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Oedema: causes, physiology and nursing management

Aim and intended learning outcomes

This article explains the underlying pathophysiology of oedema. It gives information that will enable nurses to better evaluate the underlying factors that generate oedema, and therefore to offer more comprehensive treatment and support to patients. After reading this article, you should be able to:

■ Describe the fluid compartments of the body.

■ Outline the mechanisms by which substances normally move between these compartments.

■ Describe the processes that allow the exchange of fluids and nutrients across capillary walls.

■ Explain four causative factors for oedema and name disease processes that might lead to them.

■ Explain the consequences and clinical manifestations of oedema.

■ Describe the treatment and nursing management of oedema.

■ Outline the unique aspects of pulmonary oedema and the formation of oedema in cardiac failure.

Fluids in the body

Oedema is an excess of fluid in the tissues (Underwood 2000) and can have a number of causes. Nurses can encounter oedema in many patients and conditions as either a localised or generalised problem. Appreciation of the underlying causes helps in patient assessment, care and treatment.

Water is contained in two main spaces of the body: intracellular and extracellular. Water within the cells (intracellular) is normally about two-thirds of the total body water volume – approximately 28 litres in a 70kg male and 20 litres in a 55kg woman (Bray *et al* 1999). Extracellular water can be further divided

into plasma and interstitial (between the cells) compartments. For a 70kg man, these values are three and 11 litres respectively, while for a 55kg woman they are two and eight litres (Bray *et al* 1999).

TIME OUT 1

What are the normal plasma concentrations of sodium and potassium? Why is it important that potassium levels in particular are kept in close proximity to the 'normal' range?
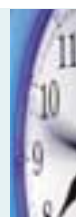
The separate compartments contain different concentrations of solutes, particularly proteins and the electrolytes potassium and sodium. Intracellular fluid (ICF) has high protein and potassium and a low sodium content, while extracellular fluid (ECF) has a high sodium and low potassium content. The normal range for plasma potassium is between 4.5 and 5.5mmol/L, while for sodium it is 145mmol/L (Bray *et al* 1999). Potassium is the key electrolyte involved in muscle and nerve excitation. If the plasma and ECF concentrations of this ion are either too high or too low, nerve, muscle and especially heart muscle function will become either over-excitable or depressed, which can lead to cardiac dysfunction.

Movement of solutes and water

Solutes such as potassium move within fluid compartments by the process of diffusion. This is the random movement of particles from regions where they are highly concentrated to areas of low concentration. Movement continues until the concentration is equally distributed (equilibrium). Movement by diffusion is rapid over short distances

Summary
In this article, Georgina Casey examines the underlying causes of oedema to enable nurses to effectively assess, care and treat patients with this condition.

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but, according to the law of Brownian motion, it takes four times as long to travel twice the distance. For example, the time to reach equilibrium can be measured for a solute in water at room temperature. At 0.1 micrometers from the point of origin it takes 0.00000456 seconds, while to travel 1 micrometer takes 0.000456 seconds and to travel 1 millimetre takes 76 minutes (Bray *et al* 1999).

The cell membrane is a barrier through which many solutes are unable to diffuse freely. Sodium, potassium and glucose all rely on the presence of channels or carrier proteins in the membrane to get into and out of cells. The cell can control the activity of these channels. In addition, active transport mechanisms ensure that the intracellular concentration of key ions is kept at the correct levels.

Protein molecules are large and do not travel freely between the ICF and the ECF. Plasma proteins usually stay within the circulation and intracellular proteins do not leave the cells unless actively secreted via the process of exocytosis, the discharge of particles that are too large to diffuse through the cell membrane. Molecules that are large and do not move out of their compartments easily are termed 'colloids'.

Water moves freely between body compartments by the process of osmosis. Osmosis is the movement of a solvent such as water from a region where it is in abundance to an area where it is scarce. This means that water will move into regions where there is a high concentration of solutes – for example ions, glucose, urea and proteins – from a region where there is a lower concentration of solutes. This process is vital to understanding oedema.

The ability of a solution to attract water into it is called osmolarity. The number of solutes in the solution determines this ability. If body fluid was surrounded by a semipermeable membrane – one that is only permeable to water, not solutes – and placed in a solution of pure water, it would draw water in. This effect can be measured by determining the amount of pressure that needs to be placed on the bag of fluid to stop the entry of water – the osmotic pressure. For body fluids this is approximately 300 milli-Osmoles per litre (mOsmol/L).

Both the ICF and ECF have an osmolarity of approximately 300mOsmol/L. An alteration of this pressure in either compartment would result in the movement of water into or out of the cells. For example, sodium is found in high concentration in the ECF and lower concentration in the cells. Cells usually dispose of any excess sodium that crosses the membrane by activating an energy-dependent sodium-potassium pump. A cell that lacks energy, perhaps due to lack of oxygen or glucose, cannot operate this pump efficiently. Sodium leaks into the cell and is not removed. This increases the number of solutes inside the cell and therefore increases the osmolarity. As the concentration of solutes inside the cell increases, water begins to flow in down its osmotic gradient and the cell swells.

'Tonicity' is the effect a fluid has on the volume of body cells. An isotonic solution has no effect on cell volumes, hypotonic solutions cause the cell to swell and hypertonic solutions cause cell shrinkage as water moves out into the surrounding fluid (Bray *et al* 1999, Tortora and Grabowski 2000).

TIME OUT 2

It is important that routinely administered intravenous infusions are of similar osmolarity and tonicity to body fluids. Why is this? What might happen to the movement of water between ECF and ICF if the infusion given was hyperosmolar? And if it was hypo-osmolar? Check your answers with the text above. Examine the labels of bags of different types of intravenous solution for osmolarity and tonicity.
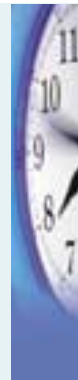
Movement of fluids across capillary walls

The exchange of water, and the nutrients and waste products dissolved in it, across the capillary wall is essential for the survival of body cells. Capillary beds are found in all body tissues. No cell within the body is more than 50 micrometers distance from a capillary (Bray *et al* 1999). Smooth muscle sphincters in the arterioles supplying the capillaries control blood flow through the capillary bed. It takes blood about two seconds to pass through a capillary and into the venule at the other end. This flow rate will increase if the pre-capillary sphincter is relaxed, but it is seldom shorter than one second. This allows ample time for the diffusion of nutrients and oxygen into the interstitial fluid and then into the cells (Bray *et al* 1999).

The structure of capillaries is designed for the transfer of water and solutes between the plasma and the interstitial fluid. The walls are composed of a single layer of cells with the supporting basement membrane. In many tissues the cells are loosely joined so that small molecules can move freely between plasma and interstitial fluid. An exception to this is the brain, where the capillary cells are tightly bonded to each other so that solutes need to pass through rather than between the cells to enter the interstitial fluid of the brain, thus creating the blood-brain barrier (Tortora and Grabowski 2000).

Diffusion and osmosis occur through the cells of the capillary wall, but the gaps between the cells also allow water to escape. This ultrafiltration of water into the tissues is countered by the effect of osmotic pressure that draws water back into the capillary. The opposing forces of hydrostatic (or blood) pressure and osmotic pressure provide a balance between water leaving the capillary at the arteriolar end and re-entering at the venule end (Figure 1). This balance is delicate and if disrupted can lead to oedema.

Plasma colloid osmotic pressure Plasma proteins in the blood are too large to escape from capillaries into the interstitial space, and are thus referred to as colloids. There are virtually no plasma proteins in the interstitial fluid of a healthy person. This difference in protein content creates an osmotic pressure in the capillaries of about 25mmHg (Bray *et al* 1999).

TIME OUT 3

If you are not sure about the effect of osmotic pressure read back over the earlier section. If this were the only pressure operating in the tissue beds, would water move into or out of the capillaries?

Hydrostatic pressure As well as the colloid osmotic pressure, which draws water into the capillaries, there is a hydrostatic pressure within the capillary generated by the pressure of blood flowing from the heart through the arterial system. Blood pressure falls from a maximum of 120mmHg in the aorta of a healthy person, to about 35mmHg at the arteriolar end of a capillary (Tortora and Grabowski 2000). By the time the blood has traversed the capillary bed, pressures have fallen to approximately 15mmHg (Bray *et al* 1999, Tortora and Grabowski 2000). The hydrostatic pressure in the interstitial space is effectively zero – it ranges between about +2mmHg and -2mmHg – and so there is a pressure gradient that tends to force water from the capillaries to the interstitial space – ultrafiltration (Figure 1).

Plasma colloid osmotic pressure opposes hydrostatic pressure with a constant pressure of around 25mmHg (Bray *et al* 1999). The balance between the two opposing pressures leads to net outward flow of water at the arteriolar end and inward flow at the venule end (Figure 1). Any excess water left in the interstitial spaces, or any plasma proteins that do manage to escape through the capillary wall, are removed by the lymphatic system. Between 20 per cent to 50 per cent of the total circulating plasma proteins escape and are returned to the circulation via the lymphatic system each day (Ganong 2003).

TIME OUT 4

Given the information above regarding hydrostatic pressure, osmotic pressure and the structure of the capillary wall, name three factors that would alter the flow of water between the capillary and interstitial space.

Any alteration in the hydrostatic or osmotic pressure will change the net pressures for movement of water between the capillary and interstitial space. Hydrostatic pressure changes with the blood pressure, and most particularly with the venous blood pressure. Plasma colloid osmotic pressure can be

Figure 1. Movement of fluids across capillary walls by filtration and reabsorption

(Adapted from Bray *et al* 1999)
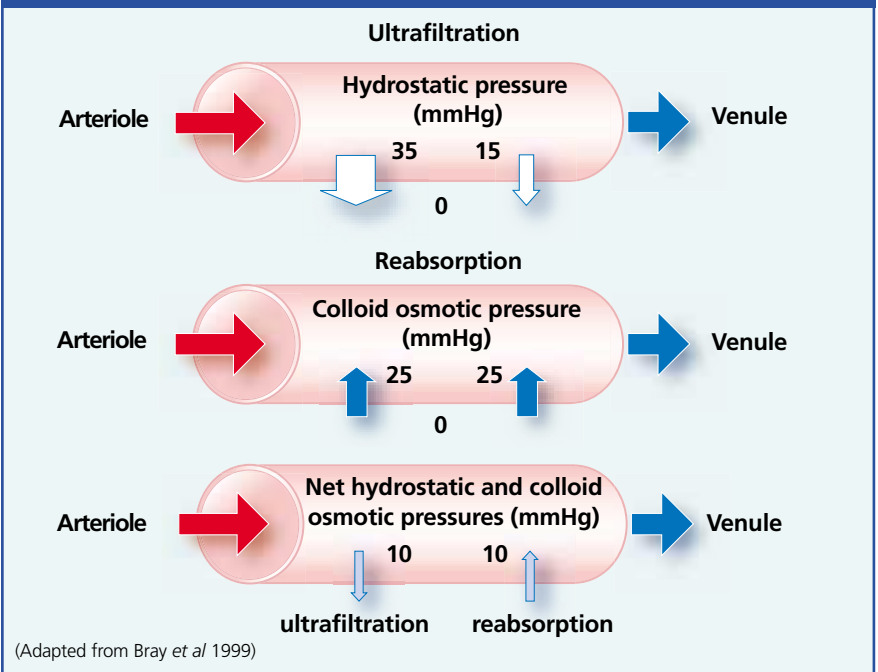
affected by alterations in the amount of plasma proteins in the blood. If the capillary wall structure allows plasma proteins to escape into the interstitial space, the net colloid osmotic pressure will change. Each of these scenarios will be described further below.

Venous hypertension

The pressure of blood in the veins is much lower than that of the arteries. Blood entering the venules from the capillaries has a pressure of about 15mmHg. By the time the blood reaches the right side of the heart, the pressure is around 0mmHg, and may even be negative, particularly during inspiration (Bray *et al* 1999, Tortora and Grabowski 2000).

Blood returns to the heart, driven for the most part by this pressure difference. Venous return is also aided by the action of the skeletal muscle pump. This is the term for the action of muscles, particularly in the legs, that compress the veins and push blood back towards the heart. This mechanism requires the presence of competent valves that only let the blood flow in one direction (Tortora and Grabowski 2000). During respiration the negative pressure generated in the thoracic cavity by inhalation also pulls blood back towards the heart (Bray *et al* 1999).

For adequate venous return it is therefore important to have:
- A pressure gradient along the vascular system.
- Competent venous valves.
- A functioning skeletal muscle pump.
- Good respiratory function.
- Unobstructed flow through the venous system.

Most commonly, where there is marked oedema

an obstruction to flow has occurred. This may be due to blockage, such as a clot in the veins, or an external obstruction such as a tumour or fetus compressing the veins. Congestion may also occur where the right side of the heart is not pumping efficiently due to, for example, heart failure, mitral valve stenosis or *cor pulmonale* (Underwood 2000).

Congestion in the venous system causes distension of the veins, which leads to valve failure as the walls of the veins are separated sufficiently to stop the flaps of the valves from meeting. Blood tends to pool in the veins and subsequently in the capillaries under the influence of gravity. High pressure in the capillaries increases hydrostatic pressure and causes separation of the cells in the capillary wall. Fluid moves into the interstitial spaces, as do plasma proteins.

Figure 2 demonstrates the impact of venous hypertension on the net movement of water across the capillary wall. If, for example, venous hypertension causes sufficient congestion in the capillary bed to increase the hydrostatic pressure from 15mmHg at the venule end to 22mmHg, the effect on the net balance of pressures would be sufficient to allow only a proportion of the water that enters the interstitial space to be reabsorbed. Fluid would then accumulate, resulting in oedema.

Oedema

Localised oedema In any capillary bed, a disturbance of the factors demonstrated in Figure 1 will lead to the presence of excess fluid in the interstitial spaces. If the hydrostatic pressure is increased,
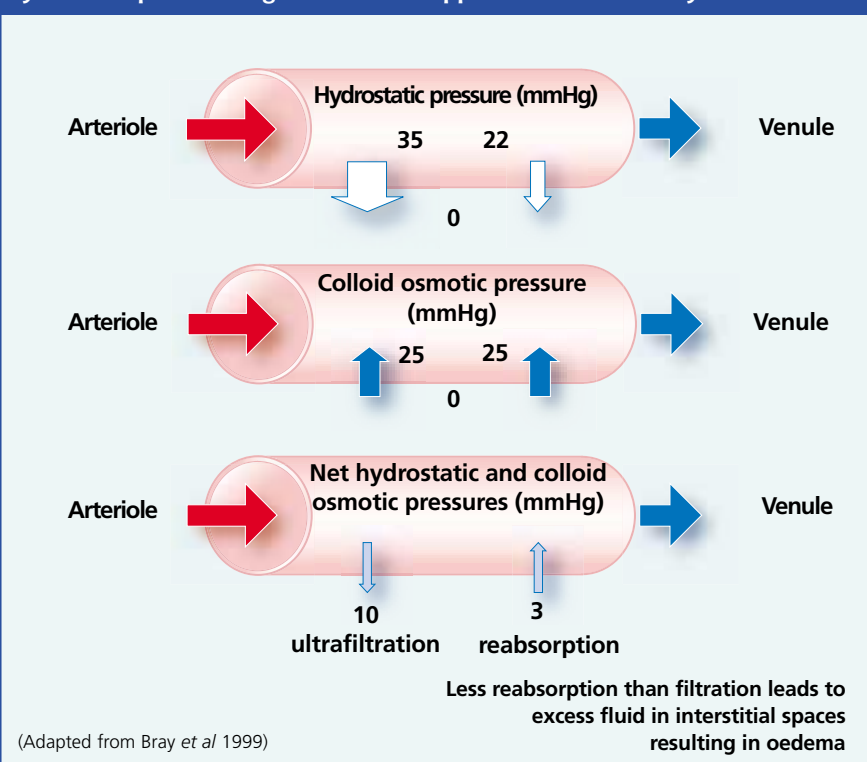
particularly at the venular end of the capillary, the net movement of water will be disrupted. Localised venous hypertension occurs where there is obstruction or reduced venous flow in some part of the venous system. This may be due to a thrombus, obstruction from pressure in the abdomen – for example, obesity, tumour, advanced pregnancy – or to venous stasis where immobility and lack of activity in the skeletal muscle pump decrease the venous return (Bray *et al* 1999, Porth 1998, Underwood 2000). Oedema evident in just one leg, in the absence of musculoskeletal injury, is a strong indication of the formation of a deep vein thrombus (Porth 1998, Underwood 2000).

Venous congestion causes the cells in the capillary wall to separate due to stretching. This effect is also seen with inflammation where, following an injury, the tissue response is designed to allow white cells and inflammatory mediators to escape the capillaries and enter the tissues. Plasma proteins may escape into the interstitial space and this will alter the balance of colloid osmotic pressure across the capillary wall (Figure 3). This in turn will create an area of localised oedema, which is a normal response to injury.

An accumulation of plasma proteins in the interstitial space can also arise where the lymphatic system is unable to remove any escaped plasma proteins. An obstruction to the lymphatic flow may be due to the presence of tumour metastases or parasites (elephantiasis), or following the surgical removal of lymph nodes (Porth 1998, Underwood 2000). Lymphoedema is sometimes called non-pitting oedema, because the swollen limb or region will not display finger marks when gently pressed.

Generalised oedema Oedema that is widespread may be caused by sodium retention or decreased plasma proteins. Where sodium retention occurs, such as in Cushing's syndrome or advanced renal failure (Underwood 2000) when the kidneys are unable to secrete sufficient sodium, excess sodium moves into the interstitial space via diffusion. Water will follow, causing widespread oedema. Where there are fewer plasma proteins than normal in the circulation, a form of generalised oedema occurs. This is referred to as hypoalbuminaemic oedema and may arise because of decreased production or increased loss of albumin (Underwood 2000).

Figure 2. Movement of fluid across a capillary wall following an increase in hydrostatic pressure. Figures used are approximate values only.

Hydrostatic pressure (mmHg)

Arteriole — 35 — 22 — Venule

0

Colloid osmotic pressure (mmHg)

Arteriole — 25 — 25 — Venule

0

Net hydrostatic and colloid osmotic pressures (mmHg)

Arteriole — 10 — 3 — Venule

10 ultrafiltration   3 reabsorption

Less reabsorption than filtration leads to excess fluid in interstitial spaces resulting in oedema

(Adapted from Bray *et al* 1999)

TIME OUT 5

Consider what diseases would cause a decrease in plasma albumin levels. Draw a diagram like Figure 1 but with a decrease in the colloid osmotic pressure of the capillary to 15mmHg. What is the net effect on the movement of fluid across the capillary wall?
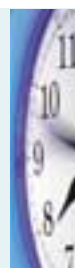
A decrease in the production of plasma proteins will occur in liver failure and severe protein malnutrition (kwashiorkor), while increased losses arise

with nephrotic syndrome (Porth 1998, Underwood 2000) or in severe burns where there is loss of proteins from the burn site. When the colloid osmotic pressure of the blood is reduced, fluid cannot be reabsorbed in the capillaries and it accumulates in the interstitial spaces.

Generalised oedema will also occur in heart failure where the inability of the heart to pump efficiently leads to venous hypertension and a raised hydrostatic pressure throughout the capillary beds of the body. Venous congestion in the portal veins of the liver, associated with cirrhosis, can cause raised hydrostatic pressure in the capillary beds of the abdomen and leads to the form of oedema called ascites (Bray *et al* 1999, Porth 1998).

Generalised oedema leads to depletion in the circulating blood volume and this triggers a cycle that will, if left untreated, exacerbate the oedema. As blood volume decreases with the movement of water from the circulation into the interstitial spaces, blood pressure drops. This triggers the renin-angiotensin-aldosterone pathway that leads ultimately to the retention of sodium and water in the kidneys (Bray *et al* 1999, Cho and Atwood 2002). Normally this would restore blood volume and pressure, but in the case of generalised oedema there is already a disruption to fluid movement across the capillary walls and the extra sodium and water also move into the interstitial spaces, thus increasing the oedema. A summary of the causative factors for oedema is shown in Figure 4.

In adults this excess fluid is generally only detectable once three to four litres have accumulated in the interstitial fluid. There is increased body weight (1kg per litre of water retained), movement of fluid under the influence of gravity (so oedema worsens in dependent areas of the body – feet while sitting and standing, sacrum while lying), and pitting of the tissue when depressed by a finger or restrictive clothing (Bray *et al* 1999). An important sign of generalised oedema and fluid retention is engorgement of the neck veins. In particular the external jugular venous pressure is raised and it is possible to measure this when the patient is lying at a 45-degree angle (Talley and O'Connor 2001).

Consequences of oedema

Whether localised or general, excess fluid in the interstitial space increases the distance between capillaries and the cells they supply. The rate of diffusion of nutrients and oxygen to the cells is decreased and may cause impaired cellular function. Thus wounds may be slower to heal and more prone to infection (Anderson 2003, Bray *et al* 1999). There may be impaired function of tissues throughout the body (Edwards 2003).

With the shift of water from the plasma to the interstitial fluid, there is a decrease in circulating volume. This triggers the renin-angiotensin-aldosterone system to increase the retention of

Figure 3. Movement of fluids across a capillary wall following an increase in colloid osmotic pressure in the interstitial fluid. Figures used are approximate values only.
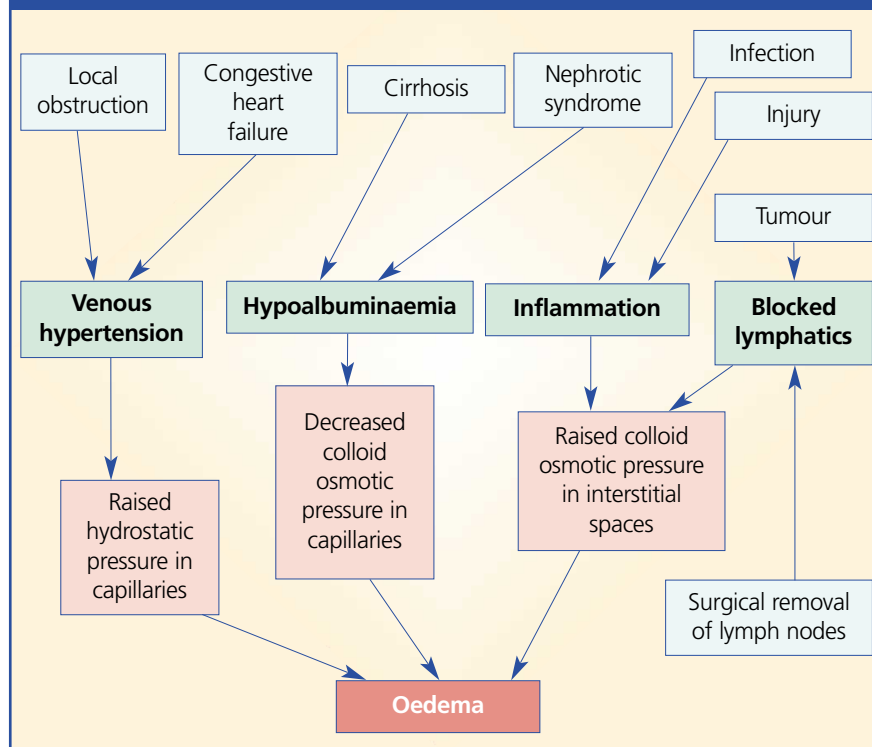
(Adapted from Bray *et al* 1999)
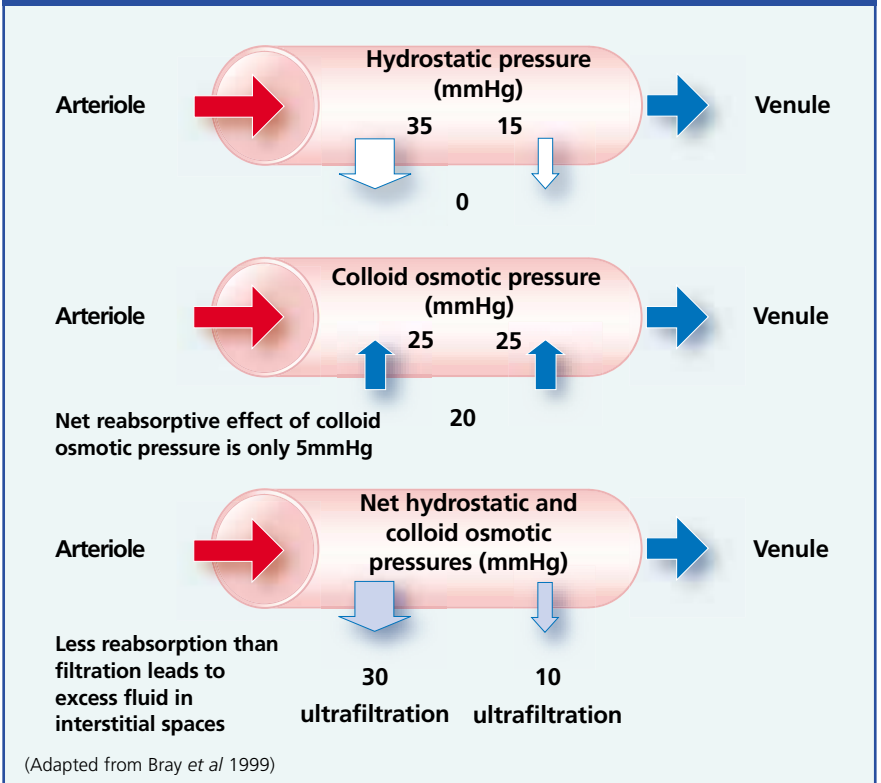
sodium and water at the kidney. Antidiuretic hormone secretion is increased and the thirst mechanism is triggered, leading to increased retention and ingestion of water. If left untreated the result will be circulatory overload.

The third major consequence of oedema is related to the organ system involved. For example, oedema of the cerebral tissue can have severe consequences because the brain is enclosed in a rigid cavity. Swelling of the brain may lead to compression of the tissue and herniation into the spinal column (Bray *et al* 1999, Porth 1998, Underwood 2000). Generalised oedema normally has little impact on the brain since the blood-brain barrier prevents extravasation of plasma proteins and controls the exchange of sodium ions. The most common fluid-related cause of cerebral oedema is the inappropriate intake of large volumes of water (water intoxication), which causes hyponatraemia in the circulation. This reduces the osmotic pressure in capillaries and water shifts into the cerebral cavity, increasing the intracranial pressure (Bray *et al* 1999).

This can also occur, though rarely, with inappropriate administration of intravenous fluids. Commonly used fluids for maintenance are 0.9% sodium chloride and 5% dextrose in water. Usually these are alternated because, while normal saline is largely iso-osmotic, it does contain slightly more sodium chloride than body tissues, so continuous administration may lead to hypernatraemia and cellular dehydration. The extra sodium in the plasma and ECF may also precipitate circulatory overload with

Figure 4. Causative factors for oedema

Figure 4. Causative factors for oedema

gases between blood in the pulmonary capillaries and air in the alveoli. The normal distance between air and blood in the lungs is about 1 micrometer (Bray *et al* 1999); any extra fluid in the interstitium or alveoli will increase this distance, increase the time it takes for oxygen and carbon dioxide to diffuse between blood and air and impair gas exchange.

Pulmonary oedema can arise from increased hydrostatic pressure in the pulmonary circulation, injury to the capillary walls that allows proteins to escape from the plasma, or blocked lymphatic drainage (Porth 1998, Underwood 2000). Most commonly seen is the pulmonary oedema that accompanies left heart failure or mitral valve stenosis. The inability of the left ventricle to pump blood into the general circulation leads to back pressure on the pulmonary venous system. This in turn causes raised hydrostatic pressure in the pulmonary capillaries and fluid shifts into the alveoli. Initially, the lymphatic system will remove excess fluid, but once this drainage capacity has been exceeded, or if there is a blockage of the lymphatics by, for example, tumour cells, signs and symptoms of pulmonary oedema become apparent.

The patient experiences dyspnoea, and may become cyanosed. The venous congestion also triggers orthopnoea (shortness of breath while lying flat) and paroxysmal nocturnal dyspnoea (sudden severe experience of breathlessness during sleep) because venous return from the main circulation is increased while lying down (Underwood 2000). The increased pulmonary venous pressure causes multiple haemorrhages in the alveoli and results in pink or rust-coloured sputum. Pulmonary hypertension due to left heart failure can cause right-sided failure and systemic venous congestion, so the patient will also experience generalised oedema and raised jugular venous pressure (Underwood 2000).

pulmonary and peripheral oedema (Edwards 2001).

Dextrose 5% is iso-osmotic on administration, but the dextrose in the solution is metabolised and the water remains, therefore excess administration can lead to hyponatraemia – due to dilution of the plasma sodium with water – and movement of water into cells (Edwards 2001). The patient may experience weakness and muscle cramps, confusion, coma and death as the brain swells (Bray *et al* 1999, Porth 1998).

Pulmonary oedema

Another organ that is adversely affected by oedema is the lungs. The pulmonary circulation is unique in the body. It is a low-pressure circulation arising from the right ventricle and terminating in the left atrium. Blood passes through the pulmonary arteries, arterioles and alveolar capillaries and then into the pulmonary veins. The right ventricle pumps at a lower pressure than the left, so the blood pressure in the pulmonary artery is 25mmHg (systolic). The pressure falls to about 10mmHg in the capillaries and 5mmHg in the left atrium (Bray *et al* 1999). The colloid osmotic pressure remains constant along the capillary at 25mmHg, so there is a net reabsorption of fluid all along the capillary (Figure 5). There is also an extensive network of lymphatic capillaries in the lungs so that any increase in colloid osmotic pressure in the interstitial space is swiftly corrected.

The low hydrostatic pressure and extensive lymphatic network prevent the accumulation of fluid in the interstitial spaces of the lungs and in the alveoli. This is essential for the efficient exchange of

TIME OUT 6

The advice you would give to a patient about their oedema varies depending on the underlying mechanism. What general advice would you give a patient with oedema of a limb? What specific advice would you give if the oedema was due to:
■ Venous hypertension.
■ Lymphoedema.
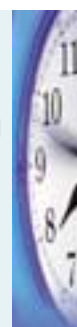
Treatment and nursing management

Since retention of sodium is a major factor in the development of oedema, restricting intake and increasing elimination of sodium are the key forms of treatment. Diuretics act on the kidneys to decrease reabsorption of sodium from the glomerular filtrate and, as a secondary effect, this increases water losses from the body.

Osmotic diuretics such as mannitol increase plasma osmolarity and thus draw fluid out of the

interstitial space. These particles of mannitol (unlike plasma proteins) are filtered at the kidney and increase the loss of water in the urine. Osmotic diuretics do not significantly increase sodium losses so are not recommended for treatment of generalised oedema, but they can be used acutely for treatment of raised intracranial pressure where there is an urgent need to reduce the volume of fluid in the cranial cavity (Rang *et al* 1999). In addition, the rise in circulating volume caused by mannitol may precipitate heart failure where there is generalised oedema.

Severe loss of circulating volume may occur with major loss of fluid into the interstitial space. This may happen with extensive burns, anaphylaxis and bacterial septicaemia, or with depletion of circulating plasma proteins (Underwood 2000). In this case the administration of plasma substitutes such as albumin, dextrans or gelatin will increase the colloid osmotic pressure of the plasma and draw fluid back into the circulation. These substances are not filtered at the kidney so do not cause diuresis. They are slowly metabolised so provide only a temporary correction of the oedema. Plasma substitutes such as these should not be used where the oedema is caused by sodium and water retention, where there is little decrease in circulating volume, because the sudden increase in circulating volume may precipitate heart failure.

Treatment of oedema will not be successful unless the underlying cause of the problem is identified and corrected, if possible (Figure 4). The nurse needs to be aware of the nature of the oedema and provide support and education to patients in line with this knowledge.

Generally, patients should be advised to keep a swollen limb elevated above the level of the heart to assist venous return. They should avoid tight or restrictive clothing on the affected limb. If the oedema is in the legs, advise the patient to avoid compression of the abdomen and suggest weight loss regimens if appropriate.

Skin hygiene is important in patients with oedema. The compromised cellular and lymphatic function mean that infection is more readily acquired and may proceed to cellulites or long-term inflammatory changes in the skin and underlying tissues. Patients should be advised to carefully and gently wash skin daily with soap substitutes. Moisturising creams should be gently applied and skin inspected for signs of breakdown or infection (Penzer 2003). Regular, gentle exercise which activates the skeletal muscle pump will be of use to patients with lower-limb oedema.

For oedema of a single limb caused by venous hypertension, the underlying cause must be identified and treated, since there may be an obstruction in the venous system – for example, a clot. Support of venous function through the use of compression hosiery may be useful. Generalised oedema due to heart failure or venous insufficiency

will be helped by restricted sodium intake and adherence to the prescribed diuretic regimen.

Lymphoedema may be assisted through manual lymphatic drainage – by appropriately trained practitioners – and compression garments that support the lymphatic system function (Mortimer 2000, Penzer 2003). Diuretic therapy will not be useful in this situation because it arises from the accumulation of proteins in the interstitial spaces rather than excess sodium and water.

Conclusion

Oedema occurs as a result of excess fluid in the interstitial space. The manner by which this excess fluid enters the tissues varies according to the underlying disease process: oedema is a symptom of disease. The consequences of oedema vary too, depending on cause, severity and location. Cerebral or pulmonary oedema can have serious consequences for the patient and need immediate treatment. Oedema in other parts of the body may lead to impaired cellular function.

Nurses can provide better education and treatment to patients with oedema by understanding the underlying physiology in the development of this condition. An appreciation of the concepts of fluid exchange across capillary walls and the factors that disrupt these helps in determining the causes of oedema and the rationale for prescribed therapies [n]

TIME OUT 7

Now that you have finished the article you might like to write a practice profile. Guidelines to help you are on page 55.

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Figure 5. Exchange of fluid across the wall of the pulmonary capillaries

Arteriole → **Hydrostatic pressure (mmHg)** 10   10 → Venule

-4

Net pressure for fluid to move out of the capillary = 14mmHg

Arteriole → **Colloid osmotic pressure (mmHg)** 25   25 → Venule

0

Arteriole → **Net hydrostatic and colloid osmotic pressures (mmHg)** 11 → Venule

reabsorption

Less filtration than absorption leads to net movement of fluid into the capillaries

(Adapted from Bray *et al* 1999)