Regulation of fluid and electrolyte balance

Wael Maher

Ross Macnab

Abstract

Adequate tissue perfusion and cellular function is dependent on the maintenance of effective circulatory volume and serum osmolality, respectively. As sodium is the principal extracellular cation with the inability to pass freely across the cellular membrane, it therefore has the greatest effect on extracellular fluid osmolality. The extracellular fluid (ECF) volume can increase or decrease independent of the surrounding osmolality, indicating that control of plasma osmolality and volume occur through distinct physiological processes. Disorders in sodium balance with consequent effect on osmolality come about mainly due to disturbances in water homeostasis rather than an abnormality of sodium intake or excretion.

Keywords Aldosterone; antidiuretic hormone; extracellular fluid; hydrostatic pressure; oncotic pressure; osmolality; osmoreceptors; sodium; total body water

Royal College of Anaesthetists CPD Matrix: 1A01, 1A02, 1A03, 2A05

Learning objectives

After reading this article, you should be able to:

- define osmolality
- calculate plasma osmolality
- explain how the renin angiotensin aldosterone system helps regulate the extracellular volume

Distribution of body water

Under normal physiological conditions 60% of the body weight is constituted of water, with a total body water (TBW) of approximately 42 Litres in a 70 kg adult. This volume is distributed in three main compartments; the intracellular compartment accounts for two-thirds of TBW (28 litres) and the remaining one-third in the extracellular compartment, which is further divided into the intravascular (3 litres) and interstitial compartment's (11 litres). A variation in TBW exists between different ages and gender; in neonates, up to 70–80% of body weight is water and due to the higher content of body fat in women, water represents 50% of body weight.

Osmolality, tonicity and osmolar gap

Osmolality is defined as the number of osmotically active solutes (impermeable to a semi-permeable membrane) per kilogram of solvent. Therefore, a compartment with an increased osmolality (hypertonic) will draw free water from a compartment with low osmolality (hypotonic). Serum osmolality is tightly regulated within a narrow value ranging from 285 to 290 mOsm/kg. Sodium level is the main contributing factor in determining osmolality hence affecting the movement of water between intracellular and extracellular compartments. As sodium can move freely across the capillary endothelium, sodium level does not affect water movement between the intravascular and interstitial compartments.

Osmolality can be calculated by the following equation

$$\text{Plasma Osmolality} = 2\ (\text{Na}) + \text{Urea} + \text{Glucose}\ (\text{mmol/L})$$

The effect of glucose on plasma osmolality occurs in severe hyperglycaemia and as urea can freely cross cell membranes it can equilibrate between compartments. Therefore, effective osmolality or tonicity as mentioned earlier is mainly dependent on sodium.

$$\text{Effective Plasma Osmolality (Tonicity)} = 2\ (\text{Plasma Na})\ \text{in mmol/L}$$

The difference between osmolality and tonicity is that osmolality takes into account total solute concentration whereas tonicity only refers to solutes that cannot cross semi-permeable membranes and consequently exert an osmotic force across the membrane. Osmolality can be measured by utilizing one of the colligative properties of a solution (depression of freezing point), which varies proportionally with solute concentration. The osmolar gap is the difference between measured and calculated osmolality and it is normally less than 10 mOsm/kg, its increase indicates the presence of solutes that are not routinely measured (e.g. methanol, mannitol).

Osmosis and osmotic pressure

Osmosis describes the passive movement of water down a concentration gradient across a semi-permeable membrane separating two fluids with different osmolality's. This movement continues until an equilibrium of the two fluids is reached. This is best explained by the shrinkage and swelling of red blood cells when they are exposed to a hypertonic and hypotonic fluid, respectively. From a clinical aspect, the administration of intravenous mannitol or hypertonic saline, which cause an increase in the effective osmolality (tonicity) of plasma, is used for the treatment of cerebral oedema by decreasing cellular water content and consequently cellular swelling. The force that opposes the movement of water between to solutions of different osmolality's is known as the osmotic pressure. Therefore the greater the movement of water, which is dependent on the osmolar gradient between the two solutions, the greater the osmotic pressure.

Wael Maher *MBBCh MSC EDAIC MD FRCA is a Clinical Fellow in Anaesthesia and Intensive Care Medicine at Manchester University Hospitals Foundation Trust, UK. Conflicts of interest: none declared.*

Ross Macnab *MB ChB FRCA is a Consultant Anaesthetist at Manchester Royal Infirmary, UK. Conflicts of interest: none declared.*

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Hydrostatic and oncotic pressures

As discussed previously, the movement of water between the intracellular and extracellular compartments is governed by the sodium concentration due to its inability to pass freely across the cell membrane. On the other hand, capillary endothelium is not an effective barrier to sodium and therefore does not influence water shifts between the intravascular and interstitial compartments. Fluid shifts in the extracellular compartment are therefore controlled by the gradient between hydrostatic and oncotic pressures. Basically, hydrostatic pressure is the force that drives fluid out of the capillaries and is dependent on the pressure within them, whereas oncotic pressure, which is dependent on plasma proteins (mainly albumin), acts to maintain or draw fluid into the intravascular compartment. An example would be to compare between the intravenous infusion of equal volumes of an isotonic crystalloid and an albumin-containing solution. With the isotonic fluid there will be an initial increase in intravascular volume which is actually short lived as the infused volume will distribute between the intravascular and interstitial spaces according to the size of each compartment. Conversely, the infusion of the albumin solution will lead to a sustained rise in intravascular volume due to the lack of permeability to capillary endothelium under normal physiologic conditions. Consequently, the oncotic pressure of plasma increases resulting in a decrease in fluid leaving the circulation and promoting fluid absorption from the interstitial space.

Volume regulation

Maintenance of adequate extracellular fluid volume is influenced by control of water and sodium balance. Which of the previously mentioned will be primarily responsible for the regulation of extracellular volume (ECV) and how the body will sense changes in volume will be the points of discussion that follow. Consider the effects of the addition of 1 litre of free water to the extracellular compartment. Water will distribute between the extracellular and intracellular compartments according to the volume of each, bringing about a minimal change in the ECV as it only represents one-third of the TBW content. The addition of sodium to free water will prevent the movement of water from the extracellular compartment and therefore play the main role in extracellular volume regulation.

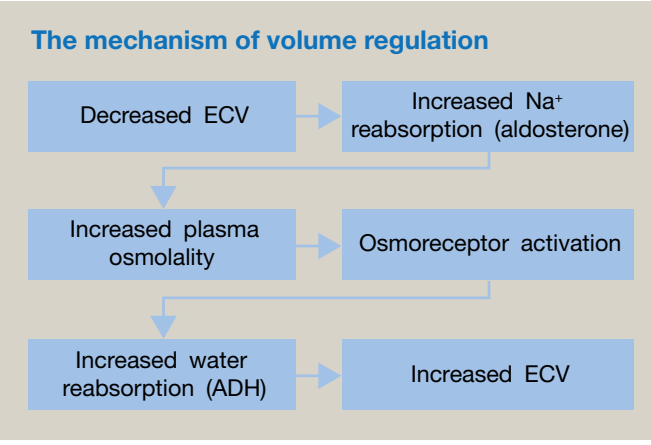

Figure 1

The whole process of volume regulation can be described as a two step mechanism (Figure 1). Initially sodium levels change, bringing about a change in serum osmolality. This change is detected by very sensitive osmoreceptors that influence the secretion of anti-diuretic hormone (ADH) regulating water reabsorption and thirst sensation. To clarify, when the osmoreceptors are exposed to hypertonic plasma they respond by increasing the release of ADH and stimulate thirst. As a result, increased water reabsorption from the kidneys with the consequent production of hyperosmolar urine and increased water intake will result in the increase in ECV and return plasma osmolality to its normal range.

ECV sensing

The human body is incapable of measuring volume and depends on pressure, as it will vary proportionally with volume under normal conditions. Pressure sensors are located in multiple sites in the circulatory system and are known as baroreceptors which respond to stretch. In the arterial circulation, high pressure baroreceptors are located in the aortic arch and carotid sinus, whereas low pressure baroreceptors are found in the walls of great veins and the atria. Their rate of firing changes with the degree of stretch, which is dependent on volume status. The afferent limb of the reflex relay their impulses into the medulla and ECV changes are brought about by the alteration of sodium reabsorption and excretion via the renin–angiotensin–aldosterone system (RAAS), pressure natriuresis, atrial natriuretic peptide and the activation or inhibition of the sympathetic nervous system.

Renin–angiotensin–aldosterone system

It is considered the principal neuro-hormonal system that is responsible for long-term regulation of ECV and arterial blood pressure. Multiple hormones (Figure 2) are involved in the cascade, starting with renin and ending in the release of aldosterone. Renin is an enzyme synthesized and secreted from the juxtaglomerular cells that are located in afferent arteriolar walls.

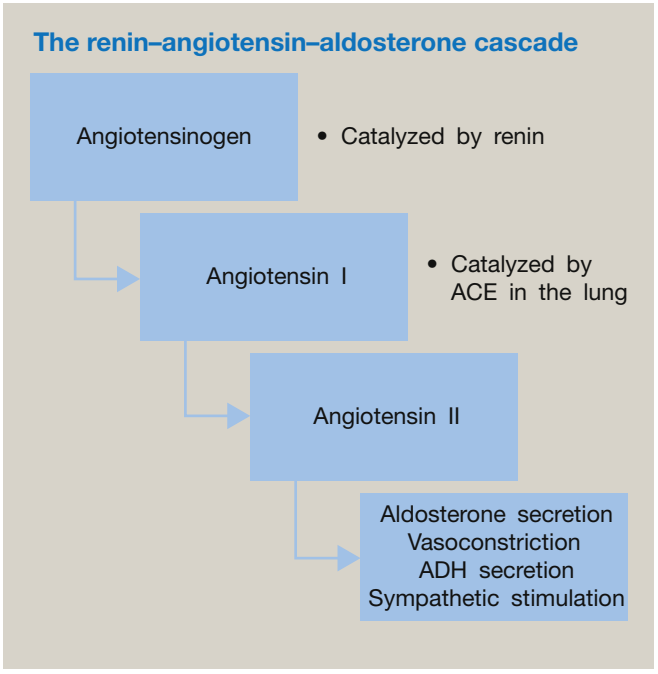

Figure 2

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