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# Regulation of water, sodium and potassium: implications for practice

NS77 Edwards S (2001) Regulation of water, sodium and potassium: implications for practice. *Nursing Standard*. 15, 22, 36-42. Date of acceptance: September 29 2000.

## in brief

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### Summary

Regulation of fluid balance is a complex subject. Sharon Edwards discusses the physiological principles involved.

### Keywords

- Anatomy and physiology
- Biological sciences

These key words are based on subject headings from the British Nursing Index. This article has been subject to double-blind review.

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## Aims and intended learning outcomes

The aim of this article is to provide an overview of the physiology and regulation of water, sodium and potassium balance in the body, and the associated implications for practice. Understanding the physiological principles involved is important to ensure patients' fluid balance is maintained during and following treatment, to explain incidents that occur in clinical practice and their rationale.

After reading this article you should be able to:

- Understand fluid, sodium and potassium physiology and homeostasis.
- Indicate the conditions and treatments that influence fluid, sodium and potassium physiology.
- Demonstrate how fluid regimens and hyperglycaemia, hyperlipidaemia and hyperproteinaemia alter fluid, sodium and potassium homeostasis.

## Introduction

To maintain fluid and electrolyte balance, water, sodium and potassium are in constant motion between the intracellular and extracellular body compartments (Edwards 1998).

Body fluids are complex aqueous solutions, in which biochemically distinct compartments are divided by the plasma membrane (between the intracellular and extracellular compartments) or by specialised cell layers (between intravascular, interstitial and transcellular compartments). Total body water (TBW) accounts for between 50 per cent (females) and 60 per cent (males) of an adult's total body weight (Marieb 1998).

The average adult has a normal fluid intake of 2-2.5l per day from drinks, water in food or from oxidation of food during metabolism, which produces 200-300ml per day (Rooney 1995). Fluid is lost via

the kidneys, gastrointestinal tract, respiratory tract and skin. In the healthy adult (Marieb 1998):

- The kidneys excrete approximately 1.5-2l fluid daily, depending on intake.
- Faecal loss amounts to 300ml daily.
- Losses from the skin and respiratory passages account for approximately 0.6/1l daily.

## Body fluid compartments

Table 1 lists the different body fluid compartments. Intracellular fluid (ICF) is defined as all the body water within cells. It constitutes about 40 per cent of total body weight. It is inhomogeneous, having different pH and ionic composition depending on the organ or tissue involved. Extracellular fluid (ECF) is found outside the cells and is commonly subdivided into plasma and interstitial fluid volumes. It constitutes about 20 per cent of total body weight. With acute or chronic illness, ICF volume is reduced and ECF volume is increased, and might even exceed ICF volume.

Plasma, or intravascular fluid, is contained in blood vessels and plasma volume, not including the volume of the blood cells. Interstitial fluid, or the third space, is water between the cells and outside the blood vessels. It is the fluid that accumulates following burns and soft tissue injuries (Rooney 1995). The transcellular compartment is a collection of biochemically distinct fluids including cerebrospinal, synovial, pleural, pericardial, intraocular and peritoneal fluids and digestive secretions, which are separated from the interstitial compartment by a layer of epithelium (Flanning 2000). Transcellular components are formed by transport activity of cells. The fluid is extracellular and is often considered as part of the interstitial volume (Worthley 1999).



The transcellular compartment is small compared to the intravascular, interstitial and intracellular compartments. However, when larger volumes occur, such as in diseased states (for example, bowel obstruction or cirrhosis with ascites), it is formed at the expense of the remaining interstitial and plasma volumes (Worthley 1999).

The extracellular fluid and intracellular fluid contain the major electrolytes, that is, products of ionic compound dissociation in solution. The concentration of electrolytes is measured in milliequivalents per litre (mEq/l). Cations carry a positive charge, and anions carry a negative charge. The main cation of ECF is sodium, whereas that of ICF is potassium. The main anions of ECF are chloride and bicarbonate, while those of ICF are proteins and organic phosphates. Molecules such as glucose or urea, which are uncharged in solution, are also present.

#### Movement between compartments

Several factors contribute to maintaining the differences in solute composition between ECF and ICF. To pass between ECF and ICF, solutes must cross the plasma membrane. Some move freely across the membrane due to concentration differences, but the majority require some form of assistance. Diffusion is the random movement of particles in all directions from an area of greater concentration to an area of lesser concentration (Bove 1994). Diffusion depends on:

- Membrane permeability.
  - The particle's electrical charge.
  - The pressure gradient around the membrane.
- An example is when sodium moves from the outside to the inside of cells because the sodium concentration is higher on the outside.

Osmosis is the movement of water from a solution of lesser to a solution of greater solute concentration. In osmosis, the membrane is permeable to water, but only selectively permeable to particles (Bove 1994). If, for example, pure water in a beaker is separated from a highly concentrated water and salt solution by such a membrane, water will flow across the membrane into the salt solution.

Osmolality is a measure of the osmotically active particles in solution for a given unit of mass. Isotonic solutions have equal concentrations of osmotically active particles in solution (whole blood, Hartmann's solution, 0.18% NaCl in 4% dextrose, 5% dextrose, and 0.9% saline). Hypotonic solutions (water, 0.45% normal saline and 4% glucose) have a lower concentration of osmotically active particles than isotonic solutions. Hypertonic solutions (25% mannitol, 10% glucose, 5-10%

**Table 1. Major fluid compartments of the body**

Total body water volume = 42l, 60 per cent of body weight			
Intracellular fluid (ICF) volume = 28l, 40 per cent of body weight		Extracellular fluid (ECF) volume =14l, 20 per cent of body weight	
Approximate volume and percentage of body weight values are noted for a 70kg male (Marieb 1998)		Interstitial fluid volume = 11l, 80% of ECF	Plasma volume = 3l, 20% of ECF

glucose combined with 0.2-0.9% normal saline and 25% albumin) have a higher concentration of osmotically active particles than isotonic solutions (Galbraith *et al* 1997).

The concentration of osmotically active solutes in the fluid compartments determines the distribution of body water. Osmotically active solutes that cannot move freely between fluid compartments influence the movement of water into and out of that compartment (Flanning 2000).

Active transport is the movement of particles against a concentration gradient. This is provided by transmembrane proteins that require energy to passively enhance permeability to a given substance by providing a channel, or pore, for easy passage of water-soluble/lipid-insoluble molecules. An example is the sodium/potassium pump, which moves sodium from the inside of a cell to the outside, where sodium concentration is greater, and returns potassium to the inside of the cell, where potassium concentration is greater.

Filtration is the movement of water and particles from an area of high pressure to an area of low pressure through a semipermeable membrane, for example, renal glomerular filtration of blood.

#### Implications for practice

Factors in practice that cause water, sodium and potassium to move abnormally between fluid compartments are physical conditions (trauma and shock, and respiratory acidosis and alkalosis) and treatments (blood transfusions, rewarming following hypothermia, drugs and insulin).

**Trauma and shock** Any type of trauma, shock or cell damage (such as surgery, myocardial infarction, head injury) will automatically trigger an inflammatory response (Bove 1994).

This causes a rapid and proportional increase in capillary permeability, due to the release of localised mediators, such as kinins. The permeability causes movement of water, electrolytes and other particles (such as albumin) into the interstitial spaces to allow the necessary factors to reach the

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site of injury. This third-space fluid shift (Bove 1994) leads to localised swelling and lymphatic blockage, causing localised interstitial oedema. A patient might then appear 'dry' or hypovolaemic, having a low circulating blood volume as fluid has moved into interstitial spaces, yet the amount of body water might not have changed.

The release of cell mediators causes a reduction in blood pressure and an increase in heart rate, further compounding the appearance of a hypovolaemic state (Huddleston 1992). This state stimulates compensatory homeostatic control mechanisms to reabsorb sodium and water and to cause vasoconstriction, in an effort to restore circulating volume and increase blood pressure. As a result, venous capacity is decreased to match the smaller blood volume, so that adequate transport of oxygen and nutrients is maintained.

As overfilling of the third space becomes critical, the ability of homeostatic mechanisms to compensate reduces: the central venous pressure (CVP), diastolic filling pressure, stroke volume and systemic arterial blood pressure fall, and oxygen diffusion from capillary to cell is impaired, causing hypoxic damage to organs. This process might predispose a major trauma victim to excessive stimulation of inflammatory pathways, leading to a severe and prolonged increase in vascular permeability with severe interstitial oedema in capillary beds remote from the site of injury. The result of this is hypovolaemia, which is associated with systemic inflammatory response syndrome (SIRS) (Reilly and Yucha 1994).

Following injury, potassium moves out of the cell. In a normal cell, high intracellular potassium and low intracellular sodium concentration is maintained by the sodium/potassium pump. The damage of trauma or shock affects the pump action, increasing sodium inside cells. This results in water entering cells by osmosis, causing cellular swelling and distortion, which might interfere with cellular organelle function (Buckman *et al* 1992). If renal function is sustained, potassium will be excreted and, as cell repair begins, hypokalaemia (inadequate levels of potassium in the bloodstream) will develop without an adequate supplement of potassium.

**Acidosis and alkalosis** Cellular uptake of potassium is regulated by the sodium/potassium pump, while movement of potassium out of the cell is governed by passive forces (cell membrane permeability and chemical and electrical gradients to the potassium ions).

Respiratory or metabolic acidosis promotes a shift of potassium out of the cell, leading to higher than normal levels of potassium in the bloodstream (hyperkalaemia). In acidosis, hydrogen ions move

into the cells in exchange for ICF potassium and sodium, increasing the ECF pH. Thus, hyperkalaemia and acidosis often occur together. In the event of a respiratory or metabolic alkalosis, ECF potassium exchanges with ICF hydrogen (hypokalaemia) correcting the alkalosis by reducing the pH of the ECF. A metabolic alkalosis enhances renal potassium loss by encouraging distal nephron sodium/potassium, in preference to sodium/hydrogen exchange.

## TIME OUT 1

David is 18 and has asthma. He develops severe breathing difficulties and is admitted to A&E with an asthma attack. On admission to your ward his arterial blood gases from A&E reveal that he has a respiratory alkalosis and laboratory blood results show that David's potassium level is low.

- Explain why his potassium level is low.
- What is potassium exchanged for in a respiratory acidosis and why?
- What occurs to the ECF pH during respiratory alkalosis? What other effects might this have?



**Blood transfusions** When blood is stored, the sodium and potassium concentration alters. A unit of stored blood contains approximately 75-80mEq sodium and 5-7mEq potassium (Contreras 1992). Patients with normal cardiac and renal function will probably be able to handle the increase in sodium and potassium. However, in patients with severe trauma and shock who have cardiac and renal dysfunction, the sodium and potassium content of stored blood can have profound effects. Initiation of homeostatic mechanisms to enhance sodium and water reabsorption can promote fluid overload. As a consequence, diuretic therapy might be given following each unit of blood or every consecutive unit (Edwards 2000).

## TIME OUT 2

Arthur needs a blood transfusion following surgery to repair a hernia. He has angina, which is stabilised on drug therapy. During his second unit of blood he feels breathless. The doctor takes a blood sample and prescribes frusemide 20mg.

- What might be happening to Arthur during this blood transfusion?
- What are the consequences of an increased sodium and potassium load, bearing in mind Arthur's heart condition?
- What homeostatic mechanisms might be allowing Arthur to become breathless?
- Why might frusemide have been prescribed?





**Rewarming following hypothermia** When rewarming patients following hypothermia, frequent estimations of serum potassium should be made. As the patient's temperature decreases, alterations in potassium levels occur due to changes in the sodium/potassium pump (Edwards 1999). This can result in increased ECF potassium levels during hypothermia. On rewarming, the potassium equilibrium is returned to normal and hypokalaemia might follow (Fritsch 1995). In the hypothermic patient, a high blood potassium level is often used as a predictor of non-survival, particularly for levels greater than 10mEq/l (Schaller *et al* 1992).

**Insulin** Potassium depletion occurs when insulin treatment is initiated. Insulin promotes cellular uptake of potassium, causing a deficit in ECF potassium independent of the movement of glucose. An insulin regimen of IV dextrose 50g with 20 units of soluble insulin, might be used in hyperkalaemia caused by excessive intake, severe tissue damage, decreased excretion (such as renal failure) or body fluid compartment shift (Worthley 1999). Severe hypokalaemia might occur if insulin is administered without potassium supplements.

Because insulin promotes cellular entry of potassium, insulin deficits, which occur with conditions such as diabetic ketoacidosis, are accompanied by hyperkalaemia. Potassium homeostasis is not possible in the absence of renal function (Flanning 2000), when an increase in serum potassium will eventually lead to the need for haemodialysis.

**Drugs** Beta<sub>2</sub> adrenergic agonists (such as salbutamol and terbutaline) promote cellular uptake of potassium (hypokalaemia), whereas alpha-adrenergic agonists (such as clonidine and its derivative apraclonidine) cause a shift of potassium from the ICF to the ECF (hyperkalaemia).

Potassium-sparing diuretics (such as spironolactone) inhibit sodium reabsorption and potassium and hydrogen secretion by the distal tubule and might contribute to hyperkalaemia. However, these diuretics are used in combination with diuretics that cause potassium wasting in an attempt to balance renal potassium gains and losses (Galbraith *et al* 1997).

Osmotic diuretics interfere with osmosis; they tend to stay in the blood and increase osmolality. These diuretics are useful in the treatment of oedematous states, such as glaucoma and elevation of intracranial pressure (Galbraith *et al* 1997). In the presence of mannitol in the ECF, fluid in the interstitial space will pass from this area into the hypertonic (mannitol-containing) blood and to the kidneys for removal. Osmotic diuretics act quickly and are useful in emergency

situations. However, prolonged use can result in reversal of the hypertonic state to interstitial/intracellular, drawing fluid back into the interstitial space and worsening the oedematous state.

Digitalis overdose can cause hyperkalaemia by inhibiting the sodium/potassium pump, which maintains high intracellular potassium and high extracellular sodium. In addition, hypokalaemia and the administration of digoxin can enhance the efficiency of the drug.

#### Fluid, sodium and potassium homeostasis

Fluid and electrolyte homeostasis is maintained by the kidneys, brain, lungs, skin and gastrointestinal system. Therefore, if patients have conditions involving these organs, fluid, sodium and potassium balance might be affected. Movement of water and solutes is influenced by the complex interaction of regulatory processes, receptor responses, enzymes, and hormones. Sodium balance regulates ECF volume and various hormonal and physical ECF volume sensors control sodium excretion.

Mechanisms for maintaining water balance in the body include hypothalamic regulation of thirst and regulation of renal excretion of water.

**Osmoreceptors** If concentrations of sodium are increased, as in the case when there is a loss of extracellular water (Bove 1994), osmoreceptors in the hypothalamus are stimulated. Osmoreceptors are highly specialised hypothalamic neurones that continually monitor the solute concentration (and thus water concentration) of the blood. When solutes threaten to become too concentrated, as in conditions that cause an increased sodium concentration (excessive sweating, inadequate fluid intake, burns), osmoreceptors transmit impulses to the hypothalamic neurones that synthesise and release anti-diuretic hormone (ADH).

The secretion of ADH from the pituitary gland is initiated by an increase in plasma osmolality or a decrease in circulating blood volume and/or a lowered blood pressure. An increase in plasma osmolality occurs with a deficit of water or an excess of sodium in relation to water, resulting in a decreased extracellular and interstitial fluid volume. The primary stimulus for release of ADH is elevation of plasma osmolality. A secondary stimulus is depletion of plasma volume and the stress response.

ADH inhibits or prevents urine formation by increasing the permeability of renal tubular cells to water. Water is then reabsorbed from the distal tubules and collecting ducts of the kidney and returned to the circulation. As a result, less urine is

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produced, urine concentration increases and thirst is aroused. The reabsorbed water decreases plasma osmolality, returning it to normal. Blood volume also increases, thereby improving venous return to the heart, cardiac output, and blood pressure.

Thirst is experienced when water loss equals 2 per cent of body weight (about 700ml) or when there is an increase in osmolality. Dry mouth, hyperosmolality, and plasma volume depletion activate the osmoreceptors. The action of osmoreceptors then causes thirst.

**Baroreceptors** Baroreceptors are specialised nerve endings that are sensitive to changes in volume and pressure. They respond to any decrease in arterial blood pressure, whether it is due to haemorrhage, peripheral blood pooling, or a decrease in myocardial contractility. With decreased arterial pressure or volume depletion, such as dehydration from vomiting, diarrhoea, or excessive sweating, baroreceptors stimulate the release of ADH from the pituitary gland. The reabsorption of water mediated by ADH then promotes the restoration of plasma volume.

## Regulation of sodium and potassium

Sodium balance is achieved by the interaction of a number of neurohormonal systems which influence renal reabsorption or excretion of sodium. The regulation of potassium is closely tied to that of sodium.

**Renin-angiotensin-aldosterone system** The kidneys have a complex role in restoring ECF volume and increasing systemic blood pressure. This set of interlinked processes involves the renin-angiotensin-aldosterone system. This system is stimulated principally when there is a decrease in blood pressure. A decrease in kidney perfusion activates the mechanism. Renin splits angiotensinogen, a substrate present in plasma, into angiotensin. This acts as a vasoconstrictor, and stimulates the release of aldosterone.

Aldosterone is a steroid hormone synthesised and secreted from the adrenal cortex. Its release is influenced by circulating blood volume and plasma concentration of sodium and potassium (when sodium levels are depressed or potassium levels are increased). Its action is to increase sodium reabsorption in the renal tubules. Because water follows sodium, there is a subsequent increase in intravascular volume, resulting in increased venous return to the heart, increased cardiac output and increased blood pressure. In exchange for sodium, potassium is excreted by the distal tubule of the kidney. Addison's disease results in decreased production and secretion of aldosterone. Aldosterone enhances potassium

excretion through mechanisms similar to those which result in sodium retention. Stimulation of aldosterone release occurs following an increase in plasma potassium levels, which increases intracellular concentrations of potassium ions.

**Atrial natriuretic peptide (ANP)** ANP is produced by the atrial muscle of the heart in response to overstretching of the atria wall. Its function is renal elimination of sodium to control sodium and water balance. It also increases glomerular filtration rate and inhibits aldosterone production and secretion.

**Blood urea nitrogen (BUN) creatinine ratio** BUN reflects changes in urine concentration based on serum levels of urea and creatinine. Fever, sepsis, steroid administration, burns and excessive muscle activity (such as seizures) increase BUN levels because of increased protein metabolism or dehydration associated with these problems. The increased BUN causes the kidneys to increase sodium and water reabsorption in an attempt to dilute the concentration of urea and creatinine and maintain circulating volume. The urine becomes more concentrated. As the stimulating factor improves, the kidneys resume normal urea excretion. The protective mechanisms described above will cease to function within a short space of time and circulatory failure will ensue. If treatment is not instigated, a metabolic acidosis, circulatory failure or progressive shock will occur in a short space of time.

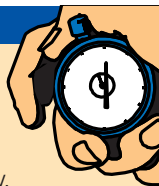
## Implications for practice

The practical application of the factors that control fluid, sodium and potassium homeostasis are fluid regimens and hyperglycaemia, hyperlipidaemia and proteinaemia. Prescribed fluid regimens need to be carefully thought out, to prevent severe fluid and electrolyte disturbances. In addition, if they are not administered accurately and documented over the stated period, fluid overload and oedema might ensue.

Hyponatraemia develops when serum sodium concentration decreases to below 135mEq/l. Hyponatraemia is caused by a sodium deficit or water excess, leading to an intracellular overhydration. This can occur in fluid regimens where fluid loss is replaced with excess intravenous 5% dextrose in water. When the body is functioning normally, it is almost impossible to produce an excess of fluid by administering 5% dextrose. In situations when large amounts of fluid are given, the haemodilution (hypo-osmolality) that occurs will stimulate osmoreceptors. There will be a reduction in ADH release, resulting in an increase in urinary output, and excess fluid will be lost.

**TIME OUT 3**

Jack has been admitted for dehydration. He lives alone and sometimes forgets to drink. A urine test reveals a specific gravity (SG) of 1035. He is thirsty, gaunt and his skin is frail and thin. He is administered 5% dextrose and encouraged to drink clear fluids, but is given no electrolytes. The dextrose restores Jack's urine SG to normal, his urine output is good, and the nursing staff assume he is no longer dehydrated. However, Jack's fluid regimen continues to prescribe 5% dextrose.



- What might be the result of only giving Jack 5% dextrose for dehydration?
- What normal homeostatic mechanisms might be stimulated in this instance?
- What extra fluid might be required?

Fluid regimens should include 0.9% saline, but if so much fluid is administered haemodilution could result, causing complications. It might be necessary to administer a blood transfusion if haemoglobin becomes so diluted. Other causes of sodium deficits or water excess are:

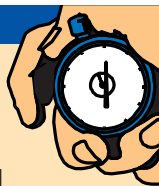
- Inadequate sodium intake or diuretic therapy.
- Excessive sweating, stimulating thirst and intake of a lot of water, which dilutes ECF sodium.
- Vomiting, diarrhoea, gastrointestinal suctioning or burns.

When there is a sodium deficit, the ECF exhibits hypo-osmolality and water moves into the cell, where the electrolyte concentration is greater. The plasma volume then decreases, leading to symptoms of hypovolaemia. When there is an excess of water, the ICF and the ECF volumes increase, causing symptoms of hypervolaemia or fluid overload.

Hypernatraemia is defined as a serum sodium level greater than 145mEq/l. Excessive serum sodium can be caused by an acute gain in sodium or a loss of water. It is always associated with hyperosmolality. The main cause of high sodium levels occurs with inappropriate administration of saline solutions (as sodium bicarbonate for treatment of acidosis during cardiac arrest, or sodium chloride).

**TIME OUT 4**

Olivia has had a cardiac arrest. Paramedics arrived promptly; she was successfully resuscitated and given 0.9% saline to maintain circulating volume and blood pressure. On admission to A&E, she has slight difficulty in breathing and is very thirsty.



- Why is fluid therapy necessary for Olivia?
- What are the homeostatic mechanisms that have been stimulated to cause Olivia's thirst?

Even though 0.9% saline is often classified as an isotonic solution (Rooney 1995), it does contain a higher concentration of sodium (154mEq/l) than plasma (140mEq/l). It should not be used for maintenance as it contains too much sodium for such a regimen, but must be alternated in some way with 5% dextrose (Rooney 1995). This is because sodium is the principal extracellular electrolyte, and the solution when infused is distributed only in the ECF, so a primary increase in extracellular sodium causes an osmotic attraction of water and intracellular dehydration.

In addition, large infusions of 0.9% saline might, because of the high concentration of sodium, increase blood osmolality so resulting in a further load onto the circulation due to the response of compensatory mechanisms. These processes cause movement of water to the ECF and signs of fluid overload might be evident, with weight gain, pallor, breathlessness, convulsions and pulmonary oedema being the most obvious.

Although 0.9% saline is much better than 5% dextrose (which causes water retention and dilutional hyponatraemia if infused in excess) for resuscitation and operative purposes (that is, correction of hypovolaemia or hypotension), it is still not ideal. Other causes of hypernatraemia are:

- Water depletion, excess sodium and water loss.
- General fever or that caused by respiratory infections that increase respiratory rate, enhance water loss from the lungs and sweating.
- Diabetes insipidus.
- Diabetes mellitus.
- Polyurea.
- Diarrhoea which can cause water loss in relation to sodium.
- Insufficient water intake, particularly in patients who are comatose, confused or immobilised.
- Oversecretion of aldosterone as in primary hyperaldosteronism, or Cushing's syndrome, caused by excess secretion of adrenocorticotrophic hormone (ACTH).

**Fluid overload** When excess fluid is administered to patients with acute renal failure, heart conditions, hypertension, cirrhosis or other conditions, this can precipitate a water excess, and there is a risk of fluid overload. It is most commonly the result of an excessive administration of a combination of intravenous fluids, culminating in an overload of salt and water.

If fluid administration is allowed to continue unchecked, the neck veins might distend, with the increased blood pressure leading to oedema formation. If the circulating volume is great enough, pulmonary oedema and heart failure can develop. If cardiac output is allowed to fall, a

**Answers to Time Out 1 and 2****TIME OUT 1. David**

- David's potassium level is low because he has a respiratory alkalosis due to his asthma, and ECF potassium will exchange with ICF hydrogen (hypokalaemia) and correct the alkalosis by reducing the pH of the ECF
- Potassium moves subscript into the cell in exchange for hydrogen to maintain plasma acid-base balance
- During a respiratory alkalosis, the ECF pH increases, which affects the oxygen dissociation curve. Less oxygen is released to the cells

**TIME OUT 2. Arthur**

- The increased sodium and potassium contained in the blood are having serious effects on Arthur due to his heart condition
- The increased sodium level would initiate homeostatic control mechanisms via the osmoreceptors to enhance sodium and water reabsorption, and if cardiac output dropped, the renin-angiotensin-aldosterone system would further promote sodium and water reabsorption to maintain cardiac output. This can promote fluid overload. In addition, baroreceptors cause vasoconstriction, increasing blood pressure and the risk of fluid overload and heart failure
- Breathlessness is due to left ventricular failure increasing the pressure through the lungs, causing cell damage and third-space fluid shift, which leads to pulmonary oedema
- To prevent fluid overload and/or cardiac failure, diuretic therapy (frusemide) is often given following each unit of blood or every consecutive unit

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## Answers to Time Out 3 and 4

### TIME OUT 3. Jack

- Jack has a sodium deficit, which leads to intracellular overhydration and haemodilution
- In this situation, the reduction in sodium in relation to fluid in the ECF stimulates osmoreceptors, reducing the release of ADH from the pituitary gland. An increase in urinary output will result and the excess fluid will be lost
- Fluid regimen, although prescribed by doctors, should alternate with 0.9% saline (overload) and 5% dextrose (haemodilution) to maintain effective fluid and electrolyte balance

### TIME OUT 4. Olivia

- Fluid therapy is necessary to counterbalance the third-space fluid shift that occurs due to cell damage and injury and to prevent the relative hypovolaemia that might occur
- A higher concentration of sodium (154mEq/l) is contained in 0.9% saline than in plasma (140mEq/l). When the solution is infused it is distributed only in the ECF, causing a primary increase in extracellular sodium which causes an osmotic attraction of water and intracellular dehydration. In addition, because of the high concentration of sodium in the ECF, there is an increase in blood osmolality (stimulating osmoreceptors, and an increase in ADH) resulting in a further load on the circulation

reduction in renal blood flow will stimulate the renin-angiotensin-aldosterone mechanism, resulting in increased reabsorption of water and salt.

In addition, if circulatory stasis occurs, osmoreceptors will stimulate the release of ADH, reabsorbing more water and further reducing urine output. The renin-angiotensin-aldosterone and osmoreceptor mechanisms can lead to circulatory collapse, whereby the left side of the heart becomes dysfunctional with the consequence of the heart becoming unable to pump the blood around the body (Edwards 2000). These mechanisms, despite attempting to protect the body, actually increase circulating volume and exacerbate the symptoms of fluid overload.

**Oedema** Oedema is the accumulation of fluid within interstitial spaces. This fluid is not available for metabolic processes. A state of dehydration can develop as a result of the sequestering of the oedematous fluid and in some conditions oedema and dehydration might be observed. Oedema is a problem of fluid distribution and does not necessarily indicate an excess of fluid. The most common mechanisms include:

- Increased hydrostatic pressure (hypertension, LVF).
- A lack of plasma electrolytes (malnutrition).
- Increased capillary membrane permeability (trauma, injury, surgery).
- Lymphatic obstruction (cancer).


The accumulation of fluid increases the distance required for nutrients and waste to move between capillaries and tissues, and an increase in tissue pressure might diminish capillary blood flow. Wounds will heal more slowly, and risks of infection and formation of pressure sores increase. Oedema of specific organs, such as the brain, lung, or larynx, can be life-threatening.

**Hyperglycaemia, hyperlipidaemia, hyperproteinaemia** Hyperglycaemia increases ECF osmolality and attracts water from the cells. Increases in plasma lipids and proteins displace water volume and decrease sodium concentration. The osmotic fluid shift to the ECF in turn dilutes the concentration of sodium and other electrolytes. These conditions cause hypertonicity due to increased amounts of impermeable solutes causing a shift of water from the ICF to the ECF to provide osmotic equilibration, thus diluting the ECF sodium. Such resultant hyponatraemia is often associated with a decreased measured osmolality.

## Conclusion

A complex interplay of physiological control systems maintains fluid, sodium and potassium homeostasis. When the activity of the physiological systems are disrupted, or when homeostatic

mechanisms can no longer maintain intracellular, extracellular or interstitial fluid, sodium and potassium disequilibrium ensues. The nurse's role in relation to assessment and monitoring of fluid and electrolyte balance is well established. A number of patients that require fluid regimens have conditions or require other treatments that interfere with or affect their equilibrium of fluid, sodium and potassium balance and/or stimulate homeostatic control mechanisms. Therefore, nurses need to understand and observe patients' fluid sodium and potassium levels.

The extent of this nursing contribution to multidisciplinary care is so far virtually unexplored. Future practice with respect to fluid and electrolyte balance might extend beyond assessment, monitoring and securing vascular access for intravenous fluid administration, towards nurse prescription of intravenous fluids .

## TIME OUT 5

Now that you have completed the article, you might like to think about writing a practice profile. Guidelines to help you write and submit a profile are outlined on page 44.



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