1

Shock

Definition.—Shock may be defined as a state of depression of the vital functions of the body due to inadequate tissue perfusion of the vital organs, resulting from insufficient microcirculation.

Basic Terminology: Microcirculation comprises that part of the vascular network which extends from the smaller arterioles to the venules, i.e. the terminal arterioles, capillaries and venules.

The terminal arterioles have thick muscle coat, the tone of which is controlled by the sympathetic nerves as well as some hormones. Thus, their calibre is altered as per need and they control the amount of blood flow through the capillaries. They make the peripheral vascular resistance, hence they are called *resistance arterioles.*

The venules are also lined by smooth muscles, though thin. The entire venous network, starting from the venules up to the major veins of the trunk and the limbs, comprises what is called the *capacitance veins.* The tone of the lining muscles of all the veins is also controlled by the sympathetic fibres. Their contraction evacuates the blood, reserved there in large *quantities* (*venous reservoir*) into the active circulation.

Types of Shock: Insufficiency of the microcirculation may be either due to inability of the central pump, i.e. the heart, to send blood at sufficient pressure to reach the microcirculation (*cardiogenic shock*) or because of a shortfall in the volume of blood available to the heart to be pumped out.

This latter condition may be due to either a reduction in the *body's total* blood volume (*haematogenic shock*) or a diminution of the actual *circulating* blood volume as a result of pooling of blood in the peripheral vessels (*vasogenic shock*). Basically, therefore, shock may be classified into three categories.

1. Haematogenic (*Syn* hypovolaemic, oligaemic)
2. Vasogenic
3. Cardiogenic.

The above is only a working classification and indicates the initial cause. Superimposition often occurs in any advanced case of shock. Septic shock is, from its onset, a combination of vasogenic and haematogenic shock.

Causes of Haematogenic (Hypovolaemic, Oligaemic) Shock

1. Haemorrhagic, i.e. Loss of Blood

a. Blood lost from injured part—external or internal.

b. Blood lost into injured part—major fractures, big loops of strangulated gut, acute pancreatitis.

2. Non-haemorrhagic

a. Loss of plasma and fluid—burns

b. Loss of fluid:

i. From and into the intestine—vomiting, diarrhoea, gut obstruction.

ii. Into the peritoneal cavity—peritonitis, acute pancreatitis.

Causes of Septic Shock.—This is a sequela to severe systemic sepsis. The common sources of infection are the genito-urinary tract, respiratory tract, intra-abdominal sepsis, burns and indwelling monitoring catheters.

Causes of Vasogenic Shock.—The essential cause is pooling of blood in the terminal arterioles and capacitance veins, leading to gross reduction in the actively circulating blood volume:

1. Neurogenic shock.—There is loss of sympathetic control of the peripheral vasculature:
   a. Spinal cord injury.
   b. Spinal anaesthesia.
2. Psychogenic shock.—Also caused by loss of sympathetic control:
   a. Sudden fright, apprehension, grief.
   b. Acute pain, e.g. blow to the testis.
3. Vasovagal shock.—Pooling of blood in the large venous reservoirs (limb muscles) and dilated splanchnic arterioles.
4. Anaphylactic shock.—Massive peripheral vasodilatation caused by release of histamine and slow release anaphylactic substances (SRSA). The common agents are shellfish, penicillin, anaesthetic agents, equine serum.

Causes of Cardiogenic Shock

1. Intrinsic cardiogenic shock.—The myocardial contractility is grossly impaired, e.g. myocardial infarction, myocarditis, cardiac arrhythmias.
2. Cardiac compressive shock.—There is compression of the cardiac chambers and/or great veins, e.g. cardiac tamponade tension pneumothorax.
3. Cardiac obstructive shock.—There is obstruction either in the pulmonary or the systemic circulation:
   a. Pulmonary embolism, pulmonary vascular disease.
   b. Mechanical obstruction to aorta, systemic arteriolar constriction.

HYPOVOLAEMIC (OLIGAEMIC) SHOCK

Pathology.—With the onset of hypovolaemia, the body's compensatory mechanisms are set into action:

1. The first aim is to divert blood, as much as possible, from the relatively unimportant areas to the vital organs, of which the brain and the heart are the most important. This is primarily effected by stimulation of the sympathetic

nervous system and die adrenal medulla discharging adrenaline and noradrenaline (*adrenergic discharge*) which causes:

a. Contraction of the systemic venules and small veins (venous reservoir), pumping out the large volume of blood reserved therein, into the circulation.
b. Constriction of the arterioles, firstly, those of the skin, subcutaneous fat and skeletal muscles, thereafter the arterioles of the splanchnic organs including the liver and, finally, the arterioles of the kidneys, thereby maintaining blood flow to the heart and the brain. Angiotensin and vasopressin, released from the ischaemic kidneys, add to the constriction of the arterioles. The cerebral and cardiac arterioles are spared of this constriction because there are autoregulating mechanisms to conserve cerebral and cardiac blood flow.

2. Simultaneously, there are natural attempts to reinforce the total blood volume (intravascular fluid):
   a. There is movement of fluid from the extracellular to the intravascular space through the capillary wall (capillary refill). This is effected by adrenergic discharge, vasopressin and angiotensin.
   b. There is also shift of intracellular .fluid, via the extracellular space, into the intravascular space. Release of epinephrine, cortisol and glucagon,and inhibition of insulin secretion lead to high concentration of glucose in the extracellular fluid, causing its hyperosmolarity.

      This draws water out of the cells into the extracellular space. The interstitial pressure rises, forcing water (with sodium and chloride) into the vascular space.
3. There are attempts to preserve whatever fluid is there in the vascular space by die kidney *(renal conservation of blood volume).* This is effected by:
   a. Diminution of glomerular filtration rate.
   b. Increased reabsorption of water (with sodium and chloride) by the distal renal tubules. This is effected by aldosterone (of adrenal cortex) as well as by angiotensin produced in the kidney when its blood flow is reduced. The chief stimulator to aldosterone secretion is also angiotensin itself.
4. The cardiac output is mechanically raised by traction on the great vessels and cardiac chambers. This is caused by the spontaneous hyperventilation due to metabolic acidosis resultant upon tissue anoxia.
5. There is an associated tachycardia in an attempt to, send blood more frequently to the tissues. This is induced by the adrenergic discharge as well as by depression of the vagal centre, caused by fall of pressure in the great vessels (Marey's reflex).

However, if the *hypovolaemia progresses,* the compensatory mechanisms gradually fail. Blood supply to the vital organs are progressively reduced and ultimately the organs start failing (*organ failure of shock*). To start with, the gastrointestinal system, pancreas and liver and, thereafter, the kidneys fail. Finally, there is a failure of the brain and the heart—the features of the former more manifest in the young and

those of the latter in the elderly. The final event is cardiac arrest. Pulmonary failure, in the absence of chest injury, is a rate event in hypovolaemic shock (of septic shock).

Clinical Features: The features correlate with the structures and organs whose blood supply has to be curtailed. This starts with the least essential structures and ends with the most essentials:

i. Firstly the skin.
ii. Then the kidneys and other viscera, including the liver.
iii. Finally the heart and the brain.

The findings depend upon the degree of depletion of the blood volume:

1. In *mild shock* (less than 20 per cent volume depletion), features of adrenergic discharge to the skin are manifest as follows:
   a. The extremities, especially the feet, are pale and cold, and may be moist with sticky sweat (clammy).
   b. The subcutaneous veins collapse (making insertion of infusion needle difficult).
   c. The patient feels cold and complains of thirst (a constant feature).
   d. The temperature is subnormal, including the 'core' temperature (e.g. midoesophageal or tympanic membrane).

   The pulse and the blood pressure show little changes, especially in the supine position.
2. In *moderate shock* (20 to 40 per cent depletion) in addition to the skin features, the urine output is low (less than 0.5 ml/kg body wt/hour).

   The pulse and the blood pressure may still be near normal, particularly in supine position.
3. In *severe shock* (more than 40 per cent depletion), there is progressive rise of pulse rate and fall of blood pressure, and features of cerebral and cardiac ischaemia set in:
   a. Restlessness and anxiousness, gradually changing to apathy and exhaustion.
   b. The ECG shows depressed S-T segment and presence of Q wave. As the ischaemia progresses, there are arrhythmias, ventricular fibrillation and, finally cardiac arrest.

Treatment.—The principles of treatment for both haemorrhagic and non-haemorrhagic shock are basically the same, having two essential components, both equally important and demanding simultaneous attention-treatment of the cause and replacement of the depleted blood volume. The replacement must be equivalent to the loss, both quantitatively and qualitatively, i.e. blood for haemorrhage, fluids and plasma expanders for burns, and electrolyte-containing fluids for gastro-intestinal loss. The treatment of haemorrhagic shock, the commonest type encountered by the surgeon, is detailed as follows:

A. Arrest of haemorrhage.—The source of bleeding is quickly identified and steps taken for immediate control:

1. External bleeding should be primarily tamponaded by compression; surgical procedure may then be carried out.
2. In traumatic internal haemorrhage the source is to be surgically explored and bleeding controlled.
3. Bleeding from the gastrointestinal tract should have the source identified and treated with usual measures, e.g. decompression of the stomach by nasogastric suction in gastroduodenal ulcer haemorrhage.

B. Replacement of blood volume.—Intravenous infusion is started immediately. An amount approximating the amount of loss has to be infused and this must be done as quickly as possible without burdening the heart.

1. Whole blood is the best replacement for blood loss because the RBCs apart from their important physiological functions, serve as the biggest molecules in the fluid, providing high osmolarity that prevents extravascular escape of the transfused fluid.
2. An effective initial fluid regime, till type specific blood is available, is infusion of a non-sugar crystalloid solution. Sugar is avoided because it induces diuresis that reduces the blood volume. Lactated Ringer's solution is the best but acetated Ringer's solution or isotonic saline supplemented with sodium bicarbonate may be used instead. The lactate, acetate and bicarbonate are necessary to combat metabolic acidosis resultant upon tissue anoxia. However, the quantity of the transfused crystalloid fluid must be large (four times the estimated loss) and the rate of transfusion very rapid because three-fourths of the transfused fluid shall escape out of the capillaries into the interstitial tissues almost immediately (as per the normal intravascular: interstitial fluid distribution 1:3). This initial fluid therapy also serves as a *therapeutic trial.* It is often found that the blood pressure returns to normal and keep stable. If it happens so:
   a. the pre-existing blood loss is not severe, and
   b. the haemorrhage is not continuing.

   In these cases transfusion of whole blood may be avoided or minimised, thereby preventing depletion of blood bank and complications of blood transfusion.

   On the other hand, if the blood loss has been severe and/or the haemorrhage is still continuing, the rise of blood pressure is only transient. Blood transfusion has to be instituted. In these cases, if the patient can be operated upon promptly, transfusion of blood is withheld till the bleeding is surgically controlled, thereby avoiding drainage of the transfused blood.
3. In the absence of blood or as its part supplement, colloid solutions may be used. They are preferable to crystalloid fluids in that, by virtue of their high osmolarity, they prevent extravascular escape of the transfused fluid at least for several hours. However, in advanced cases of shock, there is a generalised capillary endothelial damage and the colloids may come out into the interstitial tissues, e.g. lungs, and this is dangerous.

The colloid fluids that may be transfused are:

a. Plasma, available as fresh frozen plasma (FFP).
b. Plasma substitutes:
   i. Natural: 4.5 per cent human albumin solution.
   ii. Synthetic: dextran, gelatin, hydroxyethyl starch, etc. (these have molecular weight more than 30,000).

Plasma transfusion is least preferred because its entry into the interstitial space causes serious problems and also because of the high risk of transmitting diseases like hepatitis.

C. Other supportive measures:

1. *Posture.*—The time-old Trendelenburg head-down position, for bringing back the pooled blood from the capacitance veins into active circulation, is no longer advocated. Simple elevation of the lower limbs serves the purpose. Similarly, use of inflatable pneumatic garments round the lower limbs and abdomen (military anti-shock trousers, i.e. MAST) to compress these veins has been discarded for fear of compression of big vessels.
2. *Respiratory Support.*—In the absence of lung injury, respiration poses little problem. Simple oxygen inhalation with a face mask is sufficient. Only rarely ventilation with endotracheal intubation is required.
3. *Sedation.*—While no sedative is required in the absence of pain its use is imperative when pain is present (e.g. fracture, chest wall injuries, peritonitis) because pain aggravates shock. Morphine or pethidine for adults and barbiturates for children are advocated. The doses should be small and they should be administered only intravenously because peripheral collapse prevents absorption from intramuscular tissues making them useless and causing cumulative after-effects.
4. *Drugs:*
   a. Inotropic drugs may have to be used to improve myocardial contractility, inefficiency of which is indicated by a rise in the CVP but fall in the arterial pressure. Drugs commonly used are dopamine and debutamine. Only small doses should be used to avoid systemic vasoconstriction and impaired renal blood flow.
   b. Vasodilators, used at random in the past, with the idea of improving the peripheral circulation, are now generally discarded because vasodilatation in a hypovolaemic or dehydrated patient may result in disastrous fall in the arterial pressure.
   c. Steroids are of no value unless the patient has adrenocortical deficiency, e.g. Addison's disease or under steroid therapy.
   d. Digitalis is indicated in some elderly patients where the stress of shock induces or aggravates cardiac failure. Caution must be taken in its administration and dose regulation to avoid toxicity.
   e. Sodium bicarbonate, available in ampoules, is administered intravenously if there is metabolic acidosis.

SEPTIC SHOCK

Pathogenesis.—Septic shock, to start with, is usually a combination of vasogenic and hypovolaemic components.

The vasogenic component consists of pooling of a large volume of blood in the skin, reducing the circulating blood volume.

The hypovolaemic component, the more predominant, is due to a generalised leakage of intravascular fluid into the interstitial tissue through the capillary walls,which suffer widespread damage due to bacterial toxins.

Except for occasional cases of Gram-positive infection, the causative organisms are Gram-negative (hence called *Gram-negative shock*). The commonest organisms are *Esch. coli* and then the Klebsiella and the bacteroids. However, most dangerous are the Gram-negative anaerobes.

The common sources of sepsis are:

i. Genito-urinary system, especially after operations and instrumen-tations, and septic abortion.
ii. Abdominal cavity, e.g. peritonitis, intra-abdominal abscess, strangulated gut, biliary tract infections.
iii. Respiratory tract, especially after tracheostomy.
iv. Monitoring catheters, left *in situ* for prolonged periods.

Pathology.—The sequence of events in septic shock is as follows:

1. With systemic sepsis there is a hypermetabolic state and heat production increases. Heat loss is accomplished by diversion of blood to the skin by diminution of arteriolar resistance and opening of cutaneous, arteriovenous shunts. As blood is pooled in the cutaneous vascular bed, the circulating blood volume is diminished and the blood supply to the other areas and vital organs are considerably reduced. This is how the state of shock is initially superimposed on simple systemic sepsis. At this stage, though other features of hypovolaemic shock are evident, the skin is red and hot (*stage of red shock*).
2. In the mean time, bacterial toxins cause an intravascular inflammatory process. There is release of inflammatory factors which produce an intense reaction that damages the lining wall of the capillaries and allows exit of fluid from the intravascular space into the interstitial tissues. This causes a sharp fall in the total blood volume and quick progress of the state of shock. At this stage, hypoperfusion of the vital organs activates the cutaneous pressor mechanisms, diverting blood from the less essential skin to the important vital organs. Now the skin becomes cold and pale (*stage of white or cold shock*).

   This sequence of red and white shock, however, occurs only when the patient is normovolaemic prior to the onset of systemic sepsis. In contrast, if systemic sepsis develops in a subject who is already hypovolaemic, the patient passes straightway to the stage of cold shock.
3. Another important pathological component of septic shock is marked oxygen desaturation of the tissues, effected by two factors:
   a. Progressive pulmonary dysfunction:
      i. The primary cause is leakage of proteinaceous fluid through the damaged capillary walls into the interstitial tissues of the lungs and

then into the alveolar spaces, causing gradual loss of alveolar functions.

ii. The condition is worsened by superimposed bacterial infection.

b. Decreased oxygen utilisation by the tissues resulting from:

i. Arteriovenous shunting—blood by-passes the tissues.

ii. Inability of the cells to utilise oxygen as a direct effect of sepsis.

In the terminial phase of septic shock, inadequate tissue perfusion due to extreme hypovolaemia causes gastrointestinal, pancreatic, hepatic and renal failure. To this is added the marked reduction in oxygen utilisation by the tissues, further hindering the functions of all organs, including the brain and the heart. The final event is cardiac arrest.

Clinical Features.—The *onset* of the symptoms and signs of septic shock may be coincident with those of systemic sepsis or there may be an interval of few hours to several days.

The features of septic shock are essentially those of hypovolaemia, with some modifications due to the sepsis itself.

A. Features of hypovolaemia:

1. Effects of adrenergic discharge to the skin:
   a. Pale, cold and clammy (sticky moist) skin, especially of the extremities.
   b. Empty subcutaneous veins.
2. Effects of renal ischaemia—low urine output (less than 0.5 ml/kg body wt/hour).
3. Effects of cerebral and cardiac ischaemia:
   a. Altered sensorium—restlessness and anxiety, gradually changing to apathy and exhaustion.
   b. Progressive tachycardia and fall of blood pressure with evidence of coronary insufficiency, e.g. arrhythmia, ventricular fibrillation and, finally, cardiac arrest.

B. Special features due to the sepsis:

1. *Skin*.—In the early stages the skin may be red and hot (cf hypovolaemic shock).
2. *Temperature*.—Primarily, with cutaneous hyperperfusion, the skin temperature rises. Thereafter, with reduced skin perfusion, the skin temperature falls, though the 'core' temperature (e.g. midoesophageal or tympanic membrane) may still be high. At this stage the patient feels a cold sensation and there may be shaking chills. The cutaneous vasoconstriction now disrupts the body's ability to dissipate heat and this may cause another rapid rise of body temperature.
3. *Respiration*.—Adult respiratory distress syndrome (ARDS) develops in many patients and at least some of them die of respiratory failure alone (cf hypovolaemic shock). The difference from classic respiratory failure is that the patient is hypocarbic instead of being hypercarbic.

Treatment.—Septic shock is best treated by *prevention,* i.e. prompt recognition of the presence of sepsis and institution of its proper treatment before the state of shock supervenes. This prevention necessitates:

1. Identification of the source of infection.
2. Administration of antibiotics as specific as possible.
3. Institution of surgical drainage, e.g. drainage of abscess, surgical debridement or removal of septic focus.

Once shock has set in, an early manifestation of which is increased fluid requirement to maintain the urine output, very rapid action has to be undertaken to save the patient. This consists of:

A. Treatment for the sepsis.

B. Treatment for the shock.

C. Other supportive measures.

A. Treatment for the sepsis:

1. Identification of the source.—In the majority of cases the source of infection is evident. If no source is apparent, the cause probably lies in the abdomen—USG, CT or MRI may be helpful to detect localised collections.
2. Antibiotics:
   a. If previous culture-sensitivity tests are available, the specific antibiotic can be started immediately.
   b. In other cases, especially when gastro-intestinal tract organisms are suspected, combination of cefazolin, gentamicin/amikacin and metronidazole usually works well.
3. Surgery.—Drainage of abscess or localised collections, surgical debridement, removal of products of abortion, etc.

B. Treatment for the shock.—Essentially the shock is hypovolaemic and the aim is restoration of the blood volume. Prompt correction of the pre-existing fluid deficit is necessary and large quantities of fluid are often needed. However, care must be taken that there is no overloading, because often the lungs are already damaged by the septic process. The types of fluid are:

1. Usually crystalloid solutions, e.g. lactated or acetated Ringer's solution, or isotonic saline buffered with sodium bicarbonate, can effect resuscitation. As much as 10 to 15 litres may be necessary in the first 24 hours.
2. In the absence of specific needs, colloid solutions (5% albumin in isotonic saline or synthetic plasma-expanders like dextran, gelatin or hydroxyethyl starch) are better avoided because of their escape into interstitium of the lungs, resulting from enhanced capillary permeability in septic shock, may be dangerous.
3. Any deficit in RBC count should be corrected by blood transfusion because the damaged lungs must be properly oxygenated.

C. Other supportive measures:

1. *Respiratory Support.*—Many patients develop pulmonary insufficiency. Endotracheal intubation with ventilation is necessary in such cases.
2. *Drugs:*
   a. Inotropic drugs may be necessary to improve myocardial contractility. An abrupt rise in the CVP but a fall in the arterial pressure indicates inability of the heart to contract properly. Drugs commonly used are dopamine and debutamine. Only small doses should be administered to avoid systemic vasoconstriction and impaired renal blood flow.
   b. Vasopressors.—Theoretically vasopressors are indicated because, in septic shock, there is peripheral vasodilatation and pooling of blood. Norepinephrine is the drug of choice. However, vasopressors are better avoided because the degree of constriction may be severe leading to tissue anoxia.
   c. Steroids are used in the cases where the blood pressure fails to rise with adequate fluid replacement. Use of steroids in the presence of infection is discouraged by many for fear of immunosuppression.
   d. Digitalis in small doses may have to be administered in elderly patient in whom the stress of shock may induce or aggravate cardiac failure.

Monitoring a Patient in Shock: While treating a patient of shock, frequent monitoring is essential to decide improvement or deterioration and to modify the treatment accordingly.

A. *In Less Severe and Uncomplicated Cases* the following observations are usually sufficient:
   1. *Vital Signs:*
      a. *Pulse:* Progressive tachycardia and irregular pulse indicate deterioration.
      b. *Blood Pressure:* While systolic and diastolic pressures are important, the pulse pressure, i.e. the difference between the systolic and the diastolic, is a better indication of the cardiac output. Still better is the mean arterial pressure, i.e. diastolic pressure plus one-third of the pulse pressure.
      c. *Respiration:* Persistently rapid and deep respiration and presence of cyanosis are unfavourable signs.
      d. *Surface Temperature:* Warmth of the skin is a good sign while cold clammy skin is unfavourable. Hyperpyrexia in septic shock is dangerous.
   2. *Sensorium.*—Restlessness indicates cerebral anoxia while alertness is a good sign.
   3. *Urine Output.*—This is singularly the most reliable and easy guide. It reflects the renal blood flow which depends on the cardiac output. Urine output of less than 0.5 ml/kg body wt/hour is indicative of insufficient fluid transfusion.

B. *In Severe or Complicated Cases* additional investigations are necessary:

1. CVP (central venous pressure) measurement is necessary in the cases where the urine output is poor in spite of fluid transfusion. The best way is to raise the rate of transfusion till the CVP rises to 10–15 cm of water.
2. PCWP (pulmonary capillary wedge pressure) indicates the left ventricular function. In cases of severe shock and in patients with pulmonary dysfunction, there may be gross disparity between the left and right ventricular functions. A pulmonary artery floatation catheter is used for the purpose. The Swan-ganz type is the best because this can also record the cardiac output by a thermodilution technique.
3. ECG.
4. X-ray of the chest in patients with pulmonary complications.
5. Serum electrolyte estimations.—Sodium, chloride, potassium and calcium.
6. Blood gas analysis at regular intervals.

2

Fluid and Electrolyte Balance and Their Disturbances, Intravenous Fluid Therapy

BODY WATER IN HEALTH

Water accounts for 60 per cent of the body weight in the men and 50 per cent in the women. The body water is distributed in two main compartments—*intracellular* (i.e. within the cells) and *extracellular*. The proportion of intracellular water (ICF) to extracellular (ECF) is 2:1. The smaller extracellular compartment, again, comprises two components—*intravascular* (i.e. plasma) and *extravascular* or *interstitial* (i.e. tissue fluid and lymph). The proportion of interstitial to intravascular water is 3:1. Taking all these facts into account, the total body water in an adult male, weighing 70 kg is 42 litres and its distribution is as follows:

Total body weight	100 per cent	70 kg
Total body water	60 per cent	42 litres
a. Intracellular water	40 per cent	28 litres
b. Extracellular water	20 per cent	14 litres
i. Interstitial water	15 per cent	10.5 litres
ii. Plasma	5 per cent	3.5 litres

Water in these compartments continually interchange positions but this mutual interchange does not necessarily alter the net amount of water in each compartment. In fact, in health, this partition of water is remarkably constant.

However, body water is never stagnant as there are normal daily water losses (output) and allowances (intake). The daily turnover of water in health is about 2.5 litres, as follows:

A. Water intake:

1. Exogenous:
   a. Water taken as drinks—1200 ml.
   b. Water (moisture) in solid food—1000 ml.
2. Endogenous, i.e. water liberated during oxidation of food—300 ml.

B. Water output:

1. Urine— 1500 ml.
2. Faeces—100 ml.
3. Insensible loss:
   a. Drying of the skin (insensible perspiration)—500 ml.
   b. Drying of the respiratory epithelium—400 ml.

Several facts deserve special attention in this respect:

1. The amount of water taken as liquids and that as solids are, unknowingly, almost the same. Hence, a patient, kept on fluids only, should consume double the amount of his normal liquid intake.
2. In health and in the absence of visible sweating, a rough estimate of daily water turnover may be made by adding one litre to the urinary output.
3. Children require greater quantities of water in comparison to their body weight because of several reasons:
   a. They have a larger body surface area per unit of body weight.
   b. There is a greater metabolic activity because they are growing.
   c. Their immature kidneys (only the neonates) have poor concentrating ability.
4. About 8000 ml of fluid is secreted daily in the bowel lumen as digestive juices but almost the whole of this amount is reabsorbed from the gut, except a meagre amount of 100 ml, which is expelled in faeces (*see* intestinal obstruction).

It will follow from the description below *that fluid balance* in the body is effected by direct and indirect factors:

1. *Direct:*
   a. Regulation of water intake by sensation of thirst.
   b. Mechanisms that regulate the output of water by the kidneys—the ADH (antidiuretic hormone) of the posterior pituitary being the most important. ADH conserves water by increasing its reabsorption by the distal tubules and collecting tubules of the kidneys.
2. *Indirect.*—This is by way of mechanisms that regulate sodium balance. Sodium cannot be retained in the system without water, except under very abnormal circumstances (*see* metabolism of sodium, described elsewhere).

ELECTROLYTE BALANCE IN HEALTH

When inorganic salts are in solution (as in the body fluids) they dissociate into two types of *ions*—anions and cations, and these are collectively known as *electrolytes.* Ions are charged particles and they may be —(a) atoms (e.g. Na^+, Cl^-), (b) larger radicles (e.g. HCO_3^-, SO_4^-), or (c) molecules (e.g. protein). Cations are positively charged and anions are negatively charged and one positive charge is equivalent to one negative charge. Thus one Na^+ (monovalent) cation is equivalent to one Cl^- (monovalent) anion but one Ca^{++} (divalent) cation is equivalent to two Cl^- anions. The chemical reactivity of the various electrolytes in the body fluids cannot be evaluated when their concentrations in the fluid are expressed only by their weight in a given volume, e.g. mg/100 ml. Such expressions fail to allow a physiological comparison of the different solutes in a solution. They are, more or less, like specifying an electric motor by its weight rather than its work performance, i.e. horsepower. In expressing the

physiological and chemical activities of the electrolytes three factors are of prime importance:

1. The number of electric charges per unit volume.
2. The number of particles present per unit volume.
3. The number of osmotically active particles per unit volume.

1. THE NUMBER OF ELECTRIC CHARGES PER UNIT VOLUME.—This is expressed as milliequivalents (mEq) per unit volume, e.g. mEq/litre. The term 'equivalent' refers to the chemical combining activity of the electrolyte. An equivalent of an ion is its atomic or molecular weight in grams divided by its valency. Milliequivalent is that figure expressed in milligrams. The mEq of Na^+ (atomic weight 23, valency 1) is 23 mg, of Cl^- (atomic weight 35.5, valency 1) is 35.5, of K^+ (atomic weight 39, valency 1) is 39, while that of Ca^{++} (atomic weight 40, valency 2) is 20 and of Mg^{++} (atomic weight 24, valency 2) is 12. To convert a concentration of an electrolyte from mg/100 ml to mEq/litre, the following simple formula is used:

$$\frac{\text{mg}/100\text{ ml}}{\text{mEq weight}} \times 10 = \text{mEq/litre}$$

(The multiplication by 10 is required to convert 100 ml to 1000 ml, i.e. litre.)

Thus, if the plasma sodium is reported to be 322 mg/ml, the equivalent concentration is 140 mEq/litre, as calculated below:

$$\frac{322}{23} \times 10 = 140;\ 23 \text{ being the mEq weight of sodium.}$$

2. THE NUMBER OF PARTICLES PRESENT PER UNIT VOLUME.—This is important because the osmotic effect of solutes on a fluid is determined by the number of particles present in it—such particles in the body fluids may be ions (e.g. Na^+, K^+, Cl^-) or unionised molecules (e.g. glucose, urea). This is expressed as millimoles (mmol) per litre. The mole of a substance is its molecular weight in grams and mmol is that figure expressed in milligrams. Thus, one mmol of Na^+ is 23 mg, one mmol of Cl^- is 35.5 mg and one mmol of NaCl is 58.5 mg (23 mg + 35.5 mg). One mmol of Ca^{++} is 40 mg. One mmol of urea (molecular weight 60) is 60 mg and one mmol of glucose (molecular weight 180) is 180 mg.

To draw a relationship between (1) and (2) above, it may be remembered that in case of monovalent ions (Na^+, K^+, Cl^-) one mmol equals one mEq while in case of divalent ions (Ca^{++}, Mg^{++}) one mmol equals two mEq.

3. THE NUMBER OF OSMOTICALLY ACTIVE PARTICLES PER UNIT VOLUME.—The osmotic activity of an electrolyte depends upon its number of ions. For example, 58.5 mg of NaCl in one litre of water is converted into 23 mg of Na^+ and 35.5 mg of Cl^-; since the concentration of each ion is 1 mmol/litre of water, the osmotic activity of NaCl solution is 2 mmol/ litre. Again, 111 mg of $CaCl_2$ in one litre of water is converted into 40 mg of Ca^{++} and 71 mg of Cl^- (35.5 × 2); since the concentration

of Ca^{++} is 1 mmol/litre and that of Cl^{-} is 2 mmol, the osmotic activity of the solution is 3 mmol/litre.

The osmotic activity of a fluid is expressed in two terms, viz. osmolarity and osmolality. *Osmolarity* denotes solute concentration (expressed in mmol) per kg of the solvent while *osmolality* means solute concentration per litre of the solution. When the solute concentration is low and the solvent is water, with a density of 1 g/ml (as in the biological system), there is practically no difference between osmolarity and osmolality. Thus, the plasma osmolarity and osmolality are 280–310 mmol/kg and 280–310 mmol/litre respectively.

Table 2.1: Chemical composition of body fluid components (mEq/litre)

		Plasma	*Interstitial fluid*	*Intracellular fluid*
ANIONS	Cl^{-}	103	114	—
	HCO_3^{-}	27	30	10
	HPO_4^{--} (as P)	2	2	100
	SO_4^{-} (as S)	1	1	30
	Organic acids	5	5	—
	Proteins	16	1	60
	Total	154	153	200
CATIONS	Na^{+}	142	144	10
	K^{+}	4	4	150
	Ca^{++}	5	3	—
	Mg^{++}	3	2	40
	Total	154	153	200

It is evident from Table 2.1 that sodium and potassium are the predominant cations in the ECF and ICF respectively. The chief anions in the ECF are chloride and bicarbonate while in the ICF they are phosphate and proteins. The sum of concentration of cations (in mEq/litre) in each compartment, i.e. intracellular, interstitial and intravascular (plasma), equals the sum of the anions (in mEq/litre), making each compartment electrically neutral. This is called *Donnan's equilibrium.*

The mode of transport of water and electrolytes between the three compartments is noteworthy. The capillary endothelium which separates plasma in the intravascular compartment from the interstitial fluid *is freely* permeable to water, cations, anions and other soluble substances. On the other hand, the cell membrane, which separates the interstitial fluid from the intracellular, is freely permeable only to water and only *selectively* permeable to different ions. When there is an excess or deficit of water, its effects are maximum on the ICF volume, because water freely diffuses to and fro across the cell membrane. Changes in the sodium level, however, chiefly affects the interstitial fluid level because sodium carries water with it across the capillary endothelium (the intracellular fluid contains very little sodium).

METABOLISM OF SODIUM

The maintenance of the volume and distribution of body fluids, i.e. homeostasis, is essentially a function of the electrolytes, principally the salts of sodium and potassium, and body's primary aim is maintenance of the plasma volume. The sum total of body's sodium and potassium contents best signifies the total body water. Because sodium is the predominant electrolyte in the ECF, changes in the total body sodium or its distribution are immediately reflected as changes in the body water. A sodium depleted person must be dehydrated and he cannot be rehydrated unless sodium deficiency is simultaneously corrected. Conversely, retention of sodium is usually associated with overhydration, manifested as oedema, necessitating the use of diuretics to increase elimination of sodium salts in the urine.

The plasma concentration of sodium is 140 mEq/litre and the normal range is very narrow, i.e. 135 to 145 mEq/litre. In cases of sodium retention, the plasma level is seldom above 150 mEq/litre and in sodium deficiency, it is seldom below 120 mEq/litre. To keep the sodium concentration fairly so constant in the face of excess or deficit of sodium, the role of simultaneous retention or excretion of water is immediately apparent.

The total body sodium amounts to 5000 mEq, of which 44 per cent is in the ECF, only 9 per cent in the ICF and 47 per cent in the bones. Bone, therefore, is a large storehouse for sodium, ready to compensate its abnormal loss from the body. However, only a little more than half of the sodium in the bones is water-soluble and exchangeable.

The average daily intake of sodium is 100 mEq, available from 6 g of sodium chloride or, in the absence of oral feeding, from 570 ml of isotonic (0.9 per cent) saline solution. An equal amount is excreted daily, mainly in the urine, a little in the faeces. The loss in perspiration, under normal circumstances, is negligible. The daily urinary sodium output may be as little as 2 mEq or as high as 700 mEq in salt restriction and salt loading respectively. If sodium intake is stopped, kidneys stop excreting sodium (cf potassium).

It is evident from Table 2.1 that the level of sodium is the best index of the cationic concentration of plasma and this is balanced on the anionic side by the combined concentration of chloride and bicarbonate. This latter combined level is about 12 mEq less than that of sodium. Hence estimation of serum sodium is of great value in assessing the electrolyte pattern of a particular subject. Whenever possible, the serum chloride and the serum bicarbonate should also be estimated. The normal serum level of chloride is 95 to 105 mEq/litre, while that of bicarbonate is 25 to 30 mEq/litre: Variation in one is corrected by opposite effect in the other so that the sum of the two remains fairly constant, i.e. 120 to 135 mEq/litre.

Sodium (*and chloride*) *balance* in the system is effectively regulated by the kidney. There are three separate mechanisms:

1. Eighty per cent of the sodium excreted by the glomeruli is reabsorbed by the proximal convoluted tubules. This is believed to be under humoral control.

2. Regulation of the glomerular filtration rate is an important factor. If the rate is reduced, the amount of sodium excretion in the urine is diminished and sodium is retained. This occurs promptly whenever the blood volume falls, e.g. haemorrhage. The diminished blood volume works on the baroreceptors (in the carotid sinus and aortic arch). The effect is cast on the vasomotor centre and the sympathetics. The renal blood flow is reduced, the glomerular filtration is diminished and sodium is retained.
3. Reabsorption of sodium by the distal tubules. This is chiefly under control of aldosterone secreted by the adrenal cortex. The secretion of aldosterone, on the other hand, is related to the volume of extracellular fluid as well as its sodium content. The most important stimulator to aldosterone secretion is angiotensin, which again is produced when the renal blood flow is decreased. Apart from aldosterone, angiotensin itself has a direct effect in the regulation of sodium excretion by the kidney.

It is evident from the above description that the factors which act to conserve sodium (and chloride) in the system are inter-related.

METABOLISM OF POTASSIUM

The *total body potassium* amounts to approximately 3,500 mEq. It is the predominant cation of the intracellular fluid—98 per cent of the body potassium is intracellular and only 2 per cent is extracellular; 75 per cent of the potassium is in the muscles. When the muscle protein is used as a source of energy, both nitrogen and potassium are released. The released potassium passes to the extracellular fluid but the surplus is efficiently excreted by the kidneys, so that the plasma potassium concentration (3·5 to 5·3 mEq/litre) usually remains normal.

The average daily intake of potassium is about 75 mEq available from 5 g of potassium chloride. Dietary potassium is chiefly derived from animal and plant tissues as well as fruits and milk. An equal amount is excreted daily, mainly in the urine, very little in the faeces and sweat. Body cannot retain potassium in case of reduced intake (cf sodium), and hypokalaemia sets in. Hypokalaemia is commoner than hyperkalaemia.

Sudden loss of potassium from the cells into the plasma and then in the urine may occur under the following circumstances:

1. When cell protein is broken down. This is a normal response to trauma and operation. This may also occur during starvation. This is because, as has been stated, when the cell protein is used as a source of energy. both nitrogen and potassium are released.
2. When water is mobilised from cells, e.g. during water deprivation.

Potassium is actively excreted by the distal tubules of kidney. This is potentiated chiefly by aldosterone and, to a lesser extent, by cortisol.

Administration of glucose and insulin pushes potassium from the extracellular fluid into the cells, where it helps deposition of glycogen.

DISTURBANCES IN WATER AND ELECTROLYTE BALANCE

In deciding the causes, effects, and management of these disturbances, certain basic facts require attention:

1. Of the three body fluid compartments, viz. intravascular, interstitial and intracellular, body's primary aim is to maintain intravascular volume.
2. The intracellular fluid contains very little sodium but very high amount of potassium and the extracellular fluid (both intravascular and interstitial) just the reverse.
3. The levels of body water and sodium run hand in hand but potussium not so. Depletion of water (dehydration) is usually (but not always) associated with a fall in the sodium level and vice versa.

Taking into account all these facts, disturbances in water and electrolyte balance may be of the following types:

1. Total body water depletion.
2. Total body water excess.
3. Sodium depletion.
4. Sodium excess.
5. Potassium depletion.
6. Potassium excess

Total Body Water Depletion (Dehydration)

Causes.—This usually results from diminished water intake rather than from increased loss. Thus it may occur in:

1. Exhausted, depressed, apathetic or comatose patients, not taking water.
2. Severe nausea and vomiting, preventing drinking.
3. Painful lesions in the mouth or throat, or oesophageal obstruction making the patient unable to drink.
4. Diabetes insipidus, where there is defective absorption of water from the collecting tubules of the kidney and water is excreted in excess.

Effects.—The effect is an *intracellular dehydration.* This is because of the fact that the metabolic response to the stress results in potassium and nitrogen from the cell being extruded out of the cell, together with water, into the extracellular space. At the same time renal retention of sodium (due to increased aldosterone secretion) preserves this extruded water. Also, the water excretion in the urine is reduced to the minimum as a result of increased secretion of ADH of pituitary. Thus the extracellular water is not lost. Hence, pure water depletion leads to cellular dehydration but not to a circulatory failure from reduction in blood volume.

CLINICAL FEATURES:

1. Intense thirst.
2. Oliguria.
3. Weakness.—Though there is weakness, the patient remains capable of mental and physical exertion.
4. Fever.—This may be an important feature in the children.

PLASMA CHANGES:

1. The levels of plasma constituents, viz. sodium and proteins, are raised.
2. However, haemoglobin concentration and packed cell volume remain unchanged because of loss of water from the RBC.
3. The plasma urea level rises because of increased reabsorption of urea by the renal tubules (and not because of renal failure).

TREATMENT.—Non-saline fluids are given orally or parenterally. Plenty of water by the oral route is the best. If drinking is impossible, water is administered in 5 per cent solution of glucose by infusion.

RESULTS.—Recovery usually occurs. Only in long-standing cases hypotension and coma occur due to intracellular dehydration of vital organs.

Total Body Water Excess

This is also known as *overhydration* and *water intoxication.* Healthy individuals can safely take a large amount of water because they react by corrective diuresis, i.e. excreting water without electrolytes. This is done by the glomeruli increasing their rate of filtration and the distal tubules producing dilute urine.

CAUSES.—Overhydration, i.e. retention of water in the system without sodium retention, may occur as follows:

1. Overhydration with peripheral oedema.—Acute or chronic renal failure (especially acute), congestive cardiac failure, cirrhosis of liver with ascites.
2. Overhydration without peripheral oedema:
   a. Intravenous administration of large volume of electrolyte-free water. A common example is infusion of only 5 per cent dextrose solution for more than 24 hours in postoperative cases.
   b. Sudden absorption of large volume of water in the intravascular compartment, as in:
      i. Irrigating with plain water during transurethral resection.
      ii. Repeated colonic wash-out with plain water, especially in cases of megacolon, which has much higher capacity of absorbing water than normal colon.
   c. Impaired water excretion by the kidneys, as in:
      i. Syndrome of inappropriate secretion of antidiuretic hormone (SIADH).
      ii. Antidiuretic hormone-secreting tumours e.g. oat cell carcinoma of bronchus.

EFFECTS.—The primary effect of pure water excess is hyponatraemia.The plasma sodium level falls (135 mEq/litre), thereby reducing the plasma osmolarity. Water is drawn into the extracellular space from the plasma and this water is finally pushed into the cells. The final event, therefore, is intracellular overhydration, i.e. cellular oedema. This causes impaired cellular function, followed by cell damage.

CLINICAL FEATURES:

1. The features of impaired cellular function are most predominant with the brain cells. Features depend on the absolute plasma sodium concentration and its rate of decline. Initially there is apathy, dizziness and headache. With plasma sodium level below 120 mEq/litre, there is confusion and drowsiness. With further decline (< 110 mEq/litre), convulsions and coma set in.
2. Plasma sodium level below 100 mEq/litre causes cardiac arrhythmias and ventricular fibrillation.
3. Nausea and vomiting are important presenting features.

PLASMA CHANGES:

1. The plasma level of all electrolytes falls progressively because of dilution.
2. There is also reduction in plasma protein level.
3. The packed cell volume is reduced.

TREATMENT.—This consists in stopping water intake and, possibly, nothing more. Cases with severe CNS features are treated with hypertonic (5 per cent) saline at a slow rate, with close electrolyte level monitoring. Patients of SIADH may be benefited by administration of demeclocycline, which antagonises the effect of ADH on the distal renal tubules.

Sodium Depletion, i.e. Hyponatraemia

Because of the intimate relationship between salt and water balance, loss of sodium is usually associated with a reduction in the water content of the body.

CAUSES:

1. *Pure sodium depletion,* unattended by significant water loss, occurs only rarely when the person is losing both salt and water and is replacing only water by drinking salt-free liquids, e.g. diarrhoea, profuse sweating and Addison's disease.
2. More commonly the depletion is *mixed,* i.e. sodium depletion is attended by some degree of water loss. The condition is commonly called *salt depletion* because sodium and chloride are usually lost in equal proportions. In surgical practice this is most commonly encountered in conditions where there is rapid loss of gastric, biliary, pancreatic or intestinal secretions, e.g.
   a. Small intestinal obstruction, where these secretions are either vomited or are sucked out (this is the commonest cause encountered in surgical practice).