Introduction
Wound healing occurs whenever there is injury to the tissues of the body. An injury describes an area of loss of continuity in any body tissue, this may occur as a result of trauma, infection or a pathological process. The mode of wound healing depends on the powers of regeneration a particular tissue possesses. The following are some examples at specific sites.

Liver
Liver is able to regenerate very well. For example, acute hepatic injury caused by viral hepatitis or toxin exposure, can regenerate completely restoring full form and function. However, chronic insults such as ongoing exposure to alcohol or hepatitis C virus, may result in the formation of collagen based scars and the development of cirrhosis. This latter process is associated with loss of functional liver tissue, so may progress to liver failure. This gives hope for heavy drinkers; liver function may be restored, sometimes after many years of abuse, as long as the person stops drinking before cirrhosis is established.

Kidneys
In the kidneys, epithelial tissues lining renal tubules may regenerate but whole nephrons do not. This means that mild damage to the kidneys will heal completely but more extensive injuries will result in scar formation. The glomeruli are the balls of capillaries within Bowman’s capsule and do not regenerate after injury. However, if one kidney is removed, the glomeruli in the other kidney enlarge to compensate.

Lungs
Damage to the alveoli may occur as a result of infection, inhalation of irritants or shock. As long as the basement membranes of the alveoli remain intact there can be complete healing. However, more severe damage can lead to areas of pulmonary fibrosis. The epithelial lining of the respiratory tract may regenerate effectively after injury, provided that the underlying structures and extracellular matrix framework is preserved. In the trachea and bronchial passages, there is restoration of epithelium from adjacent cells after injury.

Muscle
Skeletal and cardiac muscle cells do not have the potential for significant mitosis in adults. This means that damaged muscle cells are not replaced. In skeletal muscles, if there is a relatively small area of injury, other muscle cells can enlarge to restore overall muscle strength. Damage to the myocardium which results in necrosis is permanent, as cardiac muscle cells do not divide. Healing is by the formation of granulation tissue and fibrosis (i.e. fibrous scar tissue). This means
affected areas of myocardium become non-contractile. This is why early thrombolysis is so important after coronary thrombosis, to prevent necrosis developing.

**Nervous system**

Neurones do not divide and are not capable of mitosis after injury. Any functional recovery which occurs after death of CNS neurones is only as a result of reorganisation of surviving nerve cells to re-establish neural connections. In the peripheral nervous system axons may slowly regrow, but even this does not occur in most of the CNS. Damage to the CNS results in the formation of gliosis, this is a permanent scarring of the nervous system. Gliosis occurs as a result of proliferation of glial cells, which are the supportive and structural non-neuronal cells of the nervous system. Once gliosis is established, there is no ongoing recovery of neurones; this explains why transverse spinal cord injuries cause permanent paralysis, and why dementia is irreversible.

**Common types of wound**

**Contusion**

A contusion is more commonly called a bruise. It is usually caused by a blunt blow, the overlying skin is unbroken, but tissues and blood vessels below are damaged. The discoloration is caused by bleeding from small vessels into the tissues. Red blood cells trapped in the tissue spaces become deoxygenated and dark coloured. Bruising can also develop after deeper tissues, such as bones are damaged, and may only become apparent after a period of time as blood tracks towards the body surface. If blood collects in a discrete pool within the tissues

![Diagram 24.1](image)

*Diagram 24.1*

A contusion or bruise represents the presence of blood cells in the tissue spaces. This causes a characteristic discolouration of the area. Initially a bruise is ‘black and blue’ due to the presence of reduced haemoglobin in the tissues. Over time macrophages phagocytose the red cells in the tissues and the haemoglobin is converted to bilirubin. This is why the discolouration changes from blue to yellow as the bruise fades.
this is described as a haematoma. As the blood in a haematoma is well consolidated it may cause pressure effects on surrounding tissues, these may include pain and nerve compression. There is a risk a haematoma may become infected and some need to be surgically evacuated.

**Abrasion**

An abrasion is a scrape or graze. Typically, there is a superficial surface wound involving the epidermis and part of the dermis. As dermal nociceptors are exposed in the damaged dermis, these wounds are often very painful. Some abrasions can however, be deeper wounds involving tissues below the level of the skin. Abrasions are most commonly caused by friction injuries, falling off bikes is a common cause. These wounds need to be well cleaned to remove dirt and grit which may be sticking to the wound surface.

**Avulsion**

This term describes a wound where there is tissue loss, preventing the closure of the wound edges. An avulsion may be caused by gouging or tearing of tissue.

**Laceration**

Laceration describes a wound made by a blunt object, and has often involved considerable force. The wound edges are usually split or torn with ragged edges as the skin has been burst rather than cut. After significant trauma there may be lacerations involving internal organs. Lacerations of the liver, kidneys, or spleen may be associated with serious haemorrhage requiring urgent surgical attention. This is why traumatised patients should be nursed as still as possible, as movement may dislodge blood clots and result in more serious internal haemorrhage.

**Incised wound**

This is a cut caused by a sharp object. These wounds usually appear neat and the edges can be readily approximated to allow primary healing to take place. In incised wounds the cut may also involve deeper structures such as nerves, blood vessels or tendons. Incised wounds should always be assessed for such deeper injuries and treated as required.

**Puncture wounds**

These may well present as misleadingly small wounds and are also described as penetrating wounds. They are made by pointed or sharp objects. As the edges of the wound may be closed above areas of bacterial contamination, infection is a potential hazard. Also puncture wounds may penetrate down into body cavities or other significant structures such as blood vessels. If the base of a wound cannot be seen it should be surgically assessed as a matter of urgency.
Strains

Strains are injuries to muscles, fascia or tendons caused by stretching forces. Patients complain of pain and stiffness and there may be some associated swelling. It is usually important to exclude other injuries such as fractures. Strain injuries usually resolve with rest followed by progressive mobilisation.

Sprains

A sprain describes an injury to the fibrous tissues surrounding a joint. Fibrous ligaments around the joint are injured, usually as a result of excessive movement of the joint. A mild sprain may involve tearing a few of the fibres in a ligament, in more serious cases there will be associated haematoma formation. In severe cases there may be complete tearing and disruption of a ligament. Patients usually present with local heat, pain, swelling, disability and possible discoloration over the area. Ankles are commonly sprained; if the ankle is turned inwards there will be injury to the lateral ligaments. Sprains usually take longer to recover than strains.

Priorities in wound management

Systemic stabilisation

In any person with a wound, priority is always given to their systemic condition, only after this has been assessed and treated as required, is the wound considered in detail. Airway, breathing and circulation must all first be assessed and managed as required.

Haemostasis

Haemostasis means the arrest of haemorrhage and is clearly important; indeed this may form part of systemic stabilisation if blood loss is significant. Haemostasis is normally achieved by the application of firm direct pressure over the wound. This pressure will close off local blood vessels and give the blood time to clot. Direct pressure may need to be applied for some time before the bleeding stops. In some severe wounds pressure may need to be applied continuously until surgical help is available. Direct pressure should not be applied if there is a foreign body in the wound as this will cause the foreign body to damage underlying tissues; in this case pressure is normally applied around the foreign body to reduce the blood supply to the area. If there is bleeding from an identified blood vessel, this may be clipped using artery forceps and tied off using suture thread by someone with the relevant surgical expertise. Tourniquet use is not normally recommended as a first aid measure in haemorrhage.

However, with some wounds, bleeding should be encouraged. This is true when the wound is likely to be contaminated and when blood loss will not be too severe. For example, in case of accidental needle stick injury you should encourage bleeding as much as possible by squeezing the wound and running it
under hot water. In this case, the outward flow of blood may wash out potential disease causing agents, such as bacteria and viruses. The same is true for other potentially dirty wounds such as animal or human bites.

**Wound assessment**

After bleeding has been stopped the wound should be anaesthetised if required. The wound should be explored to carry out an accurate assessment. Whenever a practitioner is confronted with a wound, they must identify which form of healing is indicated for the particular wound under consideration. This will usually involve healing by either primary or secondary intention.

**Healing by primary intention**

A wound will heal by primary intention if the edges of the wound can be approximated together. Some form of wound closure is normally employed to keep the wound edges closed. Common ways of achieving closure and stability of the wound edges include adhesive strips, sutures or super glue.

**Advantages of healing by primary intention**

Approximation and stabilisation will allow the edges of a wound to heal directly into each other. In primary healing the process is fairly rapid, normally wound edges will be closed with sufficient tensile strength to remove the sutures after 7-10 days. However, it takes much longer than this to restore full strength to the wound, even after 2 weeks the wound only has 20% of full strength. If the edges of a wound are closed, the surface area of the wound is reduced. This means that there will be a minimal amount of scar tissue formed, giving good cosmetic and functional results. As the wound is closed, there is less opportunity for secondary colonisation or infection to enter the wound from outside sources of microbiological contamination.

**Potential problems with primary intention**

Because the wound edges are closed, there is the possibility that foreign material or bacteria may be enclosed within the wound. This will allow any bacteria present to multiply and lead to wound infection with possible abscess formation. Also the presence of foreign material can lead to future complications such as pain and damage to tissues. If foreign material can be removed before closure, this particular complication may be prevented; this is one reason why wound exploration is so vital. If the wound is likely to be contaminated with bacteria from the implement which made the wound, then again it is unwise to close the wound, unless the practitioner feels they are able to adequately wash away the contamination with irrigation or other wound cleaning procedures. When primary healing is not possible, or is not advisable, healing by secondary intention will be used.
Diagram 24.2

i. Cross section of a wound.
This is an incised wound; a sharp object has cut through the epidermis, dermis and some underlying tissue. When the skin is cut the edges ‘fall apart’ or ‘pull open’, this is because intact skin is under a degree of tension.

ii. The edges of this wound have been pulled together as close to their original position as possible, after this approximation a suture has been inserted to hold the wound edges together to promote primary healing.

Healing by secondary intention
In this form of healing the wound is left open and allowed to heal by granulation. It is appropriate to use secondary healing when there is tissue loss and a wound cannot be closed because the edges will not approximate. In addition, if a wound is contaminated secondary healing may be chosen to prevent infective complications. All chronic wounds, such as ulcers, will be colonised with bacteria and so should not be closed.

It is particularly dangerous to close a wound if there are anaerobic organisms present. If a wound is closed, the amount of oxygen the wound is exposed to is reduced; this lack of oxygen promotes the growth of anaerobic bacteria, such as Clostridium strains, which may lead to tissue necrosis and gangrene. Prior to the advent of antibiotics, gangrene was the most common indication for limb amputation. Tetanus, caused by Clostridium tetani, a gram positive bacillus is also anaerobic.

Disadvantages associated with secondary intention
Because the wound is left open to heal by granulation the time taken can be much longer than for primary healing. As secondary healing may be a protracted process, more nursing time will be required before the patient can be fully independent again.

A large wound may take several months to heal. Because the wound is larger there is more scar tissue formed, therefore the cosmetic and functional results are not as good as with primary healing. In secondary healing there is a risk that
a wound may become infected from outside sources of contamination. All chronic wounds are colonised with various forms of bacteria but this does not adversely affect the healing process unless it develops into infection. Bacteria from a colonised or infected wound may however be transferred to another wound, or the wound of another patient, if we are not careful in preventing cross infection. Some wounds may be managed open for a time to allow for cleaning or removal of devitalised tissue. After this time of cleaning such wounds may be subsequently closed, this is a delayed primary closure (or DPC).

Diagram 24.3
Cross section of a wound healing by secondary intention, this involves the formation of granulation tissue which is progressively filling the wound. This will promote the processes of tissue healing with residual granulation eventually becoming scar tissue. An important part of management is the preservation of granulation tissue; this will involve the use of non adhesive dressings and the maintenance of a moist wound healing environment. Particular care needs to be taken not to damage this important but delicate tissue during procedures such as dressing changes.

Delayed primary closure
This means a wound is allowed to heal by primary intention, but only after being managed open for a period of time after the initial injury. First the wound is treated to ensure it is clean enough to permit healing by primary intention. This may involve using agents to clean the wound and the removal of any devitalised tissue, which would necrose if left. After this, the wound is closed to facilitate primary healing. Skin grafting is an example of a delayed primary closure. The donated skin is placed over the recipient area and heals in direct contact with the underlying tissue, so the healing is primary in nature.

Wound healing by regeneration and/or repair
Healing of wounds occurs by two physiological processes called regeneration and repair. In regeneration the wound heals as the lost tissue is replaced by cells
from adjacent healthy tissue. Mitosis occurs in these adjacent cells to replace the cells lost as a result of the injury. This means the tissue is restored, more or less as it was, by the process of cellular and tissue regeneration. This is the ideal form of healing giving good cosmetic and functional results.

Repair is an efficient method of closing and ‘patching’ damaged tissues. The damaged specialised tissue is replaced with collagen. Collagen is a tough protein with high tensile strength; it is the main component of fibrous scar tissue. In repair, the original tissue is replaced with fibrous tissue, so the functional and cosmetic results are poor. Most wounds heal by a combination of regeneration and repair.

**Stages in wound healing**

It is traditional to describe wound healing in terms of stages; however in practise there is overlap. These stages and the physiology involved apply to wounds healing by primary and secondary intention.

*Inflammatory phase*

This is the first stage and is essential if the wound is to progress on to further stages of healing. Damaged blood vessels undergo a reflex vasoconstriction, this is to reduce blood loss and allow the blood time to clot. As a result of haemorrhage the wound fills up with clotted blood. Shortly after the vasoconstrictive phase, release of inflammatory mediators from damaged tissue and mast cells causes an inflammatory vasodilation.

Vasodilation leads to increased local blood flow, a reaction termed a hyperaemia. This increases the flow of nutrients to the injured area as these will be needed as raw materials in the process of repairing damaged cells and producing new ones by mitosis. The increased flow of red blood cells increases the delivery of oxygen, to keep the damaged area well oxygenated. This is vital as wound healing is a very energy demanding process. Anabolic reactions are needed to build and repair cells, and like any building work, large amounts of energy are required. All energy production is dependent on the oxygenation of food based fuels, so if a wound is hypoxic, energy production and hence healing will be impeded. This explains why a good blood supply and effective tissue oxygenation is vital in the process of wound healing.

Inflammatory vasodilation has the effect of increasing the physical size of the gaps between adjacent capillary endothelial cells. This promotes increased capillary permeability resulting in them becoming ‘leaky’. Increased capillary permeability allows larger molecules, such as fibrinogen, to escape into the tissue spaces. In the wound and tissue spaces, fibrinogen is converted into long sticky strands of the clotting protein fibrin. Networks of fibrin strands form a physical barrier to compartmentalise the injured area and may play a vital role in preventing the spread of infection to healthy tissues. The framework generated
by fibrin also acts as a provisional matrix through which healing can start. Also fibrin and other proteins, within the first 4 hours, provide the initial mechanical stabilisation of a wound.

**Destructive phase**

White blood cells are also able to migrate from the blood into the tissue spaces; they can squeeze through the enlarged gaps between the capillary endothelial cells. Neutrophils arrive via the blood and migrate into the tissue spaces of the wound within the first 24 hours. Neutrophils phagocytose any foreign organisms which may have been introduced clearly reducing the risk of infection. Neutrophils also phagocytose debris in the wound such as dead tissue cells. The trauma will have killed local tissue cells and if these are not removed from the wound they will become a ready food supply for bacteria. Monocytes also migrate into the wound after about 24 hours. Once in the tissues, these cells also phagocytose bacteria and dead tissue, this causes them to grow and they become large cells called macrophages. (‘Macro’ means big and ‘phage’ relates to eating, so these cells are literally ‘big eaters’.)

Neutrophils and macrophages are able to move independently through the tissue spaces using a process called amoeboid movement. They are chemically attracted to bacteria and dead tissue, so their phagocytic activity is well targeted. This means bacteria and dead tissue are destroyed in a physiological process of debridement. To debride a wound in surgery means to clean by removing foreign, infected or devitalized material.

In addition to phagocytosis, macrophages also coordinate much of the healing process by release of growth factors. These locally acting chemicals stimulate the regrowth of epithelium, new capillaries and the migration of fibroblasts. At least 20 different growth factors are involved in normal wound healing. In the absence of monocytes, there are no growth factors to stimulate mitosis in adjacent healthy tissues. This means that regeneration of damaged tissues cannot occur.

**Proliferation phase**

This phase of wound healing starts about 2 to 3 days after the initial injury. By this time the phagocytic cells should have cleaned out the wound and disposed of any dead tissue. It is now necessary for fibroblasts to migrate into the wound. A ‘blast’ cell produces and secretes something, so fibroblasts produce the extracellular material needed for fibrous tissue formation. Fibroblasts are attracted into the wound by growth factors released from macrophages and by chemicals released from damaged matrix. (Matrix is the ground substance of a tissue; it fills up the spaces between the cells and other extracellular components. It is composed of a variable mixture of carbohydrate and protein molecules called glycoproteins.) Like phagocytes, fibroblasts are able to actively migrate through tissues, but more slowly.
Diagram 24.4
In the tissues or in a wound, monocytes become large cells called macrophages. As well as ingesting and digesting bacteria and dead tissue in the process of phagocytosis, they coordinate much of the regenerative process, by synthesising and releasing growth factors. These growth factor chemicals diffuse through the tissue fluids and stimulate mitosis and migration of adjacent healthy cells. This cellular stimulation is responsible for the processes of fibroblast migration, angiogenesis and re-epithelialization.

Fibroblasts are essential for wound healing; they synthesise and secrete collagen and ground substance. Fibroblasts also secrete further growth factors which stimulate and regulate the regeneration of new blood vessels, a process called angiogenesis. ‘Angio’ relates to blood vessel and ‘genesis’ means beginning, this process is also sometimes called angioneogenesis, literally the beginning of new blood vessels.

Once in the wound cavity, the fibroblasts secrete collagen strands, these form a three dimensional ‘scaffolding’ through which repair can occur. As soon as 1-2 days after the injury, granulation tissue begins to form. Granulation tissue is a combination of fibroblasts, collagen, new capillary loops, new matrix and macrophages. Later it also contains numerous plasma cells. These are derived from B lymphocytes and secrete antibodies. The combination of antibodies and phagocytic cells makes granulation tissue very resistant to infection. Because circulating blood can be seen through the translucent new tissue, granulation tissue is bright red in colour. Granulation tissue is fragile and bleeds readily because of the new thin walled blood vessels it contains.
Re-epithelialization is also part of the proliferation phase. This refers to the re-growth of epithelial tissue. Viable epidermal cells divide by mitosis and start to migrate over the surface of the granulation tissue. Epithelialization occurs in wounds that heal by primary as well as by secondary intention. In primary intention the epithelial migration begins within a few hours, and if no complications occur, should have effectively sealed off the underlying wound within about 48 hours. This should be the case with sutured surgical wounds, and explains why surgical patients are usually allowed to shower 2 days after surgery.

Re-epithelialization may develop from the wound edges. Anatomically, epidermis dips down into the hair follicles, into the dermis and even hypodermis. This means there are reserves of epidermal cells in these deeper structures. As a result, the epidermis may regenerate from these preserved deep elements. This means that even when the full thickness of the epidermis is lost, full regeneration is still possible.

In wounds healing by primary intention re-epithelialization takes place over the granulation tissue but below the scab on top of the wound, the scab is mostly the residue from the initial blood clot. This scab is very useful as it helps to keep bacteria out of the wound until it can be sealed by the new epithelium. It also prevents the new epithelium and granulation tissue from

---

**Diagram 24.5**
Granulation tissue is only produced when required for wound healing, at other times it is not present. It contains new capillary loops bringing in the blood supply. Collagen strands, ground substance and some growth factors are secreted by fibroblasts. Macrophages phagocytose bacteria and dead tissue and secrete most of the growth factors. Plasma cells derived from B lymphocytes secrete immunoglobulins (i.e. antibodies).
drying out. This is essential as these new cells and tissues can dry out and so dehydrate. This would kill the new cells so prevent re-epithelialization.

Larger wounds healing by secondary intention also need to have a moist environment to preserve the granulation tissue and promote cellular migration. This is best achieved by using some form of dressing to keep in the natural tissue fluids. As well as keeping the wound moist, these physiological fluids also contain essential growth factors released by macrophages and fibroblasts.

Diagram 24.6
The process of re-epithelialization in a wound healing by secondary intention. In these wounds, re-epithelialization is seen as pink tissue around the wound edges. (Re-epithelialization may also develop from preserved ‘islands’ of epithelium within a wound.)

Remodelling phase
This is also referred to as the maturational phase. It typically begins about 3 weeks after the injury and may go on for a year or more, depending on the size of the wound. Collagen fibres progressively align themselves with the tensile forces passing through the wound, this gives progressively increasing strength. Eventually the strength of the wound is about 75% that of uninjured tissue. Especially in wounds which have healed by secondary intention there is contraction of the scar tissue. This is a useful feature of remodelling as it means scars become smaller. However, contractures may develop over joints, especially after burns and may inhibit movement. Such contractures may need to be surgically divided. Wound contraction occurs because specialised fibroblasts, called myofibroblasts, join up and contract in a similar way to smooth muscle. Scar vascularity also reduces with time. Young scars have a pinkie red appearance, due to blood flowing through the tissue. As vascularity decreases the scar fades and will eventually become a similar colour to the surrounding skin. So we can reassure patients that scars will shrink and fade.
Hypertrophic and keloid scars
You may have come across cases of healing wounds where the proliferation of scar tissue continues, resulting in the formation of a hypertrophic or keloid scar. Hypertrophic scars do not proliferate beyond the limits of the original wound, do not increase in size and often regress after 2-3 months. Keloid scars however, normally extend beyond the original wound area and may continue to grow for years. Both of these abnormalities are benign.

Factors which may delay wound healing
Local and systemic factors may influence the rate of wound healing. Local factors describe the conditions in the immediate wound environment while systemic factors refer to ‘whole body’ influences on the local wounded area.

Infection
It is a common observation that infected wounds heal slowly, if at all. Infection means that bacteria are present in the wound and are generating an inflammatory host response. Living bacteria secrete waste products of their metabolism referred to as exotoxins. These substances are toxic, and so inhibit the normal function of local cells and tissues e.g. they may interfere with protein synthesis. Infected wounds need to be well cleaned and often systemic antibiotics are needed. Any foreign bodies in a wound are also likely to be associated with infection.

Poor hygiene
Patients with poor personal hygiene may infect their wounds from other areas of their body or from outside sources of contamination. This can also be a problem in people who interfere with wound dressings.

Local blood supply
Good blood supply to a wound is one of the main factors promoting healing. Wounds on areas of the body with copious blood supplies, such as the face or scalp, tend to heal quickly. Conversely areas of the body with a poorer blood supply, such as the back or feet, heal more slowly. Blood supplies leucocytes, nutrients, oxygen, removes waste products, and keeps the wound warm; all factors which promote healing. Wound ischaemia may occur as a result of the initial trauma, if blood vessels are damaged or compressed by swelling. Pre-existing vascular insufficiency is a significant adverse factor in healing. Ischaemia results in very poor rates of healing or no healing at all. For example, foot or leg wounds in patients with peripheral vascular disease are notoriously difficult to heal. Venous deficiency is an adverse factor in wound healing, as seen in venous leg ulceration. Systemic conditions affecting the cardiovascular system may also reduce local wound perfusion; these may include heart failure or shock. Immobility will also reduce the circulation of the blood and so reduce wound perfusion.
Oedema
The presence of oedema, for whatever reason, adversely affects wound healing. All cells of the body receive nutrients and oxygen from the capillary blood, via tissue fluid, by the process of diffusion. If there is an increased volume of tissue fluid, as is the case in oedema, then there will be an increase in the distance from the capillaries to the tissue cells. This increased distance means nutrients and oxygen have further to travel to reach the cells, so supplies are reduced. The result is that cells become relatively embarrassed and their ability to function is reduced. If cell function is reduced wound healing will be correspondingly adversely affected.

Inhibited wound oxygenation
This may occur secondary to wound ischaemia. Lack of oxygen in the wound is a consequence of a poor blood supply, as it is the blood which transports oxygen to the area. Any systemic cause of hypoxia will also reduce wound oxygenation and healing rates. This means if you are able to improve any underlying causes of wound hypoxia, such as respiratory infection or anaemia, wound healing will be promoted.

Smoking
Smoking may adversely affect wound healing. Nicotine causes vasoconstriction, therefore reduces blood supply to the skin and periphery. This reduces the perfusion of wounds. Smoke also contains carbon monoxide which increases the proportion of carboxyhaemoglobin in the blood. Another problem is that smokers lose more vitamin C than non-smokers. Smoke causes the breakdown and increased excretion of vitamin C from the body, resulting in a chronic shortage. As vitamin C is essential for collagen formation wound healing will be correspondingly inhibited.

Cooling of the wound
Cooling leads to localised vasoconstriction which reduces wound perfusion. As wound healing is dependent on a good blood flow to supply nutrients and oxygen, the process will be inhibited. Removal of metabolic and respiratory waste products will also be correspondingly inhibited. Wound cooling will inhibit the biochemical processes in local cells as this chemistry is dependent on the action of intracellular enzymes. Enzymes are made of proteins and only function within a narrow temperature range. Wound cooling should therefore be prevented as far as possible and should be a consideration in wound redressing procedures. In hypothermia when the whole body is cool, peripheral vasoconstriction will result in wound bed cooling. Also when the body is hypothermic, all metabolic processes will be retarded.
Insufficient diet or malnutrition

For a wound to heal it needs to be supplied with the nutritional building blocks required for the regeneration of tissues. To optimise wound healing the patient should eat an adequate balanced diet including adequate proteins, carbohydrates, fats, vitamins, minerals, fibre and water. Balanced means the dietary components should be eaten in the profile required by the body. Adequate means the components must be eaten in sufficient quantities. Malnutrition delays healing.

Proteins

Proteins in the diet are broken down into component amino acids during digestion. Amino acids then circulate in the blood and are taken up by tissues where they are needed. Human proteins contain 20 different forms of amino acids. A single protein may contain hundreds or thousands of amino acid units. For a wound to heal new cells and tissues must be constructed, amino acids are required as building block components for the new proteins. Without amino acids from the diet, wounds fail to heal properly and may even break down. Lack of protein specifically inhibits angiogenesis, collagen and matrix synthesis and fibroblast proliferation. Severe lack of protein will also lead to hypoproteinaemia and consequent oedema.

Carbohydrates

Carbohydrates are energy giving foods. Wound healing is a very energy demanding process so carbohydrates are important to fuel metabolic processes in the cells and tissues involved. Patients with extensive wounds, e.g. large burns, will need an increased intake of energy giving foods. When adequate carbohydrates are available the body does not need to break down amino acids for energy production.

Fats

Fats are composed of sub-units called fatty acids in much the same way proteins are composed of amino acids. Some components of cells and tissues e.g. cell membranes, are composed of fatty acids, so a supply of these is required for tissue regeneration. In addition to supplying essential fatty acid building blocks, fats also act as a source of energy for cell metabolism in a similar way to carbohydrates.

Vitamins

Vitamins are micronutrients which are vital in the diet. Vitamins A, D, E and K are fats soluble and so may be stored in the body. Vitamins B1-B11 and C are water soluble so cannot be stored. Vitamin C (ascorbic acid) is particularly important for wound healing as it is essential for protein and collagen formation.
When a person is very deficient in vitamin C, old wounds may fall apart as collagen in scar tissue is not adequately maintained. Vitamin A is important for regenerative processes such as re-epithelialization, collagen synthesis and angiogenesis. Vitamin A also helps to reverse the anti-inflammatory effects of corticosteroid drugs and so may promote wound healing in such patients. Vitamin B is needed to facilitate the action of several enzymes needed for normal tissue regeneration. Adequate vitamin K is needed for blood clotting and the prevention of haematoma formation.

**Minerals**

Minerals are inorganic nutrients required in small quantities for health. One essential mineral required for wound healing is zinc, this is probably essential for re-epithelialization.

**Psychological Stress**

Stress has adverse effects on the immune system; this may make wound infection more likely. During periods of anxiety, people release the hormone adrenaline from the adrenal medulla. Epinephrine (adrenaline) is a very powerful vasoconstricting agent. Peripheral vasoconstriction will reduce perfusion of the wound, with a corresponding reduction in local blood supply. Steroid hormones, such as hydrocortisone, are released during periods of stress from the adrenal cortex; these hormones inhibit the inflammatory response. As in other conditions, the way you approach and communicate with your patient has the potential to significantly reduce their anxiety levels. If the patient believes you are trying your best to help them and have the ability to do so, they will feel psychologically better and should therefore heal more rapidly.

**Delayed inflammatory response**

The inflammatory response may be delayed for local or systemic reasons. If the area is cold, there will not be significant inflammation as the vasoconstricting reaction to cold will act against the vasodilatory effect of the inflammatory process. A reduced inflammatory response is also seen in patients who are receiving corticosteroids as these drugs are very anti-inflammatory. Corticosteroids work by decreasing capillary permeability and inhibiting fibroblast activity and the phagocytic capacity of leucocytes. As discussed above, inflammation is the first essential stage in the physiology of wound healing so any factor which reduces this response will delay wound healing.

**Age effects**

Children and young adults usually heal well. It is important to remember that children with wounds need adequate nutrients for wound healing, in addition
to the normal requirements of growth and development. Wound healing in the elderly may be slow due to a reduced number of fibroblasts in their tissues and consequent reduced rates of collagen formation. Re-epithelialization and wound contraction are also slower in older people. The elderly are more likely to have underlying disease processes which may adversely affect wound healing such as diabetes mellitus, heart disease and peripheral ischaemia. They are more likely to have reduced mobility, with increased risk of pressure sore formation.

*Poorly managed or unrecognised diabetes mellitus*

It is a common observation that people with diabetes mellitus often have poor wound healing. Adverse effects on wound healing are related to poor glycaemic control. Higher blood sugar levels inhibit wound healing. Reasons for this include high levels of glucose in the tissue fluids and basement membrane thickening in arterioles, capillaries and venules.