Sodium is the principle extracellular cation. It is present in the ECF at a concentration of about 140 mmol/L. There is small increase in amount of sodium on the plasma side compared to the ISF of about 6-7 mmol due to the Gibbs-Donnan effect but the measured levels are usually very similar because the plasma solid effect reduces the measured amount in the PV by 7%. In 7% the sodium concentration is much lower due to the Na-KATPase pump, and the impermeability of the cell membrane to sodium. The distribution of sodium across the plasma membrane is 90% ECF and 10% ICF. The distribution of sodium in the body is ECF 45% and the total amount is approximately 60mmol/Kg. Sodium principally maintains serum osmolality and ECF volume. Sodium plays a crucial role in the generation of the cell membrane action potential. Disorders of sodium concentration (hyponatraemia or hypernatraemia) usually result in a relative deficit or excess of water respectively. Serum Na+ concentration and total body Na+ are closely regulated by renal functions. Sodium is easily filtered by the glomeruli of the kidneys, and most of it reabsorbed by the renal tubules. Several hormones, including aldosterone, ANP, and vasopressin control the reabsorption of sodium and water to maintain the amount of total body Na+. Aldosterone is responsible for renal Na+ reabsorption in exchange for K+ and H+ in the distal convoluted tubules and the collecting ducts of the kidneys. At physiological concentrations it tends to decrease plasma volume. Normally, changes in serum Na+ and osmolality lead to an appropriate change in vasopressin release by the posterior pituitary which in turn can vary renal free water excretion (urine osmolality 50-1400) in order to maintain Na+ in the normal range. Daily intake of sodium is approximately 10.5g (457mmol) since at rest 11 mmol is lost in faeces and 11 in sweat (nb this is highly variable) the kidneys must excrete the remaining 435 mmol (10g). The end result of these physiological processes is that in situations where there is Na+ loss, for example diurexia or excessive sweating, renal Na+ conservation is high and urinary Na+ concentration falls to very low levels. However when Na+ intake is excessive, decreased tubular reabsorption leads to the loss of increased sodium in the urine.

Potassium is the major intracellular cation and normally there is an IC concentration of 150mmol/L which is about 30 times that of the ECF (normal range 3.5-5). The total amount of potassium in the body is about 40-45 mmol/kg. The distribution of K+ in the body is IC = 90%, ECF = 5% and Bone = 5%. It is often said that potassium is 98% ICF and 2% ECF, and whilst this is factually incorrect it is useful from a clinical perspective because the bound potassium in the bone is not physiologically mobile therefore often ignored. Serum potassium is a function of two variables, the total body potassium and the distribution between ICF and ECF. There are numerous major functions of potassium in the body including cell volume regulation (as the major intracellular tonicity component and its role in the Na,KATPase pump), intracellular pH regulation, DNA and protein synthesis, growth, enzyme functions, resting membrane potential, cardiac and neuromuscular activity. Total body K+ and serum K+ are closely regulated by the renal tubules, so that potassium excretion is usually equal to daily potassium intake (1.5-9g per day). There are several hormones, including aldosterone, adrenaline and insulin that effects on K+ concentration. Aldosterone facilitates sodium reabsorption in exchange for K+ and corrects the intravascular fluid deficit. The plasma potassium concentration is largely determined by the balance between renal and extrarenal losses. The renal tubules reabsorb about 65-75% of filtered potassium and secrete 10-20% due to the activity of the Na,K-ATPase pump. The remaining potassium is excreted into the urinary tract. The plasma potassium concentration is thus controlled by the balance between renal potassium reabsorption and secretion. Of the normal serum, K+ (normal range 3.5-5.0mmol/L) free 'ionised' K+ represents approximately 50% this amount and is the physiologically active component. A variety of arrhythmias may be seen in patients with hypokalemia. These include premature atrial and ventricular beats, sinus bradycardia, paroxysmal atrial or junctional tachycardia, atrioventricular block, and ventricular tachycardia or fibrillation. Hypokalemia produces characteristic changes on the ECG although they are not seen in all patients. There is depression of the ST segment, decrease in the amplitude of the T wave, and an increase in the amplitude of U waves which occur at the end of the T wave. Hypokalaemia may also lead to renal abnormalities and glauose intolerance (through the decreased excretion of insulin).

Calcium is found predominantly in the bone (99%) and the ICF (1%). Serum calcium is approximately 40% protein bound (mostly albumin), 10% chelated to serum anions and 50% in free ionised form. It is the latter form that has a physiological effect as the free calcium is the functional biologically active component of calcium (Ca2+) in the ICF at a concentration of approximately 50 mmol/L. Ca2+ performs many important functions including neuromuscular transmission, excitation-contraction coupling, release of hormones and neurotransmitters, enzyme activation, blood coagulation, and bone structure. It is also an important intracellular second messenger. Calcium is regulated by two primary mechanisms, parathormone (PTH) and 1,25 dihydroxy-vitamin D (activated vitamin D). Both of these are secreted in response to a decrease in serum ionised Ca2+ and stimulate Ca2+ release from bone, reabsorption from the renal tubules and absorption from the intestines. They are able to maintain a tight calcium control even in the setting of decreased intake. Patients with mild hypercalcaemia (calcium 3 [3.5]mmol/L) may be asymptomatic, or they may report nonspecific symptoms such as constipation, fatigue, and depression. A serum calcium of 3 to 3.5 mmol/L may be well-tolerated chronically, while an acute rise to these concentrations may cause marked symptoms, including polyuria, polydipsia, dehydration, anorexia, nausea, muscle weakness, and changes in sensorium. As calcium is an ionothe the cardiovascular symptoms/signs are shortening of the QT interval, bradycardia, hypertension. Hypocalcaemia may be associated with a spectrum of clinical manifestations), ranging from few if any symptoms if the hypocalcaemia is mild, to life-threatening seizures, refractory heart failure or tetry (initially paroxysmal tingling, then sensorium changes and cramps progressing to laryngospasm) if it is severe. In addition to severity, the rate of development of hypocalcaemia and chronicity determine the clinical manifestations.

Magnesium is the fourth most common cation in the body (99%) and the ICF (1%). Magnesium is approximately 40% protein bound (mostly albumin), 10% chelated to serum anions and 50% in free ionised form. The latter form is the functional biologically active component. Magnesium is regulated by renal excretion. Free Mg2+ is an essential co-factor for ATP requiring enzymes such as the cell membrane Na,K-ATPase pump and many enzymes involved in excitation-contraction coupling. Many processes are dependent on magnesium (Mg2+) including the production and functioning of ATP (to which it is chelated) and the biosynthesis of DNA and RNA. It has an essential role in the regulation of most cellular functions. It acts as a natural calcium (Ca2+) antagonist. High extracellular Mg2+ leads to an increase in intracellular Mg2+, which in turn inhibits Ca2+ influx through Ca2+ channels. It is this non-competitive inhibition of Ca2+ antagonistic effect that appears to mediate many of its effects. It also competes with calcium for binding sites on sarcoplasmic reticulum thereby inhibiting its release. High concentrations inhibit both the pre-synaptic release of ACh and as well as post-junctional potentials. Mg2+ also has an antiadrenergic action: release at all synaptic junctions is decreased, and it inhibits the release of catecholamines. Central and peripheral nervous systems : Magnesium penetrates the blood–brain barrier poorly, but it nevertheless depresses neuronal excitability. It inhibits the release of all neurotransmitters at all synapses in the brain. Cardiomyocytes: It mediates a reduction of vascular tone via direct vasodilatation. It also causes sympathetic block and the inhibition of catecholamine release. Magnesium decreases cardiac conduction and diminishes myocardial contractile force. This intrinsic slowing is opposed partly by vagolytic action. Respiratory: Magnesium has no effect on heart block and finally at greater than 12 asystole.

Phosphate is found mostly inside the cell. Bone accounts for 85%. The normal serum phosphate level ranges from 0.8 to 1.3 mmol/L in adults. Serum phosphate is regulated by renal excretion. It follows that the most common cause of hyperphosphataemia is renal failure (others include tumour lysis syndrome, rhabdomyolysis and lactic acidosis). Phosphate plays a vital role in oxidative phosphorylation of carbohydrate, fat and protein metabolism. Phosphate is a structural component of nucleic acids, phospholipids and the cell membrane. It is also essential for intracellular second messenger systems including cAMP and IP3. As part of 2,3 DPG it assists in the offloading of oxygen from HB to tissue. Hyperphosphotaemia is symptomatic when the increased binding of calcium leads to hypocalcaemia (seizures, heart failure and eventually tetany). Hypophosphataemia is common in intensive care patients and may cause muscle weakness. It is often used as a marker of refedding syndrome.