypervolaemia

Hypervolaemia is a condition in which there is an excess of fluid (water and sodium) in the extracellular compartment that cannot be regulated by the body's normal compensatory mechanisms.

Extracellular fluid volume increases in the interstitial or intravascular compartments are usually rectified by adjustments to aldosterone, antidiuretic hormone and atrial natriuretic peptide which cause the kidneys to excrete more sodium and water. However, if the hypervolaemia is severe or prolonged, or if there is poor cardiac function, these compensatory mechanisms may be ineffective, with the result that fluid is forced out of the blood vessels and into the interstitial space causing oedema. Individuals with renal or cardiovascular impairments, especially the elderly, are susceptible to hypervolaemia which may produce pulmonary oedema and subsequent heart failure.

Hypervolemia – Causes

Hypervolaemia may be caused by excessive fluid intake, excessive sodium intake, retention of fluid or sodium or a shift of fluid into the intravascular compartment from the interstitial space. Excessive sodium or fluid intakes are usually due to dietary issues, although inappropriate IV treatment may be the cause amongst a small number of hospital patients. Fluid and sodium retention may be influenced by a number of factors including heart failure, cirrhosis of the liver, nephritic syndrome, corticosteroid use and low dietary protein intake. Fluid shift from the interstitial compartment to the intravascular space may be caused by such factors as the administration of hypertonic fluids or plasma proteins.

Hypervolemia – Signs & Symptoms

Signs and symptoms of hypervolaemia are the basis on which diagnosis is made. Oedema and indicators of increased cardiac output are the two major manifestations. Oedema arises from the hydrostatic pressure that builds up in the blood vessels, forcing fluid out into the surrounding tissues. It is initially evident in dependent areas, particularly the sacrum and buttocks when the patient is lying or in the legs and feet on standing. From these areas it may progress to anasarca, a condition of severe, generalised oedema. Oedematous skin is puffy, cool to the touch and pits when compressed. The condition also produces weight gains, with increases of 5% - 10% considered mild to moderate and those over 10% more severe.

Oedema may also occur in the lungs, with fluid backing up in the pulmonary arteries due to inefficient pumping by the left side of the heart. Hydrostatic pressure forces fluid out of the vessels and into the interstitial and alveolar spaces causing pulmonary oedema. This results in shortness of breath, tachypnoea and a frequent cough. Pink, frothy sputum is a key indicator of pulmonary oedema.

Cardiac output increases in an effort to compensate for the increased circulating volume. This is seen in rises in blood pressure and a rapid and bounding pulse, both of which drop as cardiac failure sets in. Additional indicators of heart failure include S_3 gallop (a third heart sound) and distended veins in the hands, when raised above the level of the heart and neck.

Hypervolemia – Treatment

Treatment of hypervolaemia involves management of causative factors along with sodium and fluid restrictions, diuretics and medications to prevent complications, especially pulmonary oedema and heart failure. If the patient has pulmonary oedema, drugs such as nitroglycerin may be prescribed to dilate the vessels and reduce pulmonary congestion. If heart failure is present it is treated with medications to increase to strength of contractions and slow their rate. Oxygen may be ordered to support the patient.

Section 4.1.4. Water Intoxication

This condition is related to toxicity and arises when excess extracellular fluid moves into the cells. The extracellular fluid is hypotonic to that in the cells and consequently, is shifted by osmosis into the cells where there is less fluid and a higher concentration of solutes. As a result, the cells swell.

Water intoxication – Causes

It may be caused by neurological disorders, pulmonary conditions, tumours, some drugs and some operative procedures that precipitate syndrome of inappropriate antidiuretic hormone secretion (SIADH) which in turn causes the body to retain water. Water intoxication may also occur in response to rapid infusions of hypotonic solution or to psychogenic polydipsia, which is a compulsive desire to drink large amounts of water even though it is not needed.

Water intoxication – Signs & Symptoms

Signs and symptoms are related to low sodium levels and raised intracranial pressure that accompanies the swelling of cells in the brain. Early signs of raised intracranial pressure include headache, abnormal behaviour, confusion, irritability, lethargy, nausea, vomiting, muscle weakness, cramping, twitching, thirst, dyspnoea on exertion and slowed mental processes. Later signs include pupillary changes, bradycardia, widened pulse pressure, seizures and coma.

Water intoxication – Treatment

Treatment is based on correcting causative factors, restricting fluid intake and avoiding hypotonic solutions until sodium levels rise.

Summary Learning Points:

Common conditions caused by fluid and electrolyte imbalances were discussed. Namely, dehydration, hypovolaemia, hypervolaemia and water intoxication.

The causes, signs and symptoms and treatment of all these various conditions were briefly outlined.

QUESTIONS & ANSWERS

Module 4: Fluid and Electrolyte Imbalances

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. Common factors associated with dehydration:

a) Activates Renin-Angiotensin-Aldosterone System (RAAS)
b) Requires subtle loss to impact haemodynamics
c) Can be fast or slow loss
d) There is pure water loss

2. A 75-year-old patient presents with end stage liver failure. He is jaundiced and his body weight has increased by 20 kilograms. His main symptoms are shortness of breath, general swelling, and lethargy. What is the likely cause of fluid shift in his condition:

a) Hypovolaemia due to fluid moving out of the ECF compartment
b) Hypovolaemia due to fluid moving out of the ICF compartment
c) Hypervolaemia due to fluid moving into the ECF compartment
d) Hypervolaemia due to fluid moving into the ICF compartment

3. Oedema is a significant sign of:

a) Dehydration
b) Hypovolaemia
c) Hypervolaemia
d) Water intoxication

4. In the condition known as water intoxication

a) The end result is damage of cells due to shrinkage
b) Excess ECF fluid moves into the cells
c) The ECF is hypotonic to that in the cells
d) The ICF is hypertonic to that in the cells

ECF = Extracellular fluid
ICF = Intracellular fluid

Module 5: Specific - Sodium Imbalances

In the following 6 modules about electrolyte imbalances the adult serum ranges are predominantly discussed. The ranges for sodium, potassium, magnesium, calcium and phosphorus are taken from the electronic Therapeutic Guidelines (eTG), whilst the range for chloride is based on the normal ranges used at the Dorevitch Laboratory based in Bairnsdale.

Background

The normal range of the serum or plasma sodium concentration is **135 – 148 mmol/L**. Sodium levels are determined by the amount ingested and absorbed and the rate at which it is excreted by the kidneys, gastrointestinal tract and skin. Sodium is very closely associated with water in the body and variations in one strongly affect the other. Under normal circumstances, the body's compensatory mechanisms are effective in maintaining sodium and water within the normal range and correcting any minor imbalances that may occur.

When sodium levels rise, the thirst sensation is initiated so that the individual drinks more, increasing fluid volume and decreasing osmolality. Furthermore, antidiuretic hormone is released which causes the kidneys to retain water which helps dilute sodium concentration and return osmolality to normal. When sodium levels fall or there is an excess of body water, serum osmolality decreases. This inhibits the thirst sensation which reduces the intake of water, thus increasing osmolality. In addition, it suppresses the release of antidiuretic hormone so that the kidneys excrete more water, increasing sodium concentration and normalising osmolality. Low sodium levels also cause the release of aldosterone which promotes sodium and water reabsorption in the renal tubules.

Section 5.1. Hyponatraemia

Hyponatraemia is an electrolyte imbalance involving a deficiency of sodium in relation to water. Because this deficiency lowers the osmolality of the extracellular fluid, there is a shift of water from this space into the cells, causing them to swell. While it affects body cells generally, distension of cells in the brain has the most serious consequences, including seizures, coma and permanent neurological damage.

Hyponatraemia results from the failure of the regulatory processes that maintain the balance between water and sodium. When this occurs and there is more water and less sodium in the intravascular compartment, fluid is moved from this space into the cells, where sodium concentrations are higher, by osmosis. This fluid shift not only causes distension of the cells, but may also bring about hypovolaemia or diminished blood volume.

Hyponatraemia can occur as the result of sodium loss, excessive fluid volumes or inadequate intake of sodium. It therefore takes a number of forms.

Hyponatremia – Causes three (3) types

Once hyponatraemia is clinically found. Clinical evaluation of ECF volume is required to determine the likely cause and therefore, treatment.

Table: Common causes of hyponatraemia (eTG)

Mechanism of hyponatraemia related to volume status	**Cause of low sodium concentration**
Hypervolaemia	Heart failure
	Liver cirrhosis
	Kidney failure
	Nephrotic syndrome
	Nephritic syndrome
Euvolaemia	SIADH; • Drug induced (e.g. carbamazepine, SSRIs, SNRIs) • Cerebral or pulmonary pathology • Malignancy-associated
	Hypothyroidism
	Psychogenic polydipsia
	Pain
	Nausea
	Secondary adrenal insufficiency
Hypovolaemia	Sodium loss with free water intake; • Vomiting and/or diarrhoea • Burns
	Thiazides & related diuretic drugs
	Other sodium-wasting states
	Hypopituitarism
	Primary adrenal insufficiency

Other mechanisms of hyponatraemia	**Cause of low sodium concentration**
Pseudohyponatraemia	Severe hypertriglyceridaemia
Osmotic dilution	Hyperglycaemia
	Mannitol administration
Mechanism uncertain	HIV

Hyponatremia – Signs & Symptoms

Signs and symptoms of hyponatraemia vary from patient to patient and are also influenced by the rate at which sodium levels fall. They are primarily neurologic and may include irritability, headache, twitching, tremors and muscle weakness, progressing to ataxia, stupor, delirium and seizures brought on by cerebral oedema as sodium concentrations decrease.

Patients with both hyponatraemia and hypovolaemia may exhibit poor skin turgour, dry mucous membranes, a weak and rapid pulse, low blood pressure or orthostatic hypotension. These are in contrast with the signs and symptoms of hyponatraemia with hypervolaemia which include a bounding pulse, hypertension, oedema and weight gain.

A high mortality is associated with acute severe hyponatraemia as a result of acute cerebral oedema resulting from the rapid ingestion of a large water volume either intentionally or unintentionally. Significant hyperglycaemia also causes hyponatraemia, due to shift of water out of the cells.

Mild hyponatraemia signs & symptoms:
When the serum sodium concentration is < than 135 mmol/L hyponatraemia is considered mild and the person is generally asymptomatic.

Moderate – severe hyponatraemia signs & symptoms:
The severity of the hyponatraemia itself increases the symptoms; including, mental confusion, gait disturbance, impaired consciousness and seizures. Acute hyponatraemia is not as well tolerated as chronic hyponatraemia.

Hyponatremia – Treatment

Treatment varies according to the cause and severity of hyponatraemia.

The severity of the clinical symptoms dictates the likely therapeutic approach (21);

- Any alteration of the conscious state
- The likely cause
- The rate of development

In mild cases with accompanying hypervolaemia or isovolaemia, fluid intake is restricted and oral sodium supplements may be prescribed. Where there is hypovolaemia, isotonic IV solutions may be ordered, along with a high sodium diet. More severe cases with neurological symptoms may be treated with hypertonic IV fluids. These, however, must be administered slowly, in small volumes and possibly with an appropriate diuretic in order to prevent potentially fatal fluid overload.

Section 5.2. Hypernatraemia

Hypernatraemia occurs when there is excessive sodium in relation to body water. Like hyponatraemia it produces primarily neurological symptoms, but is less common. It also has a much higher mortality rate. Hypernatraemia may result from a water deficit which brings about higher than normal sodium concentration, or excessive sodium intake. In both cases body fluid becomes hypertonic.

Hypernatremia – Causes

Water deficit - Water loss may occur in several ways including insensible water loss due to fever, heat stroke, hyperventilation associated with pulmonary disease, extensive burns, protracted vomiting or diarrhoea and abnormal diuresis. Those at greatest risk are debilitated patients, the elderly, infants and young children. In addition, people with diabetes insipidus are likely to experience water loss through copious urination unless intake matches loss.

Excessive sodium intake - Abnormally high sodium concentrations may result from high sodium foods, use of sodium supplements and some medications. Less common causes include excessive amounts of sodium in parenteral or enteral solutions, near drowning in salt water and excessive secretion of adrenocortical hormones (e.g. in Cushing's syndrome) which interferes with sodium concentrations.

Table: Common causes of hypernatraemia (eTG)

Mechanism of high serum sodium concentration	**Cause of high serum sodium concentration**
Water loss; Inadequate replacement of water	Inadequate water intake during enteral nutrition
	Diabetes insipidus; • Lack of arginine vasopressin (AVP, also known as antidiuretic hormone) *central* • Resistance to arginine vasopressin (AVP) *nephrogenic*
	Thiazide and related diuretic drugs (like indapamide, hydrochlorothiazide, chlorthalidone)
	GIT water loss
	Severe burns
Sodium overload	Hypertonic sodium chloride solution
	Oral sodium load (infants)
	Enemas

Hypernatremia – Signs & Symptoms

Neurological signs & symptoms:

Signs and symptoms of hypernatraemia are mainly neurological because of the effects of fluid shift on brain cells, and are more pronounced with rapid increases in sodium levels. Early indicators are excessive thirst, restlessness, agitation, loss of appetite, nausea and vomiting, flushing or mild fever. Unless the imbalance is corrected, lethargy, weakness, confusion, tremors, ataxia, stupor, seizures and coma may follow.

Cardiovascular signs & symptoms:

Where *excessive sodium concentrations* cause extravascular fluid to shift into the blood vessels, symptoms of hypervolaemia, such as hypertension, bounding pulse and dyspnoea, may be evident.

On the other hand, *water deficit* is likely to produce dry mucous membranes, reduced urine output and lower blood pressure.

Hypernatremia – Treatment

Treatment varies according to the underlying cause. If the cause is *water deficit*, the primary treatment (in addition to finding and rectifying the causative factor) may consist of *gradual oral fluid replacement* unless the patient is unable to drink, in which case salt-free IV fluids may be required. Other interventions include sodium restriction and diuretics in combination with *fluid replacement* to promote sodium excretion. Where diabetes insipidus is involved, treatment involves correcting the cause and the administration of vasopressin, *hypotonic IV fluids* and possibly *thiazide diuretics*.

Summary Learning Points:

The importance of sodium balance was pointed out and the specific electrolyte imbalances of both hyponatraemia (low sodium) and hypernatraemia (high sodium) were elaborated on. The various causes, signs and symptoms and treatments of each condition were also elaborated on.

QUESTIONS & ANSWERS

Module 5: Specific - Sodium Imbalances

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. The main signs and symptoms of hyponatraemia on the body affect the:

a) CVS system
b) Neurological system
c) Respiratory system
d) Renal system

2. More severe cases of hyponatraemia with neurological symptoms are commonly treated with:

a) Isotonic IV fluids
b) Hypotonic IV fluids
c) Hypertonic IV fluids
d) Oral fluid restriction

3. Treatment of hypernatraemia needs to be tailored to the specific cause and may include:

a) Cause = Water deficit (able to drink):
Treatment = Oral rehydration

b) Cause = Total parenteral nutrition (TPN):
Treatment = Continue current regime

c) Cause = Water deficit (unable to drink):
Treatment = 3% saline IV fluids

d) Cause = Diabetes insipidus:
Treatment = Hypertonic IV fluids

4. The normal serum range of sodium is:

a) 135 – 148 mmol/L
b) 152 – 154 mmol/L
c) 130 – 145 mmol/L
d) 125 – 138 mmol/L

Module 6: Specific - Potassium Imbalances

Background

Potassium is primarily an intracellular fluid cation as only about 2% of the body's supply occurs in the extracellular compartment. The normal range for the potassium concentration in serum is **3.8 – 4.9 mmol/L** and in plasma is 3.4 – 4.5 mmol/L. Levels in the cells are much higher at around 140 mmol/L.

Potassium is continually lost from the body so sufficient amounts must be ingested to replace it. About 80% of the potassium lost is excreted in the urine, while the remainder leaves the body in sweat and faeces. Extracellular potassium is also lost through fluid shift into the cells and through cellular anabolism.

Factors affecting ECF potassium levels

ECF Potassium levels & Sodium-potassium pump:
Potassium is removed from the extracellular fluid by the sodium-potassium pump, an *active transport mechanism* that moves sodium from the intracellular space to the extracellular compartment and potassium from the extracellular compartment into the cells.

ECF Potassium levels & Renal excretion:
Potassium is also lost via the kidneys. When concentrations rise, the rate of excretion by the kidney tubules increases and more potassium is removed in the urine. However, this electrolyte may also be excreted in the absence of rising serum concentration. Secretion of aldosterone causes the kidneys to reabsorb potassium and excrete sodium, a situation that persists even with low serum potassium levels and no intake.

ECF Potassium levels & pH changes:
Serum potassium concentration may also be affected by changes in pH because of the capacity of hydrogen ions and potassium ions to change places across plasma cell membranes. In acidosis, where there are excessive hydrogen ions in the extracellular space ions, they move into the cells and cause potassium ions move and causing hyperkalaemia. On the other hand, the low levels of intracellular hydrogen ions that occur on alkalosis can cause potassium ions to move into the cells to maintain balance, resulting in hypokalaemia.

Because potassium balance requires adequate intake to compensate for loss, any disease, injury or mediation that reduces intake, inhibits absorption or causes excessive output may cause hypokalaemia or potassium deficiency. Conversely, excessive intake or diminished output may result in hyperkalaemia or elevated serum levels. In addition, factors that cause fluid shift from the intracellular to the extracellular space can produce abnormally high concentrations in the extracellular fluid.

Section 6.1 Hypokalaemia

With normal acid-base status, a decrease of 1 mmol/L in serum concentration represents a *whole-body potassium deficit* of at least 200 mmol. The deficit is greater if there is associated metabolic acidosis and less if there is metabolic alkalosis. Only about 2% of total body potassium is extracellular.

Hypokalemia – Causes

Definition of the cause of hypokalaemia is assisted by knowing if hypertension is a key contributor. *Primary hyperaldosteronism* is suggested by the presence of hypertension and hypokalaemia.

Table: Common causes of hypokalaemia (eTG)

Mechanism of low serum potassium concentration	**Cause of low serum potassium concentration**
Increased urine losses	Diuretic drugs
	Hypomagnesaemia
	Amphotericin B
	Bartter & Gitelman syndromes
Excessive GIT losses	Vomiting and diarrhoea
	Laxative abuse
Decreased potassium intake	Poor nutrition
Hyperaldosteronism	Primary hyperaldosteronism
	Secondary hyperaldosteronism e.g. due to renal artery stenosis, accelerated hypertension
	Apparent mineralocorticoid excess e.g. genetic, excess licorice intake
	Some types of congenital adrenal hyperplasia
Potassium shift to cells	Metabolic alkalosis
	Insulin overdose
	IV insulin for severe metabolic derangement e.g. diabetes ketoacidosis
Other	Hypokalaemic periodic paralysis
	Beta-adrenergic drugs
	Phaeochromocytoma
	Anabolic state
	Liddle syndrome
	Severe ectopic adrenocorticotrophic hormone syndrome

Hypokalaemia – Signs & Symptoms

A variety of signs and symptoms, primarily affecting the nervous, muscular, gastrointestinal and cardiovascular systems, may indicate the presence of hypokalaemia.

Muscular signs & symptoms:
Muscle weakness, particularly in the legs, may indicate mild hypokalaemia.

Neurovascular signs & symptoms:
As levels drop further, paraesthesia, leg cramps and loss of deep tendon reflexes may develop. In severe cases, rhabdomyolysis or break down of muscle fibres may occur. Paralysis may also occur rarely.

GIT signs & symptoms:
GIT symptoms may include anorexia, nausea and vomiting because of the effects of hypokalaemia on smooth muscle. Decreased motility, constipation and paralytic ileus may also occur.

Renal signs & symptoms:
Renal consequences can result from prolonged potassium deficiency, with large volumes of urine being passed due to the inability of the kidneys to concentrate urine.

Cardiovascular signs & symptoms:
Cardiovascular symptoms include weak and irregular pulse, orthostatic hypotension and palpitations. Ventricular arrhythmias, ectopic beats, bradycardia or tachycardia and even cardiac arrest may occur with moderate to severe hypokalaemia.

Hypokalaemia – Diagnosis:

Diagnostic test results which show serum potassium levels of **less than 3.5 mmol/L** elevated 24-hour urine concentration levels, raised pH and bicarbonate levels, slightly elevated serum glucose, decreased serum magnesium and typical ECG findings; flattened or inverted T wave, depressed ST and a characteristic U wave.

Hypokalaemia – Treatment

Treatment is aimed at restoring potassium balance, treating the underlying cause and preventing complications. Urgent treatment for apparent hypokalaemia needs to be confirmed on a further serum sample. The optimal therapy choice includes an assessment of kidney function and acid-base status. The mechanism of ongoing potassium loss needs to be clearly identified for the therapy to be successful. Continuing potassium loss needs to be considered if hypokalaemia does not respond.

Mild hypokalaemia:
A high potassium and low sodium diet may be effective in mild to moderate acute cases, but where the condition is more prolonged, supplementary potassium in the form of potassium chloride may also be required. In most patients, oral potassium supplements are adequate but they can be difficult to swallow. In these patients an effervescent formulation could be used as an alternative.

Severe Hypokalaemia:
Severe hypokalaemia may require IV administration of potassium, as will the hypokalaemic patient who is unable to take it orally. However, care must be taken with this route to avoid serious complications.
IV potassium is required:

- When oral intake is not possible
- For severe hypokalaemia **(< 3 mmol/L)** with associated muscle paralysis
- For cardiac rhythm disturbance with ECG evidence of hypokalaemia

Rapid IV infusion of potassium may cause fatal hyperkalaemia regardless of the severity of the overall deficit.

One special consideration, however, would be the emergency management of diabetic ketoacidosis;

- Rate of potassium infusion should not exceed 10 mmol/hour
- Potassium concentration through a peripheral vein should not exceed 40 mmol/L

Premixed infusion bags should be used with potassium chloride treatment. The choice of preparation depends on the amount of potassium required, whether IV access is central or peripheral and fluid balance requirements of the individual patient. During IV potassium replacement the patient should be on continuous cardiac monitoring and the serum potassium concentration measured every 2 hours. In the case of hypokalaemia caused by an acute potassium shift into cells (e.g. periodic paralysis or insulin excess) there is a danger of rebound hyperkalaemia. While hypokalaemia due to fixed mineralocorticoid excess (e.g. in primary hyperaldosteronism) restricting sodium intake is also required in achieving potassium repletion. Chloride deficits need to be corrected when potassium depletion is associated with severe metabolic alkalosis (e.g. from vomiting acid stomach contents).

After balance has been restored, sustained-release supplements may be prescribed. Where diuretics are used, they may be changed to formulations that spare potassium to prevent excessive loss from the kidneys, however, potassium-sparing medications have no place in managing acute hypokalaemia.

Section 6.2. Hyperkalaemia

Hyperkalaemia is a very serious electrolyte disorder that occurs when serum potassium levels **exceed 5 mmol/L**. It may be due to any one of three causative mechanisms:

1. Excessive intake of potassium
2. Reduced excretion of potassium from the body; or
3. Changes in the balance between extracellular and intracellular potassium concentrations.

Hyperkalaemia – Causes

Table: Common causes of hyperkalaemia (eTG)

Mechanism of high serum potassium concentration	Cause of high serum potassium concentration
Pseudohyperkalaemia	Haemolysis
	Blood sample handling (stored, refrigerated, recentrifuged)
Kidney failure	Thrombocytosis
	Extreme leucocytosis
	Reduced potassium excretion
Fluid volume depletion	Reduced potassium excretion secondary to reduced distal tubular water and sodium delivery
Hypoaldosteronism	Hyporeninaemia (e.g. diabetes, interstitial kidney disease)
	Primary adrenal insufficiency
	Adrenal enzyme insufficiency
	HIV
	Resistance to aldosterone action (pseudohypoaldosteronism)
Drug induced	Heparin
	Nonsteroidal anti-inflammatory drugs
	Potassium-sparing diuretic drugs
	Potassium supplements
	Trimethoprim
	Angiotensin converting enzyme inhibitors
	Angiotensin II receptor blockers
	Pentamidine
	Cyclosporin
Increased potassium release from cells	Metabolic acidosis
	Insulin deficiency
	Tissue damage
	Rhabdomyolysis
Other	Hyperkalaemic periodic paralysis
	Gordon syndrome (with elevated blood pressure)

Hyperkalaemia – Signs & Symptoms

Hyperkalaemia primarily affects neuromuscular and cardiovascular function.

Neuromuscular signs & symptoms:
Early signs include paraesthesia and irritability. Skeletal muscle is affected, initially with weakness, progressing to flaccid paralysis. Weakness may also spread to the trunk and impair respiration. Smooth muscle function may be affected, especially in the gastrointestinal system, causing diarrhoea, nausea and cramping.

Cardiovascular signs & symptoms:
Early cardiovascular signs are tachycardia and irregular pulse, progressing to diminished cardiac output, hypotension and possibly cardiac arrest. Potentially lethal arrhythmias may occur when serum potassium levels exceed 7 mmol/L.

Hyperkalaemia – Diagnosis

Diagnosis of hyperkalaemia is confirmed by serum potassium levels of more than 5 mmol/L, reduced arterial pH and typical ECG changes - tall T wave, possibly in conjunction with flattened P wave, prolonged PR interval, wider than normal QRS complex and depressed ST segment.

Hyperkalemia – Treatment

Treatment varies with severity, but is directed at reducing potassium levels by increasing renal and gastrointestinal excretion, reversing the underlying cause and restoring normal cardiovascular function.

Mild hyperkalaemia:
In mild hyperkalaemia it consists of reducing the dietary intake of potassium and possibly using diuretics to boost potassium excretion and resolve any related acidosis. Medications may also be reviewed and any that contribute to the condition are ceased or adjusted. Obviously, any contributory disorders are also identified and treated.

Moderate hyperkalaemia:
Management of moderate to severe hyperkalaemia may require other treatment modalities. One of these is high *sodium cation exchange* medications which are retained in the bowel and cause sodium to pass through the intestinal wall and into the circulation, like resonium medication. This in turn causes potassium to move from the blood stream into the gastrointestinal tract, from which it is excreted in the faeces.

(Acute) Severe hyperkalaemia:
Emergency treatment for life-threatening hyperkalaemia of any cause (except acute digoxin toxicity or chronic digoxin accumulation) is to:

- Give IV calcium gluconate which has an immediate cardiac stabilising effect
- Correct any fluid volume depletion

In kidney failure the use of IV glucose and insulin are effective. IV bicarbonate is effective if metabolic acidosis is present but it may not lower potassium immediately. IV hydrocortisone should be used in the emergency treatment of primary adrenal insufficiency, insulin should not be given in this situation. Sodium or calcium ion exchange resin can be used as less urgent treatment especially for patients in kidney failure.

IV Calcium gluconate:
Calcium reduces the membrane depolarisation of severe hyperkalaemia without lowering the serum potassium concentration. In acute hyperkalaemia with the presence of life-threatening cardiac arrythmia or severe ECG changes;

- Calcium gluconate 10% 10 ml (1 g or 2.2 mmol) IV over 2 – 3 minutes into a large vein. Monitor response by ECG where possible.

This infusion has short-lived effects so the dose may need to be repeated in 30 – 60 minutes, whilst undertaking specific measures to reduce potassium.

Correcting volume depletion:
Kidney function needs to be optimised by correcting any volume depletion.

IV Sodium bicarbonate:
The first choice for treating severe confirmed hyperkalaemia when metabolic acidosis is associate with volume depletion is:

- Sodium bicarbonate 8.4% (= 1 mmol/mL) 50 ml IV over 5 – 10 minutes, under ECG control
- This may be repeated in 60 – 120 minutes

Fluid replacement with sodium chloride 0.9% may need to be continued.

Insulin:
Cellular uptake of potassium is promoted by insulin. Serum potassium decreases by 0.5 – 1.5 mmol/L over 30 minutes in response to the insulin-glucose therapy. The treatment of choice for severe hyperkalaemia associated with chronic kidney failure is insulin and glucose, when a sodium load in contraindicated. Before insulin is administered hypoglycaemia needs to be ruled out as profound refractory hypoglycaemia can be induced by giving insulin in the presence of adrenal insufficiency.

Before commencing insulin-glucose therapy, the blood glucose level needs to be measured;

- Pre-treatment
- Then every 30 minutes for 2 hours
- Then hourly for the next 4 hours
- In total, measured for 6 hours after the insulin-glucose therapy commenced

Glucose 50% may cause vascular irritation when given peripherally, glucose 10% is an alternative, if available. Use:

- Short-acting insulin 10 units IV bolus, refer to eTG guidelines for insulin formulations,
- **PLUS,** either Glucose 50% in 50 mL over 5 minutes **OR**
- Glucose 10% 250 mL IV over 15 minutes

There is a risk of hypoglycaemia, often severe, after insulin-glucose therapy so the treatment is to give glucose as for severe hypoglycaemia refer to eTG guidelines.

Removing potassium from the bowel lumen:
Polystyrene sulfonate resins can be used as a way to facilitate potassium removal from the bowel lumen in exchange for sodium or calcium. This treatment can take several hours to take effect. Each gram of resin removes about 1 mmol of potassium and delivers 2 – 3 mmol of sodium. This treatment lowers the serum potassium concentration by 0.5 – 1 mmol/L over 1 – 6 hours. Sodium loading can be a disadvantage of this type of treatment. A calcium exchange resin can be used as an alternative if adding to the sodium load is a problem. To avoid hypokalaemia due to overtreatment, ion exchange resin treatment should stop when the serum potassium is *< 5 mmol/L*.

Dialysis:
In extreme hyperkalaemia, like in extensive tissue breakdown due to rhabdomyolysis, none of the above approaches may work so dialysis is necessary.

Medication review:
Drug-related hyperkalaemia is more common when kidney function is impaired so in this instance, drugs that aggravate hyperkalaemia should be stopped, e.g. ACEI (e.g. perindopril) or ARBs (e.g. telmisartan).

Summary Learning Points:

The importance of potassium as mainly an ICF cation was described. The three (3) important factors affecting ECF potassium levels are the; sodium-potassium pump, renal excretion and pH changes were discussed. Additionally, the causes, signs and symptoms, and treatment of both hypokalaemia and hyperkalaemia were outlined.

QUESTIONS & ANSWERS

Module 6: Specific - Potassium Imbalances

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. Classic ECG changes associated with hyperkalaemia seen with the QRS complex include a:

a) narrow QRS complex
b) normal QRS complex,
c) widened QRS complex
d) QRS complex which is upright

2. The group of medications that may cause hyperkalaemia by inhibiting the movement of potassium from the extracellular to the intracellular fluid is:

a) Beta blockers
b) ACE inhibitors
c) NSAID's
d) Insulins

3. The bulk of extracellular fluid potassium loss occurs as a result of:

a) Renal excretion
b) Gastrointestinal excretion
c) Excretion by the skin and lungs
d) Fluid shift into the cells

4. Which of the following medications may be ordered to counteract the effects of hyperkalaemia on the cardiovascular system?

a) Calcium gluconate
b) Insulin
c) Sodium bicarbonate
d) Beta blockers

5. A common gastrointestinal symptom of hypokalaemia is:

a) Normal appetite
b) Increased bowel motility
c) Diarrhoea
d) Paralytic ileus

Module 7: Specific - Magnesium Imbalances

Background

Magnesium is the second most abundant cation in the body and is involved in a number of essential functions including carbohydrate metabolism, production of ATP for cellular energy, DNA synthesis, vasodilatation, cardiac muscle contractility, parathyroid hormone production and the movement of sodium and potassium ions between the intracellular and extracellular spaces. It is a key factor in neuromuscular activity because of its effect on irritability and contractility of both cardiac and skeletal muscle. Magnesium also plays a role in calcium regulation because of its involvement in the synthesis of parathyroid hormone which maintains constant extracellular fluid calcium levels.

The normal range of the adult serum or plasma magnesium is **0.8 to 1.0 mmol/L**, Different values apply to neonates and children. Serum concentrations, however, are not necessarily accurate indicators of the body's total magnesium because less than 1% of it is contained in the extracellular fluid. Magnesium levels are also closely linked to the concentrations of other substances, especially calcium and albumin. Because magnesium occurs primarily in the intracellular fluid, changes in the concentrations of other electrolytes within the cells can affect serum magnesium levels. Also, due to the fact that magnesium ions bond with proteins, particularly albumin, changes in albumin levels cause similar changes in serum magnesium levels.

As is the case with other electrolytes, magnesium balance is regulated by the interaction of absorption (from dietary intake), excretion (in the faeces and urine) and retention (by the renal tubules). If the serum magnesium level drops, gastrointestinal absorption increases as does reabsorption in the proximal tubules and loop of Henle in order to restore balance. Increases in serum magnesium are controlled by increased excretion in the faeces and urine. However, while these regulatory mechanisms are usually very effective, imbalances can occur.

Section 7.1. Hypomagnesaemia

Hypomagnesaemia occurs when the serum magnesium concentration is **lower than 0.8 mmol/L**. A severe magnesium deficiency is apparent with levels of less than 0.4 mmol/L and includes symptoms like hyperactive deep tendon reflexes, weakness, cramps, tachycardia, ataxia and balance problems. If the problem is not treated, symptoms may progress to impaired mental functioning, respiratory muscle paralysis, heart block and coma.

Hypomagnesaemia - Causes

It is common in hospitalised patients, especially the critically ill.

Table: Common causes of Hypomagneseamia (eTG)

Mechanism of low serum magnesium concentration	Causes of low serum magnesium concentration
Severe malnutrition	Poor oral intake
GIT losses	Nasogastric suction
	Diarrhoea
	Malabsorption
	Extensive bowel resection
	Primary intestinal hypomagnesaemia
	Intestinal fistulae
Kidney losses	Chronic parenteral fluid therapy
	Hypercalcaemia & hypercalciuria
	Osmotic diuresis
Drugs	Diuretic drugs
	Alcohol
	Aminoglycoside antibiotics (e.g. gentamycin)
	Cisplatin
	Amphotericin B
	Cyclosporin
	Foscarnet
	Pentamidine
	Proton pump inhibitors (PPIs)
Other	Phosphate depletion
	Post-obstructive nephropathy
	Diuretic phase of acute kidney injury (AKI)

Hypomagnesaemia – Signs & Symptoms

Signs and symptoms of the disorder range from mild to lethal and may involve the CNS, neuromuscular function, cardiovascular system and the GIT.

CNS signs:
Central nervous system symptoms arise from the neuronal irritation caused by reduced serum magnesium levels and may include altered conscious state, confusion, insomnia, ataxia, vertigo, mood swings, delusion, hallucinations, depression and psychosis.

Neuromuscular signs:
Tremors, twitching, Chvostek's sign, Trousseau's sign, tetany, parasthaesia, hyperactive deep tendon reflexes and, where respiratory muscles are involved, dyspnoea and laryngeal stridor. Low serum magnesium levels encourage the

movement of the electrolyte *out of the cells*, causing them to become *weaker* and *more irritable*.

Cardiac signs:
Low serum magnesium levels may result in arrhythmias due to irritability of the myocardium. These patients with severe hypomagnesaemia, therefore, require continuous cardiac monitoring. Low serum levels, either alone or in conjunction with concurrent calcium or potassium deficiencies, may produce such arrhythmias as atrial fibrillation (AF), paroxysmal atrial tachycardia (PAT), premature ventricular contractions (PVCs), ventricular fibrillation (VF), ventricular tachycardia (VT), supraventricular tachycardia (SVT), torsades de pointes and heart block. Typical ECG changes may show prolonged PR interval, widened QRS complex, prolonged QT interval, depressed ST segment, flattened T wave and prominent U wave (4).

GIT signs:
Common GIT signs are anorexia, dysphagia and nausea and vomiting which may exacerbate the problem by further reducing intake or increasing the rate at which magnesium is lost.

Hypomagnesaemia - Treatment

The mainstay of treatment is to correct the cause. Co-existing ion abnormalities including hypocalcaemia, hypokalaemia and metabolic acidosis are common. Mild hypomagnesaemia should be treated with oral replacement, like magnesium aspartate. Moderate to severe hypomagnesaemia with associated clinically consistent signs and symptoms may require IV magnesium and the rate of infusion depends both on the extent of the deficit and the clinical features present.

Section 7.2. Hypermagnesaemia

Hypermagnesaemia occurs when serum magnesium concentration **above 1.0 mmol/L** but it is relatively uncommon because of the effectiveness of the kidneys in excreting excess magnesium. It may result from excessive intake or impaired excretion.

While excessive intake of magnesium is not uncommon, it usually occurs in individuals with impaired health whose treatments involve the use of magnesium-rich medications, feeds or solutions. Examples include patients with renal failure who take antacids or laxatives with high magnesium content.

Hypermagnesaemia – Causes

Refer to table on following page

Table: Common causes of Hypermagneseamia (eTG)

Mechanism of high serum magnesium concentration	Causes of high serum magnesium concentration
Excessive intake	Antacids
	Enemas
	IV infusion
	Ureteral irrigation with hemiacidrin
Decreased excretion	Kidney failure
	Volume depletion
	Familial hypocalciuric hypocalcaemia
Release from cells	Rhabdomyolysis
Unknown	Lithium

Hypermagnesaemia – Signs & Symptoms

As hypermagnesaemia affects neuromuscular, respiratory and cardiovascular function and level of consciousness, signs and symptoms occur in these systems. They vary according to the severity of the magnesium excess.

Serum magnesium levels	Signs & Symptoms
1.5 mmol/L approx.	May produce flushing, feelings of warmth, mild hypotension, nausea and vomiting
2 mmol/L approx.	Deep tendon reflexes are reduced and muscle weakness may also be evident
2.5 mmol/L approx.	Drowsiness, bradycardia and more pronounced hypotension ECG changes may be seen; • Prolonged PR interval • Broader QRS complex & • Elevated T wave (4)
3.5 mmol/L approx.	Absence of deep tendon reflexes
4 mmol/L approx.	Respiratory depression
7.5 mmol/L approx.	Respiratory arrest
10 mmol/L approx.	Bradycardia / Heart block leading to cardiac arrest

Hypermagnesaemia - Treatment

Treatment is based on restoring magnesium balance and correcting the underlying cause. Where there is no renal impairment, treatment may simply involve increasing the patient's fluid intake (orally or IV) in order to promote urine output and remove excess magnesium.

If this is not effective, diuretics may be used to achieve the same result. In serum magnesium concentration higher than 2.0 – 2.5 mmol/L an IV calcium infusion

provides immediate but transient antagonism (reversal) of the toxic effects. Kidney excretion should be accelerated with IV sodium chloride 0.9% aiming for a urine output of at least 60 ml/hr. IV frusemide can be added if this urine output cannot be achieved. In patients with kidney impairment, dialysis may be needed. Mechanical ventilation may also be need to manage severe respiratory symptoms.

Summary Learning Points:

Magnesium's crucial role as a cation in the body was detailed in this module. The normal range of magnesium was outlined. The causes, signs and symptoms of hypomagnesaemia and hypermagnesaemia were elaborated on.

QUESTIONS & ANSWERS

Module 7: Specific - Magnesium Imbalances

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. Your patient has a magnesium level of 2.0 – 2.5 mmol/L, which antagonist medication would be ordered to treat this elevated level?

a) IV Phosphorus
b) IV Calcium
c) IV Chloride
d) IV Potassium

2. Low serum levels of magnesium may result in cardiac arrhythmias including:

a) AF, PVCs, VF, torsades de pointes
b) PVCs, SVT, sinus arrhythmia, VF
c) VF, SVT, PAT, NSR
d) Torsades de pointes, AF, VF, sinus arrythmia

3. What is the level that hypomagnesaemia is defined?

a) Below 1.0 mmol/L
b) Below 0.5 mmol/L
c) Below 1.25 mmol/L
d) Below 0.8 mmol/L

4. A patient has been admitted to the High Dependency Unit (HDU) following an intentional oral narcotic overdose, the patient was unconscious for a number of hours. They have been diagnosed with rhabdomyolysis, what mechanism causes hypermagnesemia in these patients?

a. Excessive magnesium intake
b. Lithium's effect on magnesium levels
c. Decreased magnesium excretion
d. Magnesium release from cells

Module 8: Specific - Calcium Imbalances

Background

In addition to its involvement in the secretion of certain hormones, maintenance of cell structure and function, and composition of bones and teeth, calcium plays an important part in muscle function (cardiac, smooth and skeletal) and in blood clotting. Only a small percentage of the body's total supply of calcium occurs in the serum as about 99% of it is found in the teeth and bones. Its levels are influenced by a number of factors including dietary intake, existing reserves in the body, hormones secreted by the thyroid and parathyroid glands, vitamin D levels, phosphorus concentrations and serum pH. Normal adult serum calcium levels range from **2.10 to 2.60 mmol/L.** Values tend to be reduced in elderly persons.

When serum calcium is low, the parathyroid glands secrete parathyroid hormone which causes calcium and phosphorus to be released from the bones into the blood, increasing the plasma concentration. Parathyroid hormone also increases calcium reabsorption in the kidneys and absorption from the intestines. Vitamin D enhances absorption and reabsorption, further raising serum concentrations.

When calcium levels are too high, the secretion of parathyroid hormone is suppressed. At the same time, the thyroid gland secretes calcitonin, a calcium antagonist that interferes with the release of supplies stored in the bones, thereby helping to reduce serum levels. It also has the effect of blocking calcium absorption in the gastrointestinal tract and increasing renal excretion.

Phosphorus is also involved in calcium regulation. When calcium levels are high, phosphorus levels drop and, conversely, when calcium levels are low phosphorus concentrations increase. Phosphorus lowers serum calcium by inhibiting absorption in the gut and raises it by causing the kidneys to conserve it. Serum pH has a similar inverse relationship in regard to calcium. When serum pH increases (ie the blood becomes more alkaline) calcium levels drop because the electrolytes bind to protein particles in the blood. Conversely, if serum pH decreases, less ionised calcium combines with proteins, so serum calcium levels rise.

In the event of these regulatory mechanisms failing to balance calcium levels, hypercalcaemia or hypocalcaemia may occur.

Section 8.1. Hypocalcaemia

Hypocalcaemia is diagnosed on the basis of a low serum total calcium concentration corrected for albumin below **2.10 mmol/L**. Serum ionised calcium needs to be directly measured if the albumin is significantly abnormal.

Hypocalcaemia – Causes

Table: Common causes of hypocalcaemia (eTG)

Causes of hypocalcaemia	Clinical examples
Severe vitamin D deficiency	
Autoimmune hypoparathyroidism	
Parathyroid injury	Following thyroidectomy or parathyroidectomy
Hypoparathyroidism related to congenital disorders	DiGeorge syndrome
Hypomagnesaemia	Association with malabsorption, ETOH abuse & Proton pump inhibitors (PPIs)
Antiresorptive drugs	Bisphosphonates, e.g. pamidronate
Autosomal dominant hypocalcaemia	
Neonate of a mother with hypercalcaemia	
Vitamin D-dependent Rickets Type 1 or 2	

Hypocalcaemia – Signs & Symptoms

Mild - Signs & Symptoms:
Hypocalcaemia is often asymptomatic.

Moderate - Signs & Symptoms:
Moderate hypocalcaemia is usually caused by hypoparathyroidism and presents with muscle cramps, spasms or parasthesia. Patients may also present with generalised signs of neuromuscular irritability such as tetany, often including positive Chvostek and Trousseau signs.

Severe - Signs & Symptoms:
Severe hypocalcaemia may result in tetany, laryngospasm and seizures. Hypocalcaemia can be life threatening depending on the severity of these seizures so patients need to managed in a High Dependency Unit (HDU). If acute and severe hypocalcaemia is left untreated in children, neuronal damage and intellectual impairment may occur.

Hypocalcaemia – Treatment

Moderate:
Oral calcium supplements can be used in adults to correct hypocalcaemia. Oral calcitriol will need to be given to patients with hypoparathyroidism while oral colecalciferol is used to treat patients with Vitamin D deficiency. In children, calcium and calcitriol are usually given together.

(Acute) Severe:
Despite the cause, severe hypocalcaemia should be treated initially with a slow IV injection of calcium. In adults, a calcium infusion should also follow this initial treatment. The aim is to control symptoms by maintaining the serum calcium concentration within the normal range. Extravasation of calcium can result in severe

tissue damage through localised skin necrosis. In Australia two (2) parenteral formulas are available, however, 10% calcium gluconate is preferred over 10% calcium chloride as calcium gluconate it is less toxic to peripheral veins. Calcium must never be administered by intramuscular or subcutaneous routes. During IV therapy replacement, the patient must have continuous cardiac monitoring on and serum calcium concentration needs to be measured every 3 - 4 hours. It may also be necessary to correct any co-existing hypomagnesaemia.

Long – term:

If the cause of hypocalcaemia is permanent, as in the case of a damaged parathyroid gland, then long-term calcium therapy will be necessary.

Section 8.2. Hypercalcaemia

There are two (2) classifications of hypercalcaemia, parathyroid-dependent or parathyroid-independent. They are differentiated by simultaneously measuring serum calcium and parathyroid hormone concentrations. The parathyroid hormone hormone concentration is usually above or at the upper end of the normal range in parathyroid-dependent hypercalcaemia.

Hypercalcaemia – Causes

Hypercalcaemia has a wide variety of causes, however, most (80 – 90%) of adult cases result from primary hyperparathyroidism or malignancy.

Table: Causes of Adult Hypercalcaemia (eTG).

Hypercalcaemia: More common causes
Primary hyperparathyroidism
Hypercalcaemia of malignancy
Hypercalcaemia: Less common causes
Thiazide diuretic drugs
Vitamin D toxicity
Sarcoidosis or other granulomatous disorders
Severe hyperthyroidism
Milk alkali syndrome
Renal osteodystrophy with tertiary hyperparathyroidism
Primary adrenal insufficiency
Familial hypocalciuric hypercalcaemia
Familial benign hypercalcaemia
Prolonged immobilisation
Cytochrome P450 24A1 gene mutation
William syndrome

Hypercalcaemia – Signs & Symptoms

Incidental detection of hypercalcaemia may occur or it may be present with various non-specific symptoms including; failure to thrive (infant), abdominal pain, constipation, polyuria and polydipsia. Abnormal cardiac rhythms may also be detected especially in older people and patients with a history of cardiac disease. Cardiac function needs to be monitored with continuous cardiac monitoring until the serum calcium level is reduced to a safer concentration. This concentration would be expected to be within or slightly above the normal calcium range.

Hypercalcaemia – Treatment

(Acute) Severe:
This is where the serum total calcium concentration corrected for albumin is ***above 3.0 mmol/L***. At this level of hypercalcaemia, the result is dehydration and electrolyte depletion, which then leads to a deterioration in kidney function. This deterioration leads to further serum calcium increases. The primary treatment of acute severe hypercalcaemia is to administer a sodium chloride infusion. This will reduce the serum calcium concentration but it will not actually normalise it. An adult would normally be ordered sodium chloride 0.9% 4 – 6 litres by IV infusion over 24 hours. The level of rehydration in a child, however, is based on the degree of dehydration and the child's weight.

Following rehydration, IV frusemide can be used in adults with Specialist supervision to counteract fluid overload, following this rehydration process. Frusemide is not used initially as it can deplete the patient's intravascular volume further.

In adults who do not respond well to rehydration, an IV bisphosphonate infusion, like pamidronate acid or pamidronate disodium may be used to temporarily lower the serum calcium concentration. These patients must, however, be well hydrated prior to IV bisphosphonate's use. Parenteral salcatonin should also be considered for use in acute life-threatening hypercalcaemia cases. This therapy is combined with the bisphosphonate to achieve a more rapid reduction in the serum calcium level.

If the cause of hypercalcaemia is related to Vitamin D toxicity, calcium supplements need to be ceased immediately, this calcium electrolyte imbalance will then normally settle over 1 – 2 days.

Summary Learning Points:

The importance of calcium in the body was outlined. Both hypocalcaemia and hypercalcaemia were defined and the more and less common causes of the conditions were tabled. The signs and symptoms and treatment of both these conditions were elaborated on.

QUESTIONS & ANSWERS

Module 8: Specific - Calcium Imbalances

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. The initial treatment of acute severe hypercalcaemia includes:

a) Vitamin D tablets
b) IV Normal saline
c) IV Frusemide
d) TPN solution

2. Damage to which of the following structures is associated with a reduction in serum calcium?

a) Parathyroid glands
b) Pancreas
c) Kidneys
d) Lungs

3. If serum calcium levels are low:

a) Serum phosphate concentrations drop
b) Serum phosphate concentrations rise
c) Serum phosphate concentrations remain unchanged
d) Serum pH increases

4. Hypocalcaemia is diagnosed with a total serum calcium concentration, when controlled for albumin, of:

a) < 2.3 mmol/L
b) < 2.10 mmol/L
c) < 1.8 mmol/L
d) < 1.5 mmol/L

5. Low serum levels of which electrolyte may result in tetany:

a) Hypocalcaemia
b) Hypophosphataemia
c) Hypomagnesaemia
d) Hypokalaemia

Module 9: Specific - Phosphorus Imbalances

Background

Phosphorus, a significant anion, occurs in the body as phosphate. Like calcium most of it is found in the bones, teeth and soft tissues, with only about 1% in the extracellular fluid. It is involved in cell membrane integrity, neurological function, muscle function, metabolism, oxygen delivery, buffering of acids and bases, provision of cellular energy, phagocytosis, platelet function and bone formation (4).

Normal serum phosphate levels in adults range from **0.8 – 1.5 mmol/L** but this does not necessarily indicate the total amount of body phosphorus because the amount contained in the cells is much higher. Serum phosphate levels are influenced by dietary intake, hormonal regulation, excretion in the urine and movement in and out of the cells. Phosphorus is readily absorbed from the gastrointestinal tract, particularly the jejunum, and the amount absorbed is reflective of the amount ingested. About 90% is excreted by the kidneys with the rest excreted in the faeces. However, excretion rates vary to accommodate changes in intake so that balance can be maintained. If intake increases, excretion by the kidneys increases in response; if it decreases the kidneys respond by reabsorbing more phosphorus.

Parathyroid hormone represents the major form of hormonal regulation of serum phosphorus, although it acts on calcium rather than directly on phosphorus. Because of the inverse relationship between calcium and phosphorus, low calcium levels are accompanied by high phosphate levels. Low calcium levels initiate the release of parathyroid hormone which increases the absorption of both electrolytes from the gut and their reabsorption from the bones. This results in increased levels of both calcium and phosphorus. However, the parathyroid hormone then causes the kidneys to excrete more phosphorus, restoring the balance. High calcium levels, on the other hand, are associated not only with low phosphate levels, but also with reduced parathyroid hormone release. Low parathyroid hormone levels promote phosphorus reabsorption in the kidneys, causing its concentration to rise and again restoring the balance.

Should these regulatory mechanisms fail, serum levels may become too low or too high, causing hypophosphataemia and hyperphosphataemia respectively.

Section 9.1. Hypophosphataemia

Hypophosphataemia - Causes

Hypophosphataemia is defined as a serum phosphate concentration **lower than 0.8 mmol/L**. Hypophosphataemia is usually due to:

❒ Movement of phosphate out of the ECF and into the cells that occurs with respiratory alkalosis, hyperglycaemia, malnutrition and hypothermia.
❒ Diminished absorption of phosphate in the intestines associated with malabsorption syndromes, vitamin D deficiency, chronic diarrhoea, starvation and over-use of laxatives or phosphorus-binding antacids.

Hypophosphataemia – Signs & Symptoms

Moderate hypophosphataemia
Serum phosphate concentration **0.5 – 0.8 mmol/L** is often seen with primary hyperparathyroidism, tumour-induced osteomalacia, vitamin D deficiency, iron transfusions and antacid abuse. It usually resolves when the cause is treated or stopped so oral replacement is rarely required.

Severe hypophosphataemia
Serum phosphate concentration **lower than 0.5 mmol/L** can be caused by alcohol withdrawal syndrome, refeeding syndrome (anorexia nervosa, starvation), trauma and sepsis, and occurs in around 1% of hospitalised patients. It is often asymptomatic, but presenting symptoms can be:

❒ Neuromuscular – ranging from progressive myopathy to paralysis, confusion and seizures
❒ Cardiorespiratory – respiratory muscular failure, left ventricular dysfunction with heart failure and arrhythmias
❒ Haematological – hemolysis, thrombocytopenia and impaired leucocyte function

Hypophosphataemia – Treatment

Treatment is not always indicated, it depends on the severity and the rate of decline of serum phosphate concentration. Management of acute hypophosphatemia in critically ill patients is guided by the severity of the deficiency. For patients with severe hypophosphataemia and normal kidney function, use:

❒ Phosphate dihydrogen phosphate 13.6% 2 to 10 mmol elemental phosphate/hour by IV infusion, for 4 hours

Measure serum calcium and phosphate concentrations hourly and adjust the dose as necessary. Monitor cardiac and renal function during the infusion. Phosphate replacement is usually required until the cause has been treated.

Chronic hypophosphataemia can occur in patients with conditions such as familial X-linked hypophosphataemic rickets and tumour-induced osteomalacia. Treatment is a combination of oral elemental phosphorus and calcitriol, calcitriol is usually only used in children and adolescents.

Section 9.2. Hyperphosphataemia

Serum phosphate levels of **greater than 1.5 mmol/L** indicate hyperphosphataemia, a condition usually associated with cell damage and the resultant transfer of phosphorus from the intracellular to the extracellular fluid. Delayed transport of samples to the laboratory can result in a spuriously elevated serum phosphate concentration.

Hyperphosphataemia – Causes

Hyperphosphataemia is typically caused by:

❒ Impaired kidney phosphate excretion (e.g. kidney disease, tumoral calcinosis, hypoparathyroidism)
❒ Increased extracellular phosphate (e.g. rapid administration of phosphate, catabolic states, tissue or cell lysis)

Chronic kidney disease is the most common cause; hyperphosphataemia caused by kidney disease is usually chronic and asymptomatic. Clinical manifestations are usually related to associated hypocalcaemia and tetany. Chronic hyperphosphataemia is primarily treated by managing the cause. It can also include restricting phosphate intake, using phosphate binders and inducing diuresis (not recommended in patients with impaired kidney function) as Specialist management is required.

Acute severe hyperphosphataemia (with electrolyte disturbance and sudden death) has been reported after taking pre-procedural oral sodium phosphate laxatives (e.g. before a colonoscopy). The rapid onset of hyperphosphataemia (in contrast to the chronic hyperphosphataemia seen with chronic kidney disease) causes cardiorespiratory collapse and seizures. Emergency treatment with volume expansion and urgent haemodialysis may be required.

Hyperphosphataemia – Signs & Symptoms

They include paraesthesia of the fingers, mouth and face, muscle weakness, spasms, cramps, hyperreflexia, positive Trousseau's and Chvostek's signs, hypotension, characteristic ECG changes, decreased mental functioning, confusion, seizures, poor appetite and nausea and vomiting.

Calcification may also result from chronic hyperphosphataemia. This arises when phosphorus combines with calcium to form calcium phosphate. When this precipitates as a salt and is deposited in the tissues, calcification occurs. It interferes with the functions of all tissues affected, but its consequences are most significant when organs such as the heart, lungs and kidneys are involved. Arrhythmias may be an early sign of cardiac calcification; dyspnoea may suggest respiratory involvement and oliguria may indicate renal calcification.

NB: Because of the inverse relationship between phosphorus and calcium in the body (i.e. when one is high the other is low), the clinical signs of hyperphosphataemia are very similar to those of hypocalcaemia.

Hyperphosphataemia – Diagnosis

Results indicative of hyperphosphataemia include an elevated serum phosphate, low serum calcium, elevated blood urea nitrogen and creatinine, ECG changes typical of hypocalcaemia and in chronic cases, radiological evidence of defective bone development.

Hyperphosphataemia – Treatment

As with other electrolyte imbalances, treatment is aimed at rectifying any underlying disorder and correcting the imbalance. **Mild hyperphosphataemia** due to excessive intake may be corrected by reducing dietary phosphorus and avoiding the use of phosphorus-rich laxatives and enemas. In more severe cases, pharmacological agents may be used to decrease absorption in the gut.

Patients with **severe hyperphosphataemia** may require IV administration of saline to increase the excretion of phosphorus in the urine, provided that there is effective renal function.

Summary Learning Points:

As a significant anion found in the bones, teeth and soft tissues, phosphorus which occurs in the body as phosphate was introduced. Hypophosphataemia and hyperphosphataemia were discussed, including the relevant causes, signs and symptoms, diagnosis and treatment of these two (2) conditions.

QUESTIONS & ANSWERS

Module 9: Specific - Phosphorus Imbalances

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. The main substance involved in the regulation of phosphate levels is:

a) Antidiuretic hormone
b) Insulin
c) Aldosterone
d) Parathyroid hormone

2. Hypophosphataemia occurs when serum phosphate levels are lower than:

a) 0.80 mmol/L
b) 1.0 mmol/L
c) 4.0 mmol/L
d) 2.02 mmol/L

3. The normal adult levels of phosphorus range from …

a) 0.8 – 1.5 mmol/L
b) 0.46 – 0.76 mmol/L
c) 1.0 – 1.5 mmol/L
d) 0.7 – 0.9 mmol/L

4. The treatment of severe hyperphosphataemia includes;

a) Increasing dietary phosphate
b) Using a Fleet enema
c) Administration of IV Normal saline 0.9%
d) Administration of IV 3% saline

Module 10: Specific - Chloride Imbalances

Background

Chloride is the major anion occurring in the extracellular fluid, making up about two thirds of all serum anions. It is involved in a number of vital functions, including:

- Provision of an acid medium for digestion by combining with hydrogen in the stomach to form hydrochloric acid
- Activation of salivary amylase
- Maintenance of serum osmolality and tonicity
- Maintenance of acid-base balance
- Maintenance of water balance
- Exchange of oxygen and carbon dioxide in the red blood cells.

Chloride moves between the intracellular and extracellular fluid in close association with sodium so that when serum sodium levels change, chloride concentrations change in proportion. Both are, to a large extent, regulated by aldosterone, although its influence on chloride is not as direct as it is on sodium. Aldosterone controls sodium reabsorption in the kidneys, and because chloride passively follows sodium across the semipermeable membrane, its levels increase or decrease according to whether sodium is being reabsorbed or excreted.

Normal serum chloride levels in adults range from **95 to 110 mmol/L**. Hypochloraemia occurs when concentrations fall **below 95 mmol/L**, while levels **exceeding 110 mmol/L** are indicative of hyperchloraemia. Chloride levels are not affected by gender.

Section 10.1. Hypochloraemia

Hypochloraemia is an electrolyte disorder characterised by low serum levels which may result from loss of chloride itself, or from deficiencies in sodium or potassium. Inadequate serum chloride levels may be caused by inadequate intake, reduced absorption or increased loss.

Hypochloraemia - Causes

Hypochloraemia due to inadequate chloride intake alone may occur, but it is more likely to be associated with a low salt diet or the administration of dextrose without electrolytes. Water intoxication, which represents relative chlorine deficiency, may also occur, but rarely. Increased loss or reduced absorption may result from such gastrointestinal system causes as prolonged vomiting, prolonged diarrhoea, fistula, ileostomy, gastric surgery and nasogastric suctioning. It may also result from Addison's disease, salt-losing nephritis, syndrome of inappropriate antidiuretic hormone release, renal failure and some steroidal medications and diuretics, all of which affect renal function. Additionally, loss may be via the skin in excessive sweating or as a consequence of extensive burns.

Hypochloraemia – Signs & Symptoms

Mild hypochloraemia may be asymptomatic, but larger deficiencies can have deleterious or even fatal consequences. In moderate to severe cases mental, respiratory and neuromuscular functioning are most affected, giving rise to such signs and symptoms as confusion, agitation, irritability, muscle weakness, hyperactive deep tendon reflexes, spasm, cramping, twitching, tetany and possible paralysis. If metabolic alkalosis occurs, the body will try to compensate by conserving carbon dioxide via the respiratory system by reducing the rate and depth of respirations.

Hypochloraemia is indicated by serum chloride levels of **less than 95 mmol/L**, serum pH of **greater than 7.45** and **elevated serum carbon dioxide**.

Hypochloraemia - Treatment

Treatment is directed at treating any underlying cause and correcting the balance. If it is related to a sodium deficiency, salt intake is increased. If it is associated with low potassium, potassium chloride may be administered IV or orally. Losses through the gastrointestinal tract are managed by IV replacement fluids and, if applicable, oral supplements.

Section 10.2. Hyperchloraemia

While hyperchloraemia is defined as an excess of chloride in the serum, it usually occurs in conjunction with imbalances involving other electrolytes or acids and bases. Because of its direct relationship with sodium, hypernatraemia is often accompanies with high chloride levels. Hyperchloraemia may also occur with bicarbonate deficiency because of the inverse relationship that exists between these two electrolytes.

Hyperchloraemia – Causes

High serum chloride levels may result from increased intake of chloride or exchange of electrolytes. An increase in the intake of chloride as sodium chloride may elevate levels, especially if water is lost at the same time, as this causes chloride levels to rise. Intake may also be increased by the administration of hypertonic IV solutions (e.g. mannitol) and by the use of other medications than contain significant amounts of chloride (e.g. ammonium chloride). Sodium-based ion exchange resins may cause chlorine to be exchanged for potassium in the bowel. Metabolic acidosis resulting from significant bicarbonate loss from the serum may also cause hyperchloraemia because the inverse relationship between bicarbonate and chloride means that chloride levels rise as bicarbonate levels fall.

Increased absorption of chloride in the intestines may also raise serum levels. This is a particular risk in patients who have undergone uretero-intestinal anastomosis because of the additional chloride reabsorption from the urine that may occur in the bowel.

Decreased chloride loss, which may also cause hyperchloraemia, can result from disorders that affect the ability of the kidneys to excrete the electrolyte. Foremost among these are renal failure, hyperparathyroidism and hyperaldosteronism.

Hyperchloraemia – Signs & Symptoms

Signs and symptoms usually reflect the contributing factors rather than the hyperchloraemia itself. If it is due to elevated serum sodium, signs and symptoms are those of fluid overload, i.e. agitation, tachycardia, elevated blood pressure, oedema and dyspnoea. If it is due to acidosis, they include tachycardia, lethargy, muscle weakness and diminished mental functioning.

Hyperchloraemia – Diagnosis

Serum chloride levels of **more than 110 mmol/L** and, if metabolic acidosis is present, a **pH of less than 7.35**.

Hyperchloraemia – Treatment

Treatment consists of correcting the underlying condition and restoring balance. If hyperchloraemia is due to hypernatraemia, diuretics are given to eliminate sodium, which is accompanied by a decrease in chloride. If it is due to acidosis, the patient maybe ordered bicarbonate which reduces chloride levels. Hartmann's solution also known as Ringer's lactate solution may also be given as the liver converts the lactate to bicarbonate.

Summary Learning Points:

As a major anion in the ECF, the electrolyte chloride was introduced. Chloride and sodium have a very close association with each other in relation to the way chloride moves between the ICF and the ECF. Hypochloraemia and hyperchloraemia were discussed, including the causes, signs and symptoms and treatment of these conditions.

QUESTIONS & ANSWERS

Module 10: Specific - Chloride Imbalances

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. There are many causes of hyperchloraemia, including;

a) The administration of hypotonic IV fluids
b) The administration of potassium-based ion exchange resins
c) High bicarbonate levels
d) Metabolic acidosis

2. Hypochloraemia is a result of a number of reasons, including;

a) A high salt diet
b) A high potassium level
c) Nasogastric suctioning
d) Normal renal function

3. The treatment of hyperchloraemia can consist of …

a) Administration of IV Hartmann's solution
b) Administration of IV Albumin
c) Administration of a hypertonic solution
d) Administration of an anti-diuretic

4. Chloride levels in adults normally range between …

a) 95 – 110 mmol/L
b) 80 – 92 mmol/L
c) 70 – 85 mmol/L
d) 100 – 115 mmol/L

Module 11: Acids and Bases

The various chemical reactions that take place in the body to sustain life involve a number of acids and bases. These must be kept in a state of homeostasis or balance for the maintenance of metabolism and essential body functions.

Section 11.1 pH

The concept of pH is essential to understanding acids and bases. This term refers to the activity of hydrogen ions, and provides a measure on a scale of 0 to 14 of the acidity or alkalinity of a solution. According to this scale, seven is neutral; more than seven is more alkaline and less than seven is more acidic.

Section 11.2 Acids

Acids consist of molecules that give up hydrogen ions to other molecules. Bases are molecules that can accept hydrogen ions. A solution that contains more bases than acid has fewer hydrogen ions and consequently a higher pH. If its pH is above seven, the solution is bases or alkaline. A solution that contains more acid than bases has more hydrogen ions, so its pH is lower. Solutions with a pH below seven are acidic.

Maintenance of normal pH **between 7.35 and 7.45** is essential to well-being. Movements above or below this narrow range can adversely affect electrolyte balance, enzyme activity, muscle contractions and cellular function, any of which may interfere with vital functions. To maintain this fine balance and avoid life-threatening consequences, the body regulates acids and bases through three regulatory systems: chemical buffers, respiratory regulation and renal regulation.

Section 11.3 Chemical buffers

When pH rises or falls outside the normal range, chemical buffers act immediately to protect cells and tissues by combining with the excessive acid or base to temporarily neutralise its effects. The main chemical buffers, which occur in the blood, intracellular fluid and interstitial fluid, are bicarbonate, phosphate and protein.

Bicarbonate buffers

Of these, the bicarbonate buffering system is the most important. It buffers blood and interstitial fluid by means of a series of chemical reactions in which pairs of weak acids or bases combine with stronger bases or acids to reduce the concentration of the latter. Bicarbonate, a weak alkali, is commonly involved in this process in combination with weak acids like carbonic acid. The bicarbonate buffer system, by decreasing the strength of potentially dangerous substances, reduces their impact on pH balance. It is assisted in this function by the renal buffering system which regulates the production of bicarbonate, and the lungs which adjust the production of carbonic acid to meet requirements.

Protein buffers

Phosphate buffers also employ a series of chemical reactions to reduce the effects of pH changes. They react with either acids or bases to produce compounds that alter pH by affecting passive transportation, a process that is particularly effective in the renal tubules where high concentrations of phosphates occur.

Protein buffers operate in both intracellular and extracellular fluid. Their mode of operation is similar to that of bicarbonate buffers in that they bind with acids and bases to neutralise them. Proteins are the most abundant form of buffers in the body, and haemoglobin is the most common type of protein buffer. Haemoglobin in red blood cells combines with hydrogen ions to reduce excessive acidity.

As noted previously, the lungs and kidneys follow up the initial work of the chemical buffers in controlling acid-base imbalances.

Section 11.4 Respiratory regulation

The respiratory system contributes by regulating blood levels of carbon dioxide which, when combined with water, forms carbonic acid. Rising levels of carbonic acid increase blood pH. Chemoreceptors in the medulla register this change and initiate a process whereby the rate and depth of respirations are increased and more carbon dioxide is expelled from the body. This in turn causes a decrease in the amount of carbonic acid that is produced so that pH rises.

The respiratory system can manage about twice the amount of acids and bases as chemical buffering, but its effects are only temporary.

Section 11.5 Renal regulation

Long-term pH adjustments are made by the renal system. The kidneys can re-absorb acids or bases during the filtration process, or they can excrete them into the urine. They are also able to produce bicarbonate when supplies diminish. These functions, however, can take hours or days to complete.

If there is too much acid or not enough base in the blood, the resulting drop in pH stimulates the kidneys to reabsorb sodium bicarbonate. They may also increase the amount of hydrogen that is excreted, resulting in urine that is more acid than normal. The reabsorption of bicarbonate and the increased rate of hydrogen excretion cause more bicarbonate to be formed in the tubules and later distributed throughout the body. This in turn raises blood bicarbonate levels and increases pH. If pH rises because of increased base levels or reduced acid levels, the kidneys assist in re-establishing normal pH by excreting bicarbonate and retaining more hydrogen ions. Less bicarbonate and more acid causes a drop in pH.

The respiratory and renal systems operate co-operatively to overcome acid-base imbalances. If the cause is metabolic, the lungs compensate in one of two ways. In response to acidosis due to a deficiency of bicarbonate, the rate of breathing increases, blowing off more carbon dioxide and restoring pH to normal. If, on the other hand, excessive bicarbonate levels cause alkalosis, the lungs decrease the

rate of respirations which retains carbon dioxide, increases carbonic acid levels and lowers pH. If the acid-base imbalance stems from the respiratory system, the kidneys compensate by adjusting levels of bicarbonate or hydrogen ions. When carbon dioxide (and consequently carbonic acid) levels are high, and a state of acidosis occurs, the kidneys retain bicarbonate and increase the excretion of hydrogen ions, raising pH. When alkalosis occurs due to high levels of bicarbonate and/or low levels of acid, the kidneys lower the pH by excreting bicarbonate and retaining hydrogen.

Summary Learning Points:

Understanding the concept of PH and acid base balance is the key to appreciating homeostasis within the body. The concepts of pH and acid and bases were introduced. The normal pH of 7.35 – 7.45 was outlined.

The role of chemical buffers; bicarbonate, phosphate and protein were discussed with bicarbonate being the most important of them all. The role of the respiratory system in short term regulation of pH and the role of the renal system in the long-term control regulation of pH was also mentioned.

QUESTIONS & ANSWERS

Module 11: Acids and Bases

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. Which of the following statements is correct?

a) Normal body pH lies within the range 7.25 to 7.45
b) Bases give up hydrogen ions; acids receive them
c) Alkaline solutions have fewer hydrogen ions than acids
d) A pH of 7.5 is considered neutral

2. Acids consist of molecules that give up what substance to other molecules?

a) Hydrogen ions
b) Bicarbonate
c) pH
d) Carbonic acid

3. The most important chemical buffering system mechanism in acid base balance is:

a) Phosphate buffer
b) Bicarbonate buffer
c) Protein buffer
d) Renal buffer

4. Short term pH adjustments are made by which body system?

a) Cardiovascular
b) Lungs
c) Renal
d) Neurological

Module 12: Acid-base - Imbalances

Concept – Acidosis

Acidosis is a condition produced by higher than normal acid level (or lower than normal base level), and consequently a lower pH reading in the blood.

Concept – Alkalosis

Alkalosis occurs when there is a higher than normal level of alkali (or lower than normal acid level), causing an increased pH. Acidosis and alkalosis they actually serve to indicate the presence of underlying disorders.

There are two (2) sub-types, metabolic and respiratory, depending on their primary cause. Respiratory acidosis and alkalosis are associated with breathing or other pulmonary disorders whilst metabolic acidosis and alkalosis arise from ineffective renal compensation for acid or base imbalances.

Because large quantities of acids are a by-product of normal metabolism, effective mechanisms for their removal from the blood are essential, because even slight changes in its hydrogen concentration (or pH) can have harmful effects on cells and tissues. Maintaining acid levels within the narrow **pH range of 7.35 to 7.45** is achieved by the lungs, kidneys and various chemical buffering systems.

Section 12.1. Regulatory systems

The Respiratory system (lungs)

The lungs help to maintain levels in this narrow range by releasing carbon dioxide on exhalation. Carbon dioxide is a mildly acidic waste product of oxygen metabolism which occurs constantly and involves all cells. Consequently, it is continuously produced and released into the bloodstream. If it accumulates, it causes blood acidity to increase and its pH to decrease. However, on reaching the lungs, the carbon dioxide is released from the blood and eliminated from the body on expiration, so that the pH balance in the blood is restored. Sensors in the brain monitor carbon dioxide levels in the blood and adjust the rate and depth of respirations to accommodate the carbon dioxide load, causing faster, deeper breaths when the pH is low, and slower, shallower respirations when it is higher. The respiratory system is therefore able to make adjustments to blood pH in short time frames, usually minutes.

The Renal system (kidneys)

The kidneys provide the second mechanism for regulating blood acid levels. They adjust the amount of acid or base that is filtered from the blood and excreted in the urine, thus raising or lowering the pH. However, renal regulation occurs more slowly than that provide by the lungs, so that compensation usually takes days.

Chemical buffering agents

The third regulatory mechanism is made up of the chemical buffering systems. Each of these comprises a combination of a weak acid and a weak base that remain in balance while the blood pH is normal. But, because their function is to prevent sudden changes in acid or base levels, when there is an increase in one of the other, the appropriate buffer is released to minimise its effects. For example, if the blood becomes more acidic, an alkaline buffering agent is released to reduce the concentration of acid; or if there is an increase in alkalinity, a weak acid is released to counter it. The most important buffer system involves *carbonic acid* (a weak acid formed when carbon dioxide dissolves in the blood), and *bicarbonate* (the corresponding alkali).

Section 12.2 Acid-base imbalances

ACIDOSIS

Respiratory Acidosis

Respiratory Acidosis - Definition

Respiratory acidosis is an acid-base disturbance that occurs when the lungs are unable to remove all of the carbon dioxide from the blood due to reduced alveolar ventilation. The resultant hypercapnia causes the blood to become excessively acidic.

Respiration involves the exchange of oxygen from inhaled air for carbon dioxide from the blood, a process which takes place across the semipermeable membranes between the alveoli and the capillaries. When this exchange is impaired and carbon dioxide builds up in the blood, carbonic acid is formed which reduces the pH of the blood.

Respiratory Acidosis - Causes

Impaired gaseous exchange causing acidosis may be due to:

- diseases that affect pulmonary tissue and impair the function of the lungs, e.g. chronic obstructive pulmonary disease, severe asthma, emphysema, chronic bronchitis, pulmonary oedema

- damage to the central nervous system as a result of such mechanisms as stroke, trauma or tumour which interfere with the respiratory centre in the brain or with the transmission of neural impulses to and from the lungs
- obstruction of the airway by foreign body, vomitus or swelling

- neuromuscular disorders that affect the muscles involved in breathing and consequently impair lung function, e.g. myasthenia gravis, poliomyelitis, Guillain-Barre syndrome

- some drugs such as sedatives, narcotics and anaesthetic agents that depress the respiratory centre

- some conditions that cause metabolic alkalosis may also trigger respiratory acidosis.

Respiratory acidosis may be chronic or acute. Because chronic respiratory acidosis develops over a long period of time, the kidneys are able to compensate to some degree by excreting more acid and maintaining levels of bicarbonate to restore the acid-base balance. Acute respiratory acidosis, on the other hand, is characterised by sudden and rapid accumulation of carbon dioxide.

Respiratory Acidosis – Signs & Symptoms

Early symptoms are primarily respiratory, with shortness of breath, slowed or difficult breathing, wheezing and persistent coughing. Central nervous symptoms such as restlessness, drowsiness, headache and irritability may arise, and progress to tremor, lethargy and confusion as acidosis becomes more severe. Hypertension and distension of the blood vessels in the eyes may also be noted. Other possible cardiovascular symptoms include tachycardia and dysrhythmias. In some cases cyanosis, a sign of metabolic acidosis, may occur as a result of lack of oxygen brought about by compensatory mechanisms. Severe acidosis can lead to coma and death.

Respiratory Acidosis - Treatment

Treatment is concerned with correcting the causative disorder. Chronic lung disease may be treated with bronchodilators or steroids. Supplemental oxygen may also be used, although with caution as oversupply of oxygen may exacerbate acidosis. Antibiotics may be used to treat infections associated with acidosis. Measures appropriate to the type of airway obstruction are taken to resolve or relieve it, e.g. endoscopic removal of foreign body or adrenalin to relieve swelling due to anaphylaxis. Naloxone may be administered to counteract the effects of narcotic overdose. Where metabolic alkalosis has resulted from the effects of compensatory mechanisms for acidosis, its symptoms may need to be treated as well.

Metabolic Acidosis

Metabolic Acidosis - Definition

Metabolic acidosis is a pH imbalance in which there are elevated levels of acid in the blood and not enough bicarbonate to neutralise its effects.

Metabolic Acidosis - Causes

While elevated blood acid levels may arise from a variety of causes, the following are the most common reasons:

1. A reduction of bicarbonate levels,
2. An increase in hydrogen ions, and
3. An inadequate excretion of acid.

Cause - Reduction in bicarbonate levels

Acid is a by-product of fat metabolism and other processes in the body. It is normally neutralised by bicarbonate, but some conditions deplete the supply of bicarbonate so that there is not enough available to counteract the acid. Such conditions include:

- prolonged diarrhoea, which results in excessive bicarbonate loss in the faeces
- renal tubular acidosis, a relatively rare kidney disorder that impairs the reabsorption of bicarbonate into the bloodstream
- gastrointestinal conditions such as fistulas and disorders requiring ileostomy, colostomy or uretero-intestinal anastomoses, all of which are associated with increased bicarbonate loss.

Cause - Increased hydrogen ions

Increased hydrogen ion content in the blood may be due to internal (endogenous) or external (exogenous) reasons. Internal (endogenous) causes of excessive acid include:

- high fat, low carbohydrate diet which causes increased acid production from fat metabolism
- poorly controlled diabetes mellitus in which insufficient insulin causes fat to be metabolised instead of carbohydrate, increasing acid production in the form of ketone bodies which may lead to ketoacidosis
- malnutrition and chronic alcoholism, both of which may cause ketone production
- build-up of lactic acid due to severe illness, stress or prolonged strenuous activity
- shock.

External (exogenous) causes of hydrogen ion gain may include the ingestion of acids or substances that metabolise into acids. These include both toxins and overdose of normally harmless substances, particularly medications. Examples of toxic agents are ethylene glycol, methanol, formaldehyde, cyanide, ethanol, strychnine, carbon monoxide and heavy metals. Similar effects may also arise from excessive amounts of such drugs as NSAID's, valproic acid, isoniazid, ACE inhibitors, beta blockers and spironalactone, but salicylate poisoning occurs most frequently.

Cause - Inadequate excretion of acid

Inadequate acid excretion may also be a factor in metabolic acidosis. Because the kidneys play an important role in acid excretion, any impairment to this function can result in the accumulation of hydrogen ions. Renal failure is the most significant of these because it reduces the glomerular filtration rate, leading to a reduction in the capacity to excrete acid and other wastes.

Metabolic Acidosis – Signs & Symptoms

In mild acidosis the clinical features may be those of the underlying cause, although in come cases fatigue, loss of appetite, nausea and vomiting may also occur. Rate and depth of respirations may increase as the lungs attempt to reduce acidity by 'blowing off' more carbon dioxide.

In more severe cases signs and symptoms specific to acidosis occur in addition to those of the underlying condition. Initially there may be increasing drowsiness, weakness and nausea and confusion may occur. As the acid load continues to accumulate causing severe acidosis (pH < 7.2), cardiovascular, respiratory, central nervous system and gastrointestinal effects develop. Nausea may become even more severe and abdominal pain may occur. Kussmaul respirations (abnormally deep with increasing rapidity) may be noted and CNS function may deteriorate to coma. Myocardial contractility may be impaired, possibly leading to cardiogenic shock and death.

Metabolic Acidosis - Diagnosis

Diagnosis is made on the basis of the presenting symptoms and is confirmed by the following diagnostic results:

- Blood pH below 7.35
- Reduced bicarbonate levels (below 22 mmol/L)
- Urine pH less than 4.5, depending on the causative condition potassium; glucose, ketones or lactic acid may exceed the upper limit of their respective ranges, depending on the underlying condition.

Metabolic Acidosis - Treatment

Treatment is determined primarily by the causative mechanism. If, for instance a toxin is involved, treatment involves removal of the offending substance or initiation of the appropriate antidote. If the acidosis is due to diabetes, interventions to control blood glucose levels will be required. Persistent diarrhoea would need to be treated, bicarbonate-reducing fistulas repaired and bicarbonate loss via various anastomoses addressed.

Management of the acidosis itself may simply involve the administration of IV fluids. In more severe cases, bicarbonate may be given intravenously, although this therapy is controversial. It has been found effective in managing the condition where certain causative factors apply, but in general it is temporary in its effectiveness and has been associated with adverse outcomes.

ALKALOSIS

Respiratory Alkalosis

Respiratory Alkalosis – Definition

Respiratory alkalosis is an acid-base imbalance in which carbon dioxide levels in the blood fall below the normal range. It produces a shift in pH, causing the extracellular fluid to become more alkaline.

The condition results from deep, rapid breathing or hyperventilation. When hyperventilation occurs, the exchange of oxygen for carbon dioxide in the alveoli is speeded up, causing excessive exhalation of carbon dioxide which reduces the acidity of the blood, thereby raising its pH.

Respiratory Alkalosis – Causes

Hyperventilation may be caused by a number of factors. It can be a consequence of respiratory conditions like pneumonia or asthma, but it is more commonly associated with anxiety, serious infections, carbon monoxide poisoning, drug overdose or liver disease. It may also arise from CNS disorders which affect the respiratory centres in the brain or from other stresses including pregnancy, high altitude and metabolic acidosis.

Respiratory Alkalosis – Signs & Symptoms

Signs and symptoms of respiratory alkalosis depend on the extent of the drop in serum carbon dioxide and the rate at which it falls. Hyperventilation (tachypnoea or hyperpnoea) is the primary sign and often the only sign. Acute respiratory alkalosis, in which there is a sudden and significant decrease in carbon dioxide, may produce light-headedness, confusion, tingling peripherally and around the mouth, cramps and fainting, probably due to changes to cerebral blood flow and pH. In severe cases there may be tetany, with Chvostek's and Trousseau's signs and carpopedal spasms, due to serum ionised calcium depletion.

Respiratory Alkalosis – Diagnosis

Confirmation of diagnosis is based on arterial blood gas analysis (pH and bicarbonate levels) and serum electrolytes (decreased ionised calcium and possibly minor reductions in phosphate and potassium).

Respiratory Alkalosis – Treatment

Treatment is directed at the underlying disorder. As respiratory alkalosis itself is not life threatening, it is not necessary to initiate measures to raise serum pH, although rebreathing from a paper bag may be helpful where anxiety is the cause of hyperventilation. This intervention, though, is contraindicated where there may be CNS disorders as the pH of the cerebrospinal fluid may already be below normal.

Other interventions will depend on the causative conditions. Appropriate medications are employed to manage such problems as infections, lung disease, liver disease and anxiety disorders. Treatment for poisoning is required if respiratory alkalosis is due to drug toxicity, as is the case with salicylate overdose and, where it arises as a consequence of metabolic acidosis, symptoms of that condition may need to be treated as well.

Metabolic Alkalosis

Metabolic Alkalosis – Definition

Metabolic alkalosis is an acid-base imbalance in which there is an excess of alkali and not enough acid to neutralise it. It may be mild with causes that are readily treatable, or it may be more severe and persistent, indicating a serious problem with the kidneys or other major organs.

Metabolic Alkalosis – Causes

It involves excessive concentrations of bicarbonate in the serum which may be caused by:

- Acid loss,
- Administration of alkali,
- Intracellular shift of hydrogen ions, or
- Bicarbonate retention.

Acid loss may occur from the gastrointestinal tract with persistent vomiting, gastric suctioning or diarrhoea. It may also be lost via the kidneys as a result of thiazide and loop diuretic use and disorders such as hyperaldosteronism, hypokalaemia and hypomagnesaemia. Alkalosis may result from excessive intake of bicarbonate where there is renal failure or from chronic ingestion of calcium carbonate antacids. Conditions such as hyperkalaemia that cause hydrogen ions to move into the cells from the extracellular fluid increase the concentration of bicarbonate in the serum. Alkalosis may also result from volume depletion or hypokalaemia which both stimulate increased bicarbonate reabsorption. However, impairment of renal bicarbonate excretion must be present for metabolic alkalosis to persist.

Metabolic Alkalosis – Signs & Symptoms

Signs and symptoms may not be evident in mild cases and alkalosis is diagnosed on the basis of elevated blood pH and below normal sodium, potassium and chloride serum levels. Urine pH may also be elevated. In more serious cases symptoms may include:

- Bradynoea, possibly with periods of apnoea of 15 seconds or longer
- Cyanosis
- Irritability, hyperexcitability, mental confusion
- Twitching, muscular weakness, impaired gastrointestinal peristalsis.

- Tetany may occur if serum calcium falls
- Rapid and irregular pulse and hypotension
- CNS symptoms may progress to convulsions and coma.

Metabolic Alkalosis – Treatment

Treatment initially focuses on correcting the imbalance and usually involved IV replacement of extracellular fluid. However, in more severe cases, especially where there is marked hypokalaemia and ECG changes an intensive care setting is indicated. Because alkalosis cannot be corrected where there is a severe potassium deficiency, potassium chloride is used to restore levels. Measures to regulate blood pressure and heart rate and rhythm and to control nausea and vomiting may also be required. The underlying cause of the alkalosis must then be identified and treated.

Anion gap

An anion gap is the difference between the sum of the main cations (Na^+ and K^+) and the main anions (Cl- and HCO^3). The anion gap is due to unmeasured anions such as lactate, phosphates and sulphates. The normal anion gap is less than 15. When the anion gap is increased in a metabolic acidosis, it suggests that an excess of some unmeasured anion is causing the acidosis.

Common causes include:

• Ketoacid overproduction due to fat metabolism (diabetes, alcohol, starvation)
• Lactic acid overproduction due to hypoxia, shock or tissue ischaemia
• Inability to excrete acids (sulphate and phosphate) due to renal disease (usually with an elevated BUN (blood urea nitrogen) and creatinine
• An overdose of medications such as salicylates (e.g. aspirin)
• Toxins such as ethylene glycol, methanol

Summary Learning Points:

The body constantly aims to achieve a balanced acid-base level. Three regulatory systems are key to maintain the pH in this limited range; the respiratory system, the renal system and chemical buffering agents. The primary mechanism is short term changes which are facilitated through carbon dioxide production and release in relation to acid-base changes. The secondary mechanism is the renal system which controls the filtration of acid-base levels in the body, this is a longer-term response. Finally, the chemical buffer agent carbonic acid (weak acid) or bicarbonate (weak base) are released to assist in controlling the pH levels.

The concepts of acidosis and alkalosis was briefly discussed. The two (2) sub-types of acid-base imbalances were elaborated on, respiratory and metabolic. The definition, diagnosis, signs and symptoms, treatment of respiratory acidosis, respiratory alkalosis, metabolic acidosis and metabolic alkalosis were clearly outlined.

QUESTIONS & ANSWERS

Module 12: Acids and Base Imbalances

NAME_____ **DATE**_____

Circle the correct answer. Unless informed otherwise, there is only one correct response for each question.

1. Laboratory findings which include blood pH of 7.2, serum bicarbonate of 12 mmol/L and serum potassium of 5.6 mmol/L are indicative of:

a) Metabolic acidosis
b) Hypokalaemia
c) Metabolic alkalosis
d) Hypovolaemia

2. The most common external (exogenous) form of hydrogen ion increase leading to metabolic acidosis is:

a) High fat, low carbohydrate diet
b) Malnutrition
c) Medication overdose
d) Shock

3. Simple management of metabolic acidosis includes …?

a) Intravenous adrenaline
b) Intravenous bicarbonate
c) Intravenous magnesium
d) Intravenous therapy

4. The primary sign of respiratory alkalosis is:

a) Tachypnoea or hyperpnoea
b) Bradypnoea
c) Dyspnoea and shortness of breath
d) Apnoea

5. Which of the following statements about acid-base imbalances is correct?

a) Acidosis occurs when blood acid levels are ↑ and the pH reading of blood is ↑
b) There are two (2) sub-types of alkalosis; metabolic & respiratory alkalosis
c) Renal regulation of acid-base balance occurs more quickly than in the lungs
d) Sensors in the kidneys primarily monitor carbon dioxide levels in the blood

6. Bradypnoea accompanied by periods of apnoea lasting up to 15 seconds is a symptom of severe:

a) Respiratory alkalosis
b) Respiratory acidosis
c) Metabolic alkalosis
d) Metabolic acidosis

7. Which of the following is a common cause of respiratory alkalosis?

a) Prolonged diarrhoea
b) Hypercapnia
c) Anxiety
d) Renal failure

8. Which of the following buffering systems is mainly responsible for long-term pH adjustment?

a) Respiratory buffering system
b) Renal buffering system
c) Bicarbonate buffering system
d) Protein buffering system

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Fluids & Electrolytes Package Evaluation

Date: _____ How long did this package take to complete? _____

Please indicate your response to each of these statements by ticking the appropriate box and return to the Nurse Educator at your facility.

	Strongly Disagree	Disagree	Neutral	Agree	Strongly Agree
Overall, I found this learning package worth while	❑	❑	❑	❑	❑
The way in which the learning package was presented made it easy to understand	❑	❑	❑	❑	❑
My knowledge of this topic was improved after completing this learning package	❑	❑	❑	❑	❑
My skills in this area have been enhanced since completing this learning package	❑	❑	❑	❑	❑
The resources provided were sufficient for me to answer the review questions adequately	❑	❑	❑	❑	❑
I would recommend the learning package to others	❑	❑	❑	❑	❑
I will be able to apply knowledge and skills acquired in my clinical practice	❑	❑	❑	❑	❑

Comments (Optional)

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Thank you for taking the time to complete this evaluation. Your comments are valued and appreciated. Please return this form to the Nurse Educator