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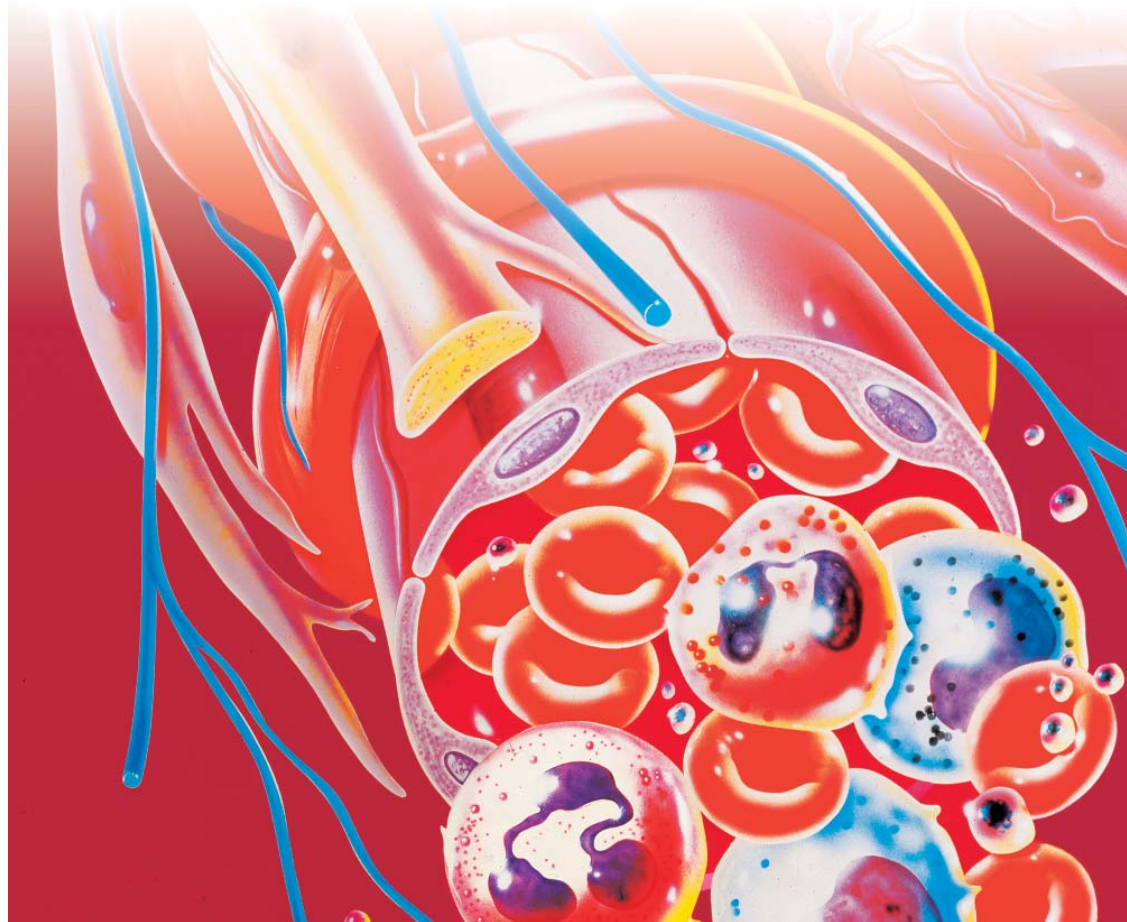
HARTMANN **medical**edition – Compendium Wounds and Wound Management

medicaledition

Compendium
Wounds and Wound Management



Compendium
Wounds and Wound Management

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Foreword

Wound healing is a natural phenomenon. In the physiological situation, nature follows a uniform pattern which, beginning with blood coagulation, then cleanses the wound of damaged tissue, foreign bodies and bacteria in catabolic processes. The process ends with production of new tissue to fill the defect which changes in time into scar tissue.

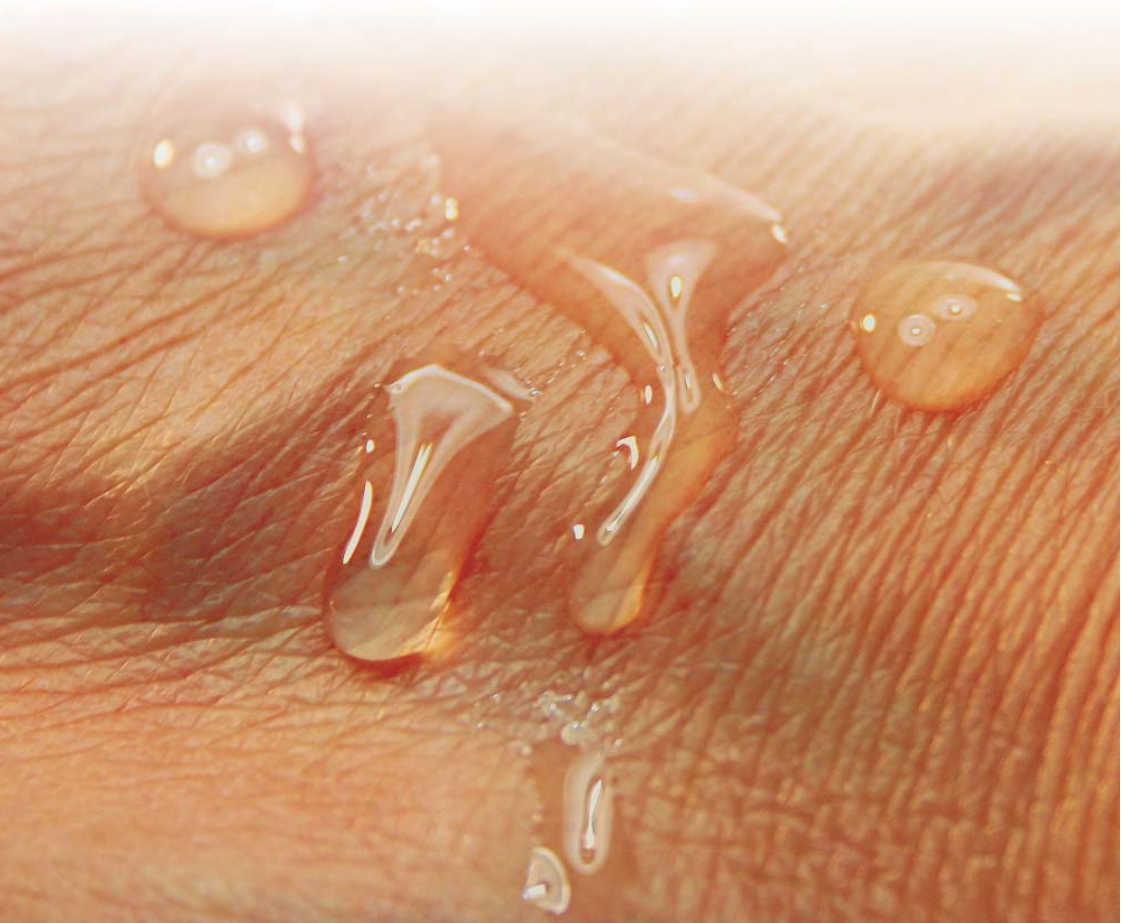
By no means is everything known about the physiology of wound healing, and this can result in many problems, especially in the case of abnormal wound healing. Nevertheless, therapeutic measures can be deduced from current knowledge which support the body's own efforts to restore the continuity of the skin covering.

An attempt has been made in this compendium to represent the basic features of the complex subjects of wounds and wound healing. The structure and functions of the skin are described, followed by the processes of wound healing, influences on wound healing and possible disturbances arising from them, principles of treatment of acute and chronic wounds and treatment with dressings as an important localised therapeutic measure. A later chapter focuses on modern hydro-active wound dressings giving a detailed description of their characteristics and uses. These expand the range of therapeutic options especially in the treatment of chronic wounds when they are used in the appropriate phase.

Wound treatment concerns all the practical disciplines of medicine and nursing. The present compendium is aimed at providing doctors and nursing staff with information and further education in this multifaceted subject.

The skin and wounds

The healing of skin wounds is based on the skin's capacity for epithelial regeneration and the repair of the skin connective tissue. Regeneration means that the injured skin heals without scarring and is possible when only the uppermost skin layer is damaged. Repair, on the other hand, involves the formation of replacement tissue in order to close a skin defect. This is always the case if an injury involves the deeper skin layers. The basis for our understanding of current knowledge of wound healing is, firstly, adequate basic knowledge about the skin, the organ where this takes place.



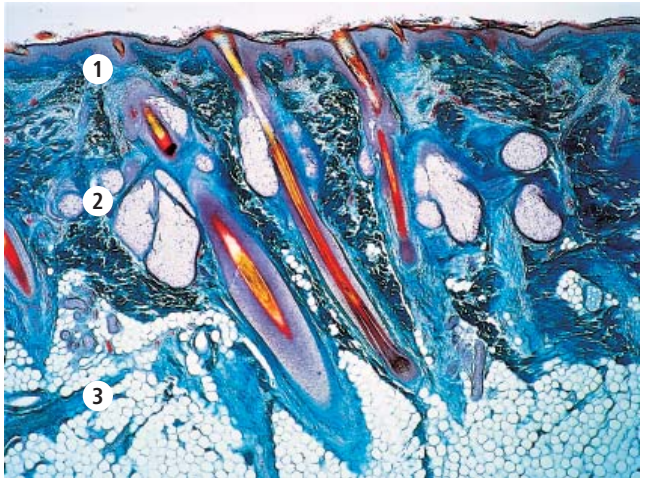
Functions of the skin

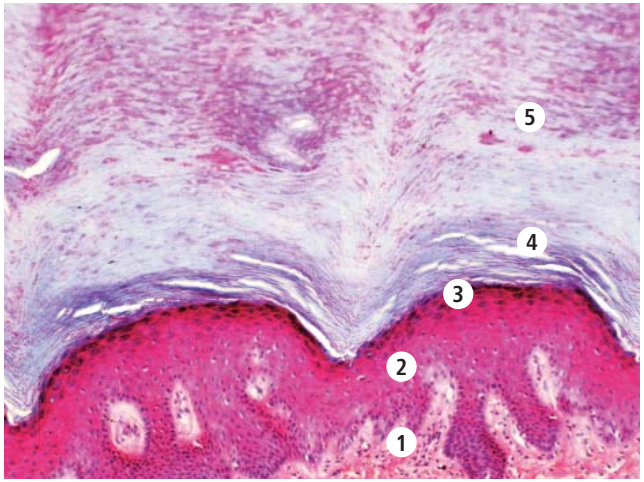
With an area of between 1.6 and 2 m^2 in an adult and a weight of up to 1/6 of the body weight, the skin is the largest human organ. It constitutes the outer boundary layer between the human body and its environment and functions at this exposed site both as a barrier against the outside world and simultaneously as an interface between the outside world and the internal organs. It also has a large number of tasks to fulfil that are essential to life, which is why its undamaged state is so important to the individual's health.

- When the surface is intact, the skin prevents the loss of body fluids and offers protection against the invasion of microorganisms into the body's interior.
- Its mechanical resistance to pressure, impacts and blows is astonishingly high, which is why it is able to protect the internal organs against damage.
- To a certain extent, the skin is able to protect against the harmful effects of chemicals and ultraviolet light.
- It plays a decisive role in heat regulation – by expansion and contraction of the blood vessels and by perspiration – and so contributes to maintaining the vitally essential body temperature of 37 °C.
- As a sensory organ, the skin enables the perception of mechanical stimuli such as pressure, touch and vibration, as well as temperature and pain. Many character-forming sensations are obtained only through the skin, and the human development process could not take place at all without it.

Finally, of particular importance is the fact that the skin is capable of regeneration and repair, which means nothing other than that in the event of its being interrupted or damaged, it can heal itself and restore its own continuity.

The skin consists of the avascular epidermis (1) and the dermis (2), a highly vascularised and innervated connective tissue. This is attached to the subcutis (3) which consists of loose connective tissue containing adipose tissue. The thickness of the skin varies from 1 – 4 mm depending on the demands made on it in the different areas of the body; it is thickest on the palms of the hands and the soles of the feet.





Cross-section through the epidermis at the fingertip, clearly showing the five different cell layers:
1) Basal layer – stratum basale (also called stratum germinativum)
2) Prickle cell layer – stratum spinosum
3) Granular layer – stratum granulosum
4) Lucent layer – stratum lucidum
5) Horny layer – stratum corneum

The structure of the skin

Like any other organ, the skin has a specific fine structure in order to be able to perform its many tasks. For this reason, it is formed in layers having different tissue types: From outside in, three layers of tissue can be distinguished: the outer layer (epidermis), the dermis (also called the corium) and the subcutis. Epidermis and dermis together constitute the cutis, i.e. the skin in the strict sense. The skin also includes the skin appendages, such as hair, nails and various glands.

The epidermis

The epidermis represents a keratinising stratified epithelium composed of five differentiated cell layers, which is perfectly equipped for protective functions, given its stability and imperviousness. Cell division, which is a precondition for growth and regeneration, takes place in the two deepest cell layers. From there, cells push their way to the surface with complete keratinisation occurring in the course of this migration. The uppermost horny layer is shed in a continuous flaking process. Under physiological conditions, renewal of the epidermis from cell division to shedding of the keratinised cells takes about 30 days. The epidermis is avascular and receives its nutrients by diffusion from the blood vessels of the dermis. If the skin bleeds due, for example, to an abrasion, then this means that the capillaries of the dermis have been opened.

The epidermis bears the main brunt of the protective functions of the skin, including defence against ultraviolet rays. Wound healing is therefore seen as complete only once a new fully-functioning epithelium, which is able once more to protect the body against the outside, has formed.

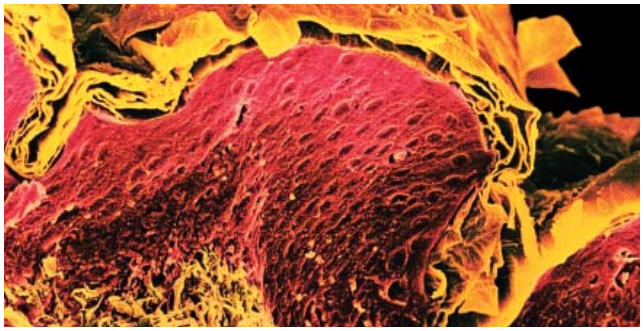
The dominant cell type in the epidermis is the keratinocyte, which earns that name from its ability to synthesize keratins. Keratins are insoluble structural proteins which are highly resistant to extremes of temperature and pH and to enzymatic degradation. They are divided essentially into hard and soft keratins: hard keratins form hair and nails, whilst soft keratins are a main component of the horny cells of the outer epidermal layers.

Apart from the keratinocytes, the epidermis has other cells known as migratory cells, these are cells that are distributed through the tissues without any firm connection to similar cells and undertake particular functions of the epidermis. Important cell types:

- Melanocytes produce the brown/black skin colouring agent melanin, which they release in the form of melanosomes to the keratinocytes. These store the pigment, which then appears as a visible coloration of the skin. This is intended to protect the keratinocytes against damage by UV light while they undergo cell division. The more UV light that falls on the skin, the stronger is the melanosome formation, leading ultimately to tanning of the skin. The quantity and distribution of the melanin are also responsible for differences in skin and hair colour.
- Merkel's cells, also known as Merkel's tactile cells, are flattened, broadened nerve endings which function as slowly-adapting pressure receptors, i.e. they perceive longer-lasting contact. They therefore appear plentifully in the palm skin of the hands and the soles of the feet.
- Langerhans cells play an important role in the immune functions of the skin. They recognise a foreign antigen, absorb and process it, before performing interactions with the immunocompetent T-lymphocyte cells.

Stratum basale – basal layer (1)

The basal layer is the deepest cell layer of the epidermis. It consists of cylindrical keratinocytes which are capable of cell division (mitosis), ensuring continuous regeneration of the epidermis. Cell division is subject to control by numerous substances such as growth factors, hormones and vitamins. The so-called chalones, in particular, appear to play an important part in keeping the regeneration process constant by their inhibitory effect on the obviously unlimited mitotic potential of basal cells. When there is a loss of epidermis, which is associated with a fall in the chalone level, there is rapid regeneration due to "disinhibition" of mitotic activity in the basal cells.



Section through the epidermis: At the top, the stratum corneum (brown) with its layers of corneocytes is visible. Adjoining this are the layers containing living cells (lilac). At bottom left, the dermis (yellow), via which the epidermis is fed, can be seen.

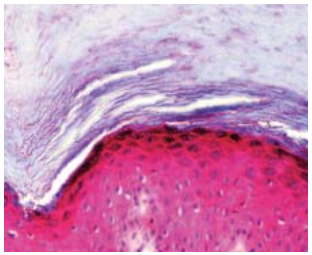
The basal layer runs in a wavy form along the plug-like projections (papillae) of the dermis. Between the basal layer and the dermis is the avascular basal membrane. It separates the two skin layers but at the same time it serves to anchor the basal cells and controls the transport of proteins.

Stratum spinosum – prickle cell layer (2)

The prickle cell layer contains up to six layers of irregularly shaped cells which synthesise keratin peptides and still have slight mitotic activity. They are connected to one another by cell bridges (desmosomes) which give the cells their "prickly" appearance. Fluid is stored between the desmosomes.

Stratum granulosum – granular cell layer (3)

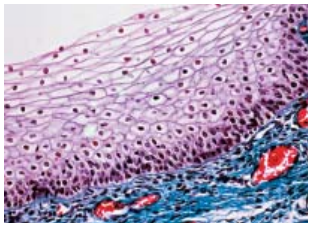
Gradual keratinisation begins in the granular cell layer. Depending on the thickness of the horny layer, it comprises one to three layers of flat cells which contain large granules of keratohyalin. The granules contain, beside others, a precursor protein which is believed to be involved in the formation of keratin fibres in the intercellular space.



The stratum lucidum protects against the ingress of aqueous solutions.

Stratum lucidum – lucent layer (4)

The lucent layer consists of non-nucleated cells in which intense enzymatic activities take place. Keratinisation is continued here which includes the breakdown of the keratohyalin granules of the granular cell layer to eleidin. Eleidin is a fat- and protein-rich acidophilic substance with strong light-diffracting properties. It appears as a homogenous shiny layer – and it is from this that it gets the name of stratum lucidum – and protects the epidermis from the effect of aqueous solutions.

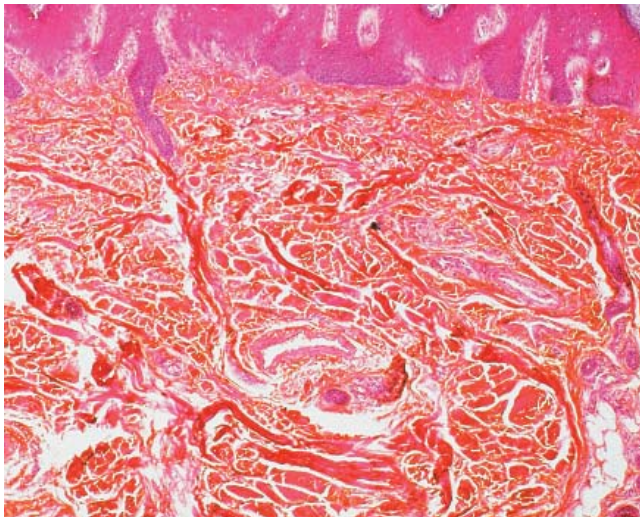


The upper skin section reveals the thickness of the corneal layer. The scanning electronmicrograph of the corneocytes shows that they are layered like roof tiles.

Stratum corneum – horny layer (5)

It is in this layer that the process of cornification is completed: The keratinocytes are filled with the horny material keratin and are now designated corneocytes. They lie on one another like roof tiles and are firmly bound to one another by keratohyalin and very fine fibrils (tonofibrils). The cell layer has about 15 to 20 layers of cells, the outermost of which is lost as skin scales.

The corneal layer is involved, together with the secretions of the sweat and sebaceous glands, in forming the surface film (the hydrolipid film), also known as the acid protective layer. It helps to keep the colonisation of the skin with microorganisms in a physiological equilibrium. If the epidermal layer is damaged by eczema or injuries, germs and harmful substances can penetrate into the skin unhindered.



The dermis is a connective tissue rich in vessels and nerves, which is classified histologically into two layers, the papillary layer and the reticular layer.

The dermis

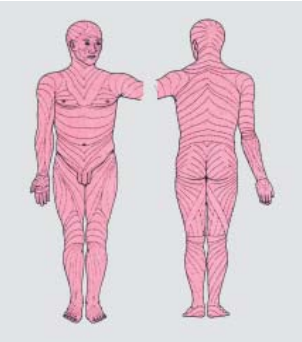
The dermis is attached to the basement membrane of the epidermis. It is a connective tissue rich in vessels and nerves which is subdivided histologically into two layers: the outer papillary layer (Stratum papillare) and the inner reticular layer (Stratum reticulare). The layers are distinguished by the thickness and arrangement of their connective tissue fibres but are not separated from one another.

Stratum papillare – Papillary layer

The papillary layer is bound firmly to the epidermis by projections of connective tissue, the papillae. There are capillary loops in the region of the papillae which ensure nutrition of the avascular epidermis as well as free nerve endings, sensory receptors and incipient lymph vessels. The connective tissue itself consists of a framework of fibrocytes (resting form of the fibroblasts), interspersed with elastic collagen fibres. The intercellular space is filled with a gel-like ground substance, the extracellular matrix, in which mobile blood and tissue cells can move.

Stratum reticulare – Reticular layer

The reticular layer consists of interwoven strong bundles of collagen fibres between which elastic fibre meshes are stored. This structure gives the skin its elasticity so that it can adapt to movements and volume fluctuations of the organism. It is also capable of absorbing and giving off water in a dynamic process.



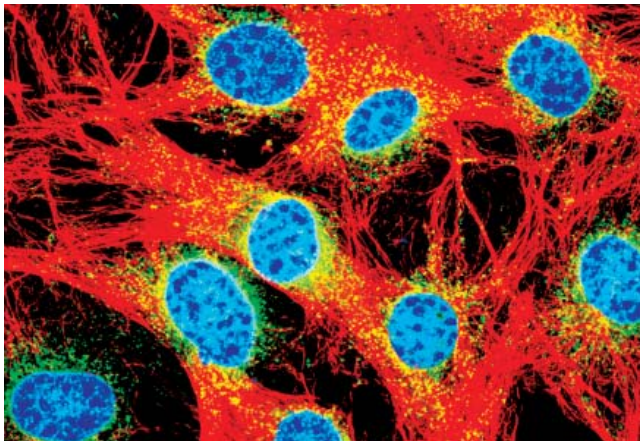
The course of the Langer's skin crease lines should be borne in mind where possible for incisions to obtain cosmetically inconspicuous scars.

The collagen fibres run in all directions but are oriented mainly obliquely to the epidermis or parallel to the surface of the body. The skin's natural crease lines, which run in the direction of the least skin elasticity and perpendicular to the skin tension lines, are called Langer's skin crease lines. Incisions should follow their course where possible. Skin incisions along these lines do not gape and give virtually invisible scars while incisions running across them leave markedly wider scars.

Cellular components of the dermis

The predominant cell type in the skin connective tissue is the fibrocyte which in its activated form is designated as a fibroblast and provides a range of substances for producing new tissue: Fibroblasts synthesize and secrete precursors of collagen, elastin and proteoglycans which mature outside the cells into collagen and elastin fibres and, in non-fibrous form, form the gel-like ground substance of the extracellular matrix.

In the dermis there are also found mast cells, the granules of which contain heparin and histamine, macrophages, which derive from the blood monocytes, and lymphocytes. The cells participate in the non-specific and specific defence mechanisms of the body (phagocytosis, humoral and cell-mediated immunity). In addition, they secrete biochemically active substances which have communicating and regulatory functions and thus are essential for the progress of the repair processes.

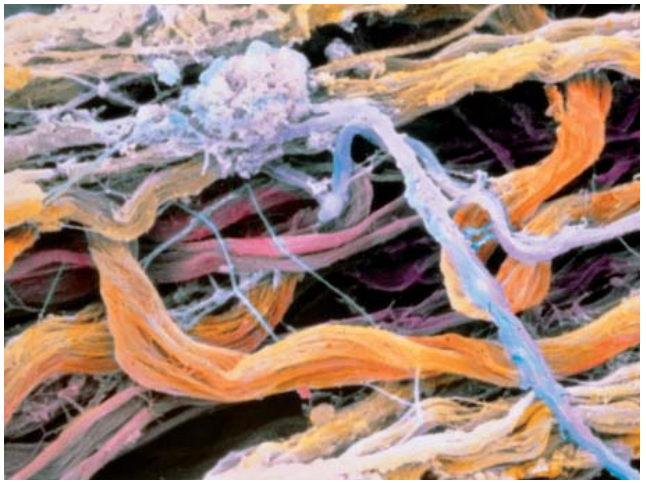


The fibroblasts represent the most important secreting cells for forming skin connective tissue (nucleus blue, cellular skeleton orange).

The fibre proteins of the dermis

The connective tissue fibres of the dermis consist of the structural protein collagen which is an extremely resistant biological material accounting for 60 – 80 % of the dry weight of the tissue. The name "collagen" is derived from the fact that these proteins swell when being boiled and yield a glue, "colla" in Greek. Of the four biochemically distinguishable collagen types occurring in the human body, the main one found in the dermis is the fibre-forming type I collagen.

The production of collagen fibres takes place in an intracellular stage and an extracellular stage, beginning with the fibroblasts. First, fibroblasts release intracellularly a triple helix of procollagen into the extracellular space. This triple helix is made up from two thirds of the characteristic amino acids of collagen (glycine, proline, hydroxyproline) and from one third of other amino acids. In the extracellular space, further enzymatic modifications take place by which the still soluble procollagen is changed into insoluble collagen fibrils, finally being assembled into collagen fibres.



Electron microscopic appearance of skin connective tissue with collagen bundles and elastic fibres. The substances required for the formation of the fibre proteins are provided by the fibroblasts. They synthesize precursors of collagen and elastin which are released into the extracellular space and mature through various enzymatic processes into collagen and elastin fibres.

Another fibre protein of the dermis is flexible elastin which is also synthesized and secreted by the fibroblasts. Elastin is a spiral polypeptide chain with highly elastic properties from which a two-dimensional framework similar to a trampoline net is produced outside the cell. This structure allows reversible stretching of the skin so that overstretching and tearing is largely avoided.

Non-fibre ground substance of the dermis

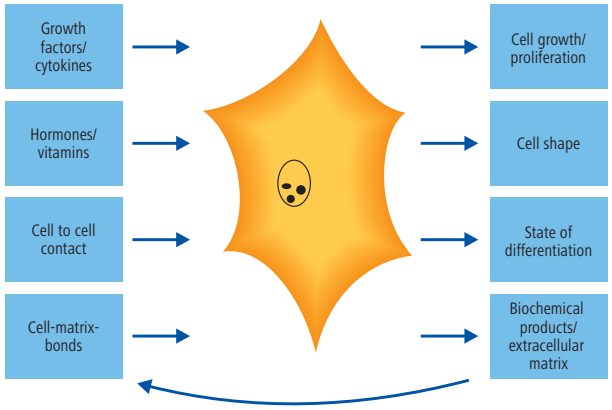
The space between the fibres of the skin connective tissue is filled with amorphous ground substance, salts and water. An important constituent of the ground substance are proteoglycans. These are compounds of polysaccharides and proteins with a high proportion of carbohydrates which were formerly known as mucopoly-saccharides.

Proteoglycans are very hydrophilic and can bind a large volume of water so that a sticky gel-like substance is formed. They are not purely structural proteins but also appear to have an influence on cell migration, cell adhesion and cell differentiation.

Furthermore, the ground substance also contains a range of other glycoproteins with a lower proportion of carbohydrate such as thrombospondine, laminine/nidogen complex, k-laminine and tissue fibronectin. These have a similar multiplicity of function as the proteoglycans. Fibronectin, for instance, is an adhesive protein which binds cells to collagen and thus also plays an important part in wound healing.

Extracellular matrix

In tissue, the cells are usually closely bound to the substances they secrete themselves. To achieve this, the macromolecules of the extracellular substances form complex three-dimensional networks which are called the extracellular matrix (ECM). Such a matrix can be found in every body tissue, where the structure and composition have tissue-specific differences and depend on the type of matrix-producing cells and the function of that tissue.



Schematic diagram of the flow of information: cell – extracellular matrix

Although all the functions of the ECM are not yet known, it is assumed that it serves not only as a filler substance between individual cells, tissues and organs but also fulfils a variety of tasks in the transmission of information between the cells embedded in it.

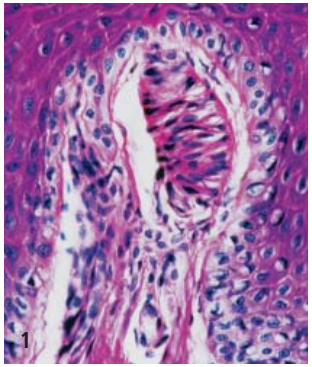
The subcutaneous tissue

The subcutaneous tissue is the deepest layer of the body's outer covering. This layer consists of loose connective tissue and is not sharply demarcated from the dermis. In its depths, it is bound to muscle fasciae or periosteum. Apart from a few sites in the body, fat can be deposited in the entire subcutaneous tissue which has insulating, storage and modelling functions.

Sensory receptors in the skin and subcutaneous tissue

The skin is innervated by different types of free nerve endings and stimulus-receiving receptors which enable it to function as a sensory organ. The Merkel cells in the epidermis can perceive prolonged touch. Along the papillary bodies of the dermis are aligned the Meissner corpuscles which serve as touch receptors for very fine pressure.

A large number of nerve receptors makes the skin a sensory organ that is essential to life. Some examples of this:
1) Meissner's corpuscles
2) Free nerve endings
3) Vater Pacini corpuscles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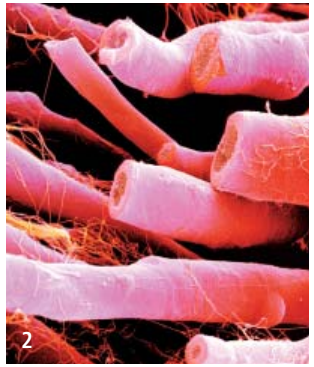
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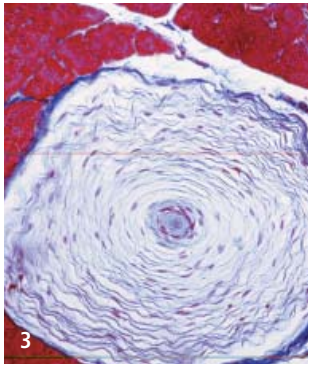
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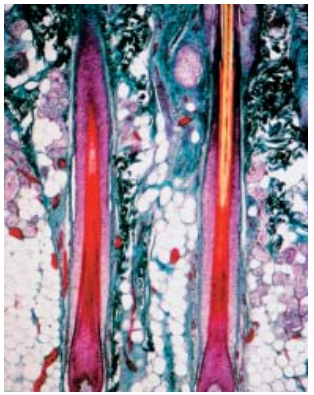
They are accordingly found in greatest density on the fingertips. The Kraus end bulbs are important in the perception of cold, the Ruffini corpuscles in the subcutaneous tissue function as warmth receptors. Free nerve cells close to the skin's surface transmit pain sensations. The Vater Pacini corpuscles of the subcutis react to mechanical deformation and vibration.

The skin appendages

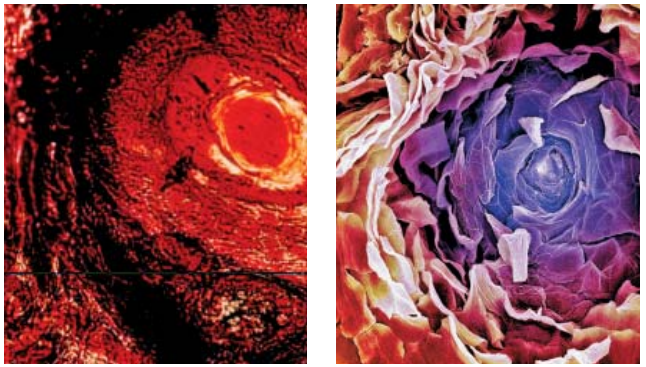
The appendages of the skin include hair and nails in addition to sebaceous, sweat and scent glands.

Hairs are flexible and at the same time strong thread-like structures made of the horny substance keratin. They develop from inward projections of the epidermis and the hair shaft, which is at an angle to the skin surface, extends deep into the dermis. Their growth follows an endogenous cycle which is specific for every hair root. Therefore no synchronous growth between neighbouring hairs takes place. Hair roots cannot be regenerated so scar tissue always remains hairless. However, epithelialisation can take place from the remaining epithelium of a damaged hair shaft.

Finger- and toenails are translucent keratin plates which grow outwards from the nail root to the free edge. Growth is about three millimetres a month and is closely associated with many organ functions. Thus the condition of the nails can often give important diagnostic clues.



Electronmicrographs of hairs (above). The illustration below shows hair roots with clearly recognisable epithelial cells. In the event of injuries, re-epithelialisation can take place from the residual epithelial cells. The hair roots themselves cannot be regenerated so scars always remain hairless.



Electronmicrographs of a sebaceous gland (left) and a sweat pore (right). Apart from the hairless skin on the soles of the feet and the palm of the hands, sebaceous glands are found at all sites on the body and are particularly numerous on the face and scalp. Here there can be up to 900 sebaceous glands per square centimetre. The human body also has a plentiful covering of sweat glands, totalling about 2.5 million.

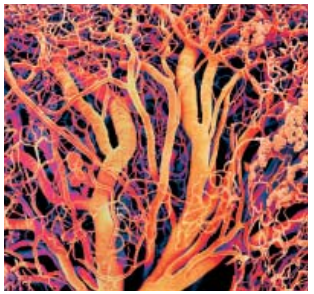
Sebaceous glands open into the hair funnels of the hair follicles so that their presence is associated with hair follicles with few exceptions. The sebum, a mixture of fats, cells and free acids, lubricates skin and hair and protects them from drying out. Control of sebum production is a complex process which has not yet been investigated in every detail.

Sweat glands also arise from cells of the epidermis, which then sprout into the depths of the dermis so that the actual gland is in the corium. The excretory ducts open in the pores on the skin's surface. Sweat is an acid secretion consisting, besides others, of water, salts, volatile fatty acids, urea and ammonia. Sweat covers the skin surface with a protective acid coating. Sweat secretion serves mainly for temperature regulation.

Scent glands, in contrast to the sweat glands, produce alkaline secretions. Scent glands are found mainly in the axillae, around the nipples and in the genital area. They commence their secretory activity with the onset of puberty.

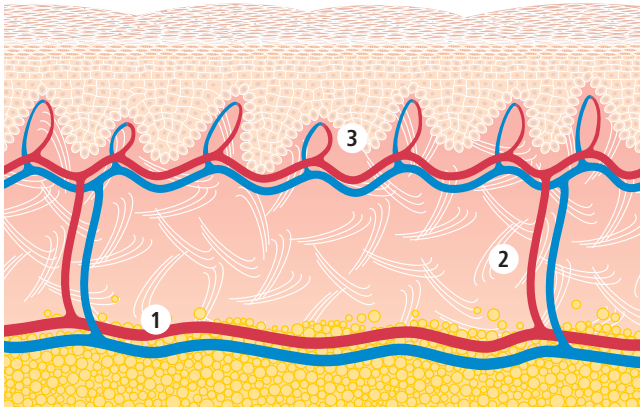
The blood supply of the skin

The stepwise distribution of vessels in the skin corresponds to the layered surface structure of this organ. Numerous vessels arise form the arteries lying under the subcutaneous tissue, which form a cutaneous plexus between the subcutaneous tissue and dermis. Wherever the skin is more mobile, the vessels take a pronounced tortuous course. Individual arterioles run outwards perpendicular from the deep cutaneous plexus and branch at the foot of the papillary layer into the subpapillary plexus. Very fine looplike capillaries extend from there into the papillae of the dermis and thus ensure perfusion of the avascular epidermis.



Blood vessels of the skin (electronmicrographs)

The papillary layer is supplied copiously with vessels while the reticular layer is relatively avascular. Removal of metabolic products is effected via the corresponding venous networks and partly also via the lymphatic system.



Schematic diagram of the blood supply in the skin. From the cutaneous plexus between subcutaneous tissue and dermis (1), individual arterioles (2) run perpendicular to the surface and branch at the foot of the papillary layer into the subpapillary plexus (3) which supplies the epidermis.

The constituents of the blood

The blood, also called the liquid organ of the body, serves as a transport medium for respiratory gases, nutrients, products of metabolism etc. Moreover, the cells of the immune system circulate in the blood in addition to the constituents of the coagulation system which contribute in the event of injured blood vessels to rapid sealing of the leaking sites. The soluble (plasma) and cellular components (red and white cells, platelets) of the blood can be separated by centrifugation.

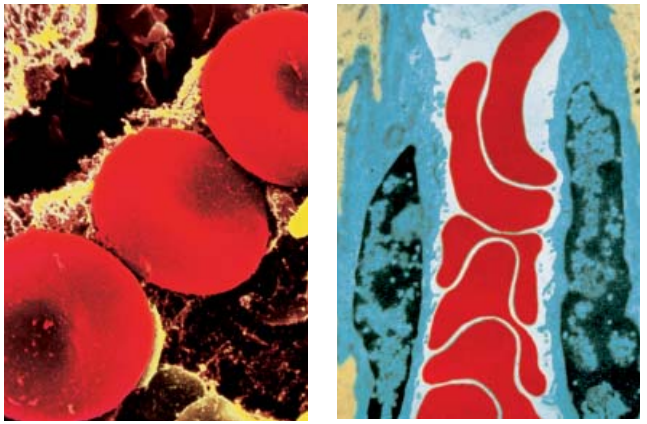
	Composition	Function
Blood plasma	**Water (90 %)** **Electrolytes** Cations: Anions: magnesium chloride potassium bicarbonate calcium phosphate sodium sulphate **Organic constituents** Proteins (7-8 %) albumins, globulins fibrinogen (factor I) → lipids glucose	maintenance and regulation of water and electrolyte balance maintenance of oncotic pressure, protein reserve, transport proteins blood coagulation
Platelets	→	blood coagulation
White cells	granulocytes monocytes lymphocytes	immune system
Red cells	carriers of the red blood pigment haemoglobin	transport of the respiratory gazes: oxygen and carbon dioxide

Blood plasma

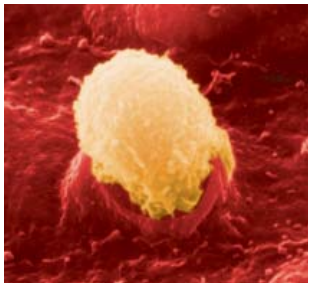
Plasma is a yellowish clear liquid consisting of water (90 %), proteins (7 – 8 %), electrolytes and nutrients (2 – 3 %). Of the proteins, about 60 % are albumins and 40 % globulins. A constituent of plasma which is important in wound healing is fibrinogen (factor I), which is essential for blood coagulation. Plasma which no longer contains any clotting factors is called serum.

Red blood cells (erythrocytes)

About 95 % of blood cells are red cells: non-nucleated disc-shaped cells with a central concavity, containing high concentrations of the red blood pigment haemoglobin. Their main function is the transport of oxygen and carbon dioxide which are bound reversibly with haemoglobin. The gas exchange itself is favoured by the hollows in the sides of the cells due to an increase in the surface area of the small blood cells. This shape also facilitates deformation of the cells when passing through the smallest capillaries. The erythrocytes are formed in the red bone marrow. Their life-span is about 120 days after which they are broken down, mainly in the spleen.



The shape of the red blood cells with their central concavity favours the exchange of oxygen and carbon dioxide and facilitates passage through the capillaries.



Subtraction-stained representation of a white blood cell migrating through the endothelium of a blood vessel. Because of their capacity for movement, the leucocytes can migrate to the "scene of the action" and, for instance, reach an injured skin area where they can act as defence cells.

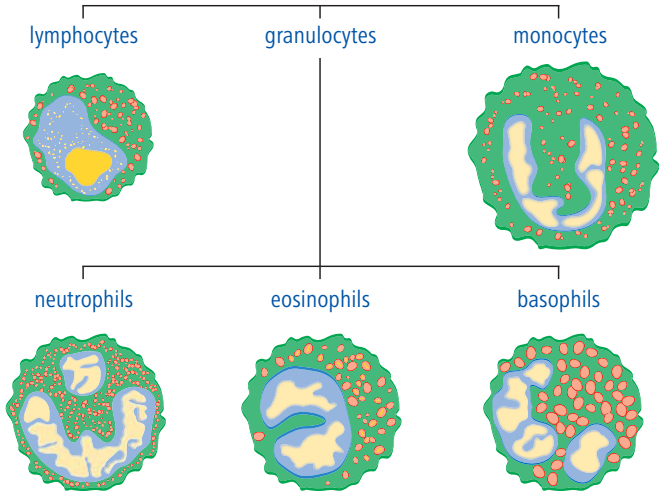
White blood cells (leucocytes)

In contrast to the erythrocytes, the white blood cells have a nucleus. They are not a single cell type but are classified into granulocytes, monocytes and lymphocytes according to their shape or the shape of their nucleus, their function, staining characteristics of the cytoplasmic granules and their site of formation.

Granulocytes and monocytes arise from bone marrow stem cells. Precursor cells of lymphocytes also arise in the bone marrow but later multiply in lymphatic organs such as the spleen and lymph nodes. Only about 5 % of the total lymphocytes in the body circulate in the blood, while the majority are stored in organs and tissues or are loosely associated with vessel walls.

Leucocytes serve for non-specific or specific defence and play a crucial part in the removal of bacteria and detritus (damaged or denatured cell and tissue substance). Their capacity for amoeba-like movement which varies according to the cell type is a precondition for them to be able to carry out their functions. When activated by chemotactic stimuli, the leucocytes can migrate from blood vessels into the surrounding area, the site of inflammation.

Classification of white blood cells (leucocytes)

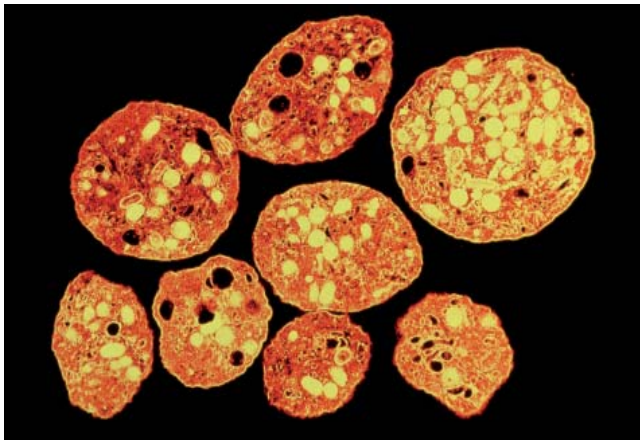


Granulocytes represent 60 – 70 % of all leucocytes. They are classified according to the staining characteristics of their granules into eosinophilic (stainable with acid eosin stains) basophilic (stainable with basic stains) or neutrophilic (neutral staining) granulocytes. The neutrophils are the largest group, accounting for about 70 % of the granulocytes. They play a major part in wound cleansing and defence against infection. Their nuclei contain a range of proteolytic enzymes which enable them to dissolve detritus (injured or denatured cell and tissue substance) and to phagocytose bacteria.

Monocytes are the largest blood cells. In the region of an injury, they leave the circulation and migrate into the injured area. There, they mature into tissue macrophages which take care of the removal of devitalised tissue by phagocytosis (elimination of large particles) or pinocytosis (elimination of dissolved material). The processes of phagocytosis and the other functions of the macrophages which have a key role in wound healing are described in detail in the chapter on "The processes of wound healing" from page 34.

Lymphocytes are globular cells with a round or oval nucleus which are capable of migration. They represent the main functioning agents of specific immunity. B lymphocytes are responsible for humoral immunity (antibodies) and T lymphocytes for cellular immunity (phagocytosis).

Non-nucleated platelets in cross section: their numerous granules can be seen clearly, containing several coagulation factors. Platelets take part in the initiation of coagulation and thrombus formation.



Platelets (thrombocytes)

Platelets are round non-nucleated discs which are produced by fragmentation of cytoplasm from giant cells of the bone marrow (megacaryocytes). They are the smallest cellular elements of the blood. Their most important function is haemostasis. They take part in the initiation of coagulation and thrombus formation. Accordingly, their numerous granules contain important blood coagulation factors (platelet factors). The coagulation processes are also described in the chapter on "The processes of wound healing" from page 34.

Wounds and wound types

A wound signifies a break in the continuity of tissues covering the body which is usually associated with a loss of substance. Deeper injuries which involve the muscle tissue, the skeletal system or internal organs are defined as complicated wounds. Wounds are distinguished into different types depending on their cause, depth and extent of the defect:

- mechanical or traumatic wounds
- thermal and chemical wounds
- ulcer wounds

Mechanical/traumatic wounds

Mechanical wounds occur as a result of a variety of forces, including the planned surgical wound.

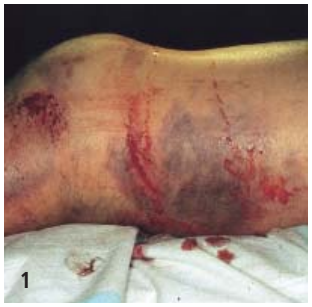
The type of trauma and the extent of damage are in turn used for further classification for prognosis and for treatment. In particular, the aetiology of the wound permits assessment as clean, dirty and/or as infected. This assessment is of fundamental importance for subsequent wound management.

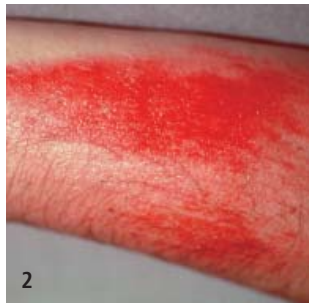
Closed wounds are characterised by injury of tissue, bones, blood vessels and nerves lying under the skin without separation of the skin taking place. Examples are closed head injuries with brain contusion, closed fractures or sprains and dislocations. Visible effects of the trauma are mainly soft tissue swelling, haematoma, and pain.

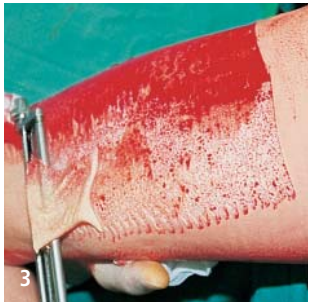
Superficial or epithelial wounds affect only the avascular epidermis. Since the epidermis is capable of regeneration, the wounds heal without scarring. The skin surface later looks no different from normal. The abrasion is an epithelial wound. Split skin graft donor sites and Reverdin graft sites should be regarded as superficial wounds.

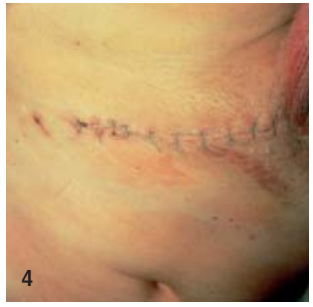
Perforating wounds occurs when the division of the skin affects the epidermis and dermis and sometimes the sub-cutaneous tissue. Examples of perforating wounds, which are also designated penetrating wounds include cuts and stab wounds, tearing, bursting and contused wounds, bites and gunshot wounds. Depending on the type of trauma, muscle tissue and internal organs may also be involved so that the boundaries between a perforating wound and a complicated wound are often fluid. The condition of the wound and tendency to heal differ considerably depending on the aetiology.

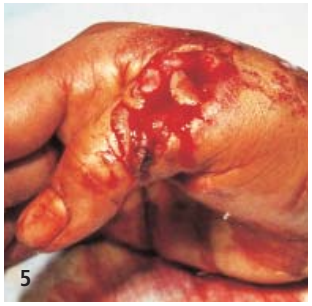
Complicated wounds such as extensive soft tissue trauma, open fractures, severe crushing with degloving, amputations and avulsion injuries can be the result of perforating and blunt force. In addition they may also be due to thermal or thermomechanical injuries. In the case of such complex patterns of injury there is also a major problem with further secondary damages. This may be due to vessel injuries with resulting ischaemia, reperfusion phenomena or compart-ment syndromes. Infections or inadequate primary care can also be further causes.

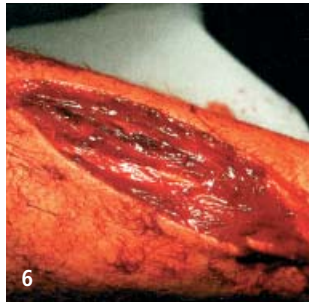


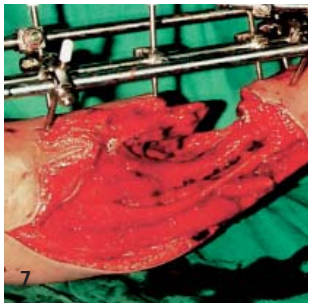


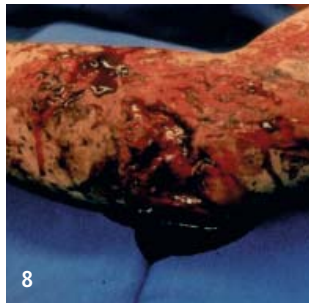












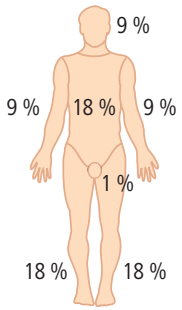
1) Haematoma in closed fracture
2) Abrasion or superficial (epithelial) wound
3) Split skin graft donor site which is classified as a superficial wound
4) Perforating wound (incised) wound as planned surgical wound
5) Crushing wound to thumb
6) Complicated wound; fracture with significant soft tissue damage
7) Complex open lower leg fracture after traffic accident with severe soft tissue damage
8) Crushing wound with extensive tissue destruction

Thermal and chemical wounds

Thermal and chemical wounds arise from the effects of heat and cold, tissue damaging rays, acids or alkalis. The skin damage depends on the severity of the impact (duration, intensity, extent). Classification of burn and cold injuries into three or four degrees of severity, respectively, aids in prognostic assessment and therapeutic planning.

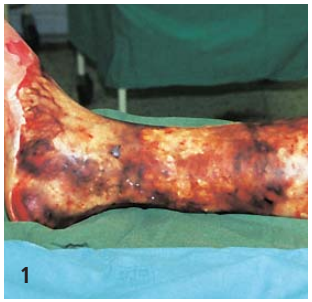
The four grades of burns are:

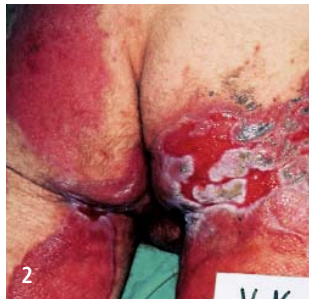
- First degree:
  functional damage to the epidermal layer (stratum corneum) manifested as erythema
- Superficial second degree:
  destruction of the epidermis down to the basal layer with formation of blisters
- Deep second degree:
  deep dermal burn affecting the epidermis and nearly the entire thickness of the dermis
- Third degree:
  necrosis with complete irreversible destruction of epidermis, dermis and often some of the subcutaneous tissue (full thickness burn)
- Fourth degree:
  carbonification involving muscles, tendons and bones. The classification fourth-degree is not normally used nowadays.

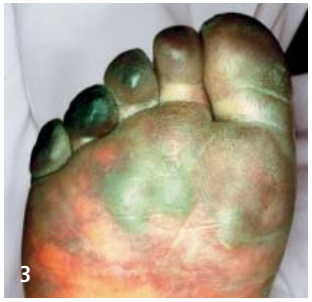


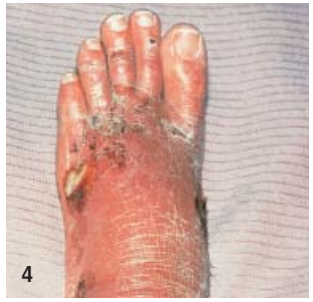
Wallace's rule of nines to measure the surface area of a burn

The degree of severity of thermal injury relates solely to the depth of the injury. However, an equally important criterion for prognosis and treatment is the area extent of the burn. Therefore, particularly in emergency situations, this is normally estimated using the "rule of nines" according to Wallace, and expressed as a percentage. Also possible is an estimate of the area by comparing it with the palm of the hands area of the burned person, which represents approximately 1 % of the body surface area. A more precise area assessment can then be made using tables in which the special features of children's body dimensions are also taken into account.









1) Third-degree burn with necroses of the epidermis, dermis and portions of the subcutis
2) Third and fourth-degree burns
3) Freezing
4) Alkali burn ("chemical burn")

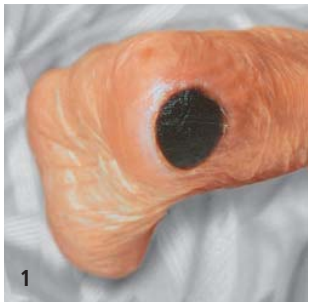
Cold injuries are also classified into four degrees of severity according to what proportion of skin is destroyed: Grade I = erythema, Grade II = blister formation, Grade III = necrosis and Grade IV = formation of thrombus and vessel obliteration.

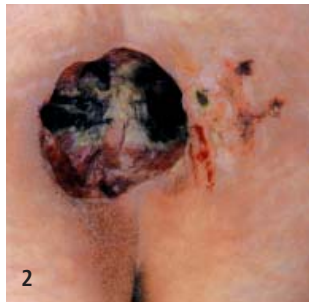
Injuries due to acids or alkalis should be classified according to their pattern of damage as burn wounds ("chemical burn"). They are classified and treated as burn wounds after neutralisation of the causative acid or alkali.

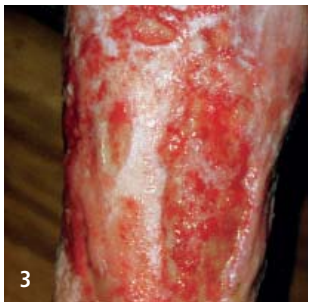
Ulcer wounds

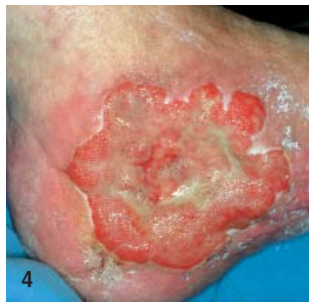
A further group of wounds presenting particular healing problems is the ulcera, commonly known as ulcers. In contrast to acute wounds, they usually occur as a result of local disorders of nutrition in the skin, caused by venous, arterial or neuropathic vascular damage or prolonged local pressure or radiation. However, an ulcer can also be a symptom of a systemic disease, e.g. as a result of certain tumours, infectious skin diseases or haematologic disorders. According to the severity of the trophic disorder, the damage can affect all skin layers and extend as far as bone.

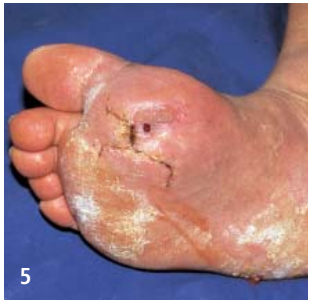
Ulcer wounds usually require more than 8 weeks for healing and are therefore classified by definition as chronic wounds. The most important chronic wound conditions are described under this classification from page 96.

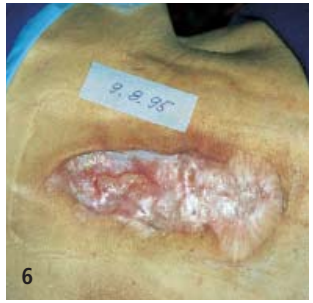








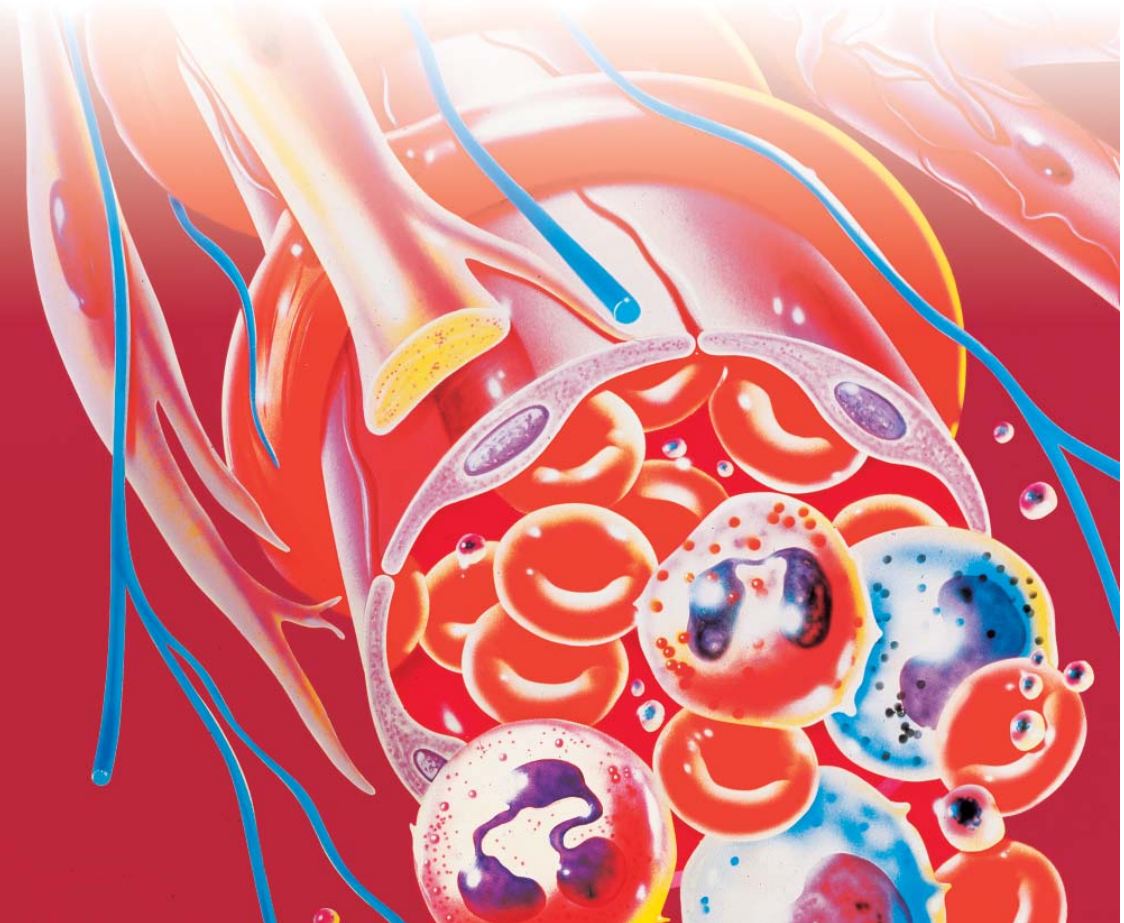




1) Decubitus ulcer on heel with closed cap of necrosis
2) Sacral decubitus ulcer with necrosis and wound pockets
3) Venous leg ulcer which has involved the entire lower leg, a so-called gaiter ulcer
4) Leg ulcer, caused by a basal cell carcinoma
5) Diabetic ulcer (mal perforans)
6) Radiation ulcer

The processes of wound healing

The regeneration of epithelium and, in particular, the demanding repair of skin connective tissue, are biologically and chronologically well organised cooperative efforts involving a variety of blood, immune system and tissue cells. These drive the healing process forward step-by-step in a variety of wound-healing phases.



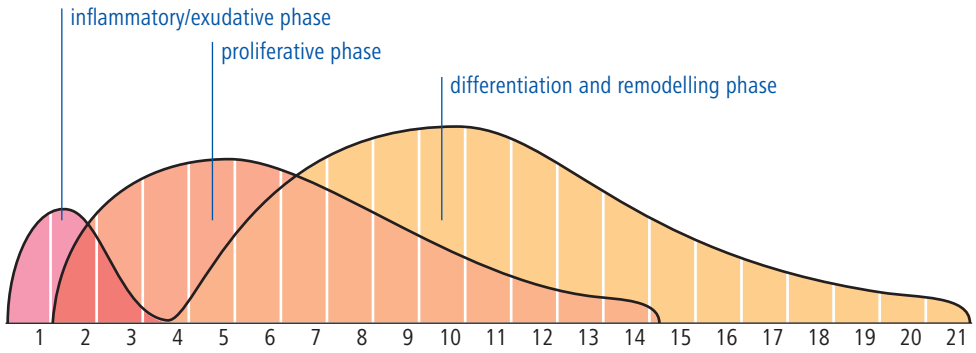
The phases of wound healing

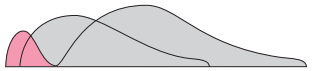
Irrespective of the type of wound and the extent of tissue loss, the healing of every wound takes place in phases which overlap in time and cannot be separated from one another. The division into phases refers to the fundamental morphological alterations in the course of the repair process without reflecting the actual complexity of the process. The usual division is into three or four wound healing phases; the system of three basic phases will be used for the following descriptions:

- inflammatory or exudative phase for blood clotting and wound cleansing
- proliferative phase for production of granulation tissue
- differentiation phase for maturing, scar formation and epithelialisation

In clinical practice, the three phases of wound healing are also, for short, called the cleansing, granulating and epithelialising phases.

Schematic representation of the temporal course of the wound healing phases:





The inflammatory/exudative phase

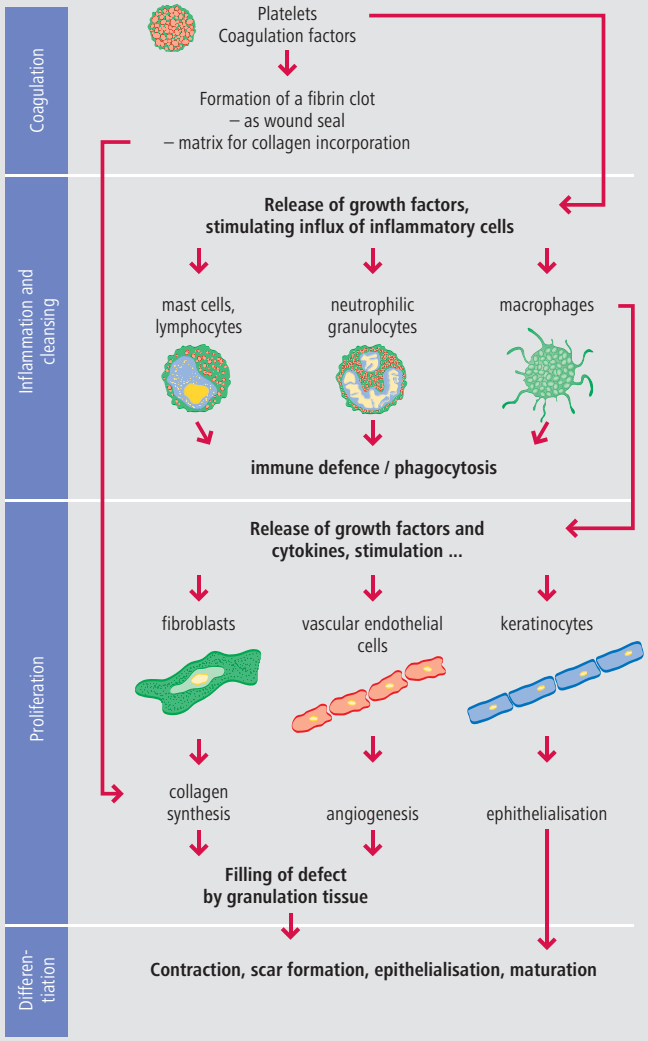
The inflammatory/exudative phase begins at the moment of injury and lasts about three days under physiological conditions. The first vascular and cell reactions lead to haemostasis and are complete after about 10 minutes.

Vasodilatation and increased capillary permeability result in an increase in the exudation of plasma into the interstitium. This promotes the migration of leucocytes into the wound area, especially neutrophilic granulocytes and macrophages. It is their job to defend against infection and to cleanse the wound by phagocytosis. At the same time, they release biochemically active mediator substances, by means of which cells which are important for the next stage are activated and stimulated. The macrophages play a key role in that process. Their presence in adequate numbers is crucial to the progress of wound healing.

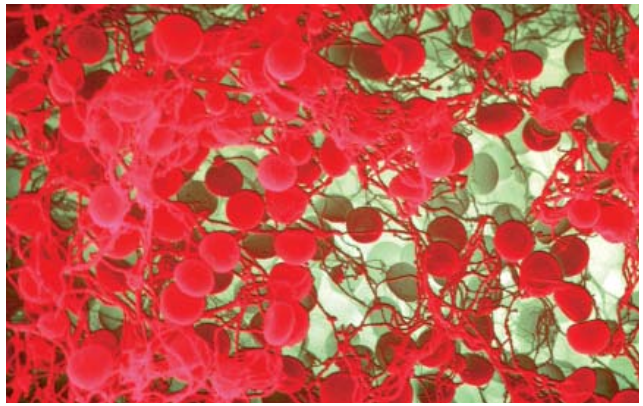
Haemostasis

The first goal of the repair process is to stop bleeding. Vasoactive substances are released from the injured cells, producing narrowing of vessels (vasoconstriction) to avoid blood loss until the vessel can be first sealed by platelet aggregation. The platelets circulating in the plasma adhere to the damaged vessels at the site of injury and form a plug which first seals the vessels provisionally.

The complex process of platelet aggregation activates the coagulation system in order to seal the site of injury permanently. The coagulation cascade takes place in steps in which about 30 different factors participate. This leads to the formation of an insoluble fibrin mesh from fibrinogen. A clot is produced which seals the wound and protects against further bacterial contamination and fluid loss.



Sequence of physiological wound healing. In the ideal case, the tissue missing from a wound is replaced by functional scar tissue by various interdependent processes such as blood coagulation, inflammation and breakdown of devitalised tissue, new vessel formation, formation of granulation tissue, epithelialisation and maturation. The chronologically correct appearance of the participating cells is essential for the wound healing cascade to take place in a regular fashion. If there is a disorder of only one of these partial steps, the entire wound healing process can be influenced.



Blood clot consisting of platelets, red blood cells and fibrin strands

In order not to endanger the whole organism by spreading thrombotic processes, platelet aggregation and the coagulation processes must remain restricted to the site of injury. In flowing blood, the coagulation process is therefore countered constantly by substances of the fibrinolytic (clot-dissolving) system.

Inflammatory reactions

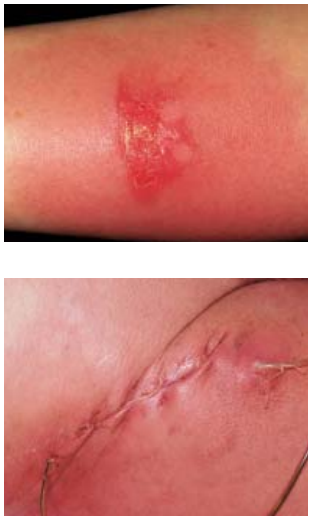
Inflammation represents the complex defence reaction of the organism against the effects of a variety of noxious agents either mechanical, physical, chemical or bacterial origin. The goal is to eliminate or inactivate the noxious agent, cleanse the tissue and provide conditions for the subsequent proliferative processes.

Inflammatory reactions are thus present in every wound, even a closed wound with an intact skin surface. However, they are increased in the case of open skin wounds, which are always contaminated by bacteria.

Inflammation is characterised by the four classical symptoms of infection, which are: erythema (rubor), warmth (calor), swelling (tumor) and pain (dolor). The arterioles which constrict briefly at the start of the injury subsequently dilate due to vasoactive substances such as histamine, serotonin and kinin. This leads to increased perfusion within the wound area and therefore an increase in the local metabolism required to eliminate the noxious sources. The process is apparent clinically as erythema and warmth.

The vasodilatation at the same time leads to an increase in vascular permeability with increased exudation of plasma into the interstitium. The first phase of exudation takes place about 10 minutes after the injury has occurred, and a second phase occurs one to two hours later. Oedema, which is apparent as swelling, develops due to the slowed circulation and also to the localised acidosis in the wound area. Today, it is assumed that the local acidosis increases catabolic processes and that the toxic breakdown products of tissue and bacteria are diluted by the increase in tissue fluid.

Pain develops not only as a result of exposed nerve endings and swelling but also due to a number of inflammatory mediators such as bradykinin. Severe pain can result in a limitation of function (functio laesa).



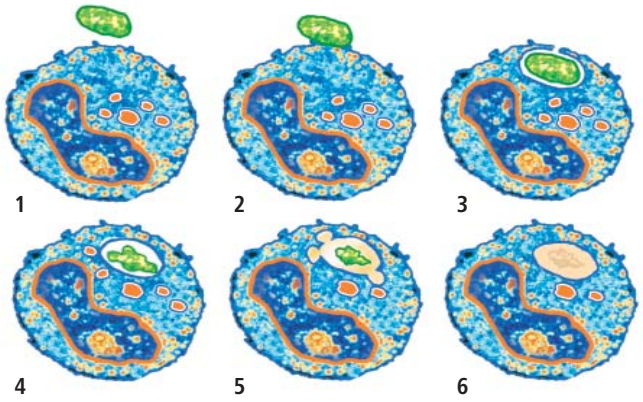
Inflamed wounds with the clearly visible symptom of reddening; above, a burn wound; below, an operation scar following a vascular operation.

Phagocytosis and immune response

About 2 – 4 hours after the injury, as part of the inflammatory reaction, inward migration of leucocytes commences. These are now called phagocytes and are capable of phagocytosing debris, foreign material and micro-organisms.

In the initial inflammatory phase, neutrophilic granulocytes predominate, secreting various inflammation-promoting messenger substances, the so-called cytokines into the wound. These cells also phagocytose bacteria and release proteolytic enzymes (proteases) which remove damaged and devitalised constituents of the extracellular matrix. This signifies the first cleansing of the wound.

About 24 hours later, monocytes migrate into the wound area in the train of the granulocytes. These then differentiate into macrophages and continue the phagocytosis. They also intervene crucially in the process by secreting cytokines and growth factors.



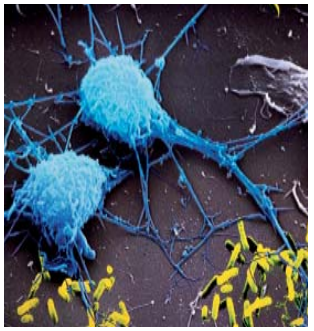
Course of phagocytosis:
After opsonisation of the foreign body, the phagocyte moves deliberately to the foreign body (1) and adhesion occurs (2). In the next stage, the phagocyte encloses the foreign body with pseudopodia (3). By further merging of the pseudopodia (4), a vacuole (phagosome) is produced which merges with lysosomes to form phagolysosomes (5) in which "digestion" of the foreign body can then take place (6).

Leucocyte migration continues for a period of about 3 days until the wound is "clean" and the inflammatory phase is approaching its end. However, if infection occurs, the leucocyte migration persists and phagocytosis is increased. This leads to a prolongation of the inflammatory phase and thus to a delay in wound healing.

The detritus-laden phagocytes and dissolved tissue form pus. The killing of bacterial material inside the phagocytes can only take place with the assistance of oxygen which is why an adequate oxygen supply in the wound area is of paramount importance for the immune response.

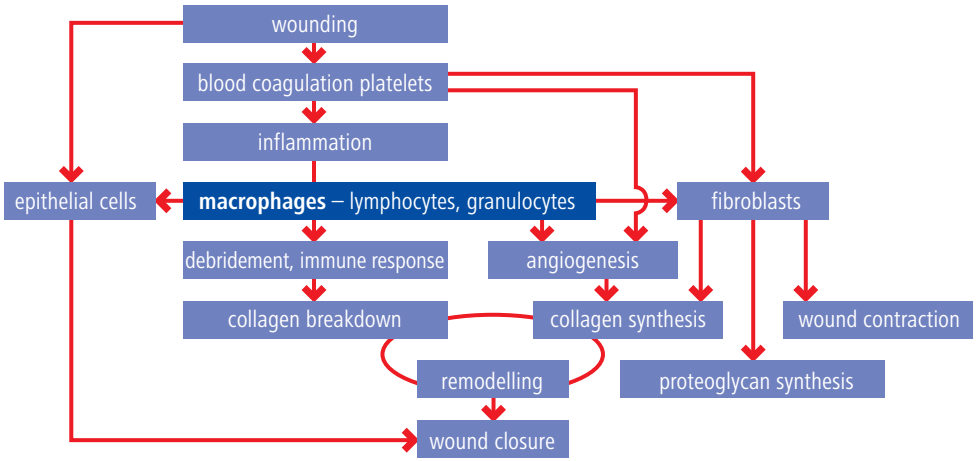
The dominant role of macrophages

It is now believed that wound healing is not possible without functioning macrophages. The majority of the macrophages have their origin in haematogenous monocytes whose differentiation and activation to macrophages take place in the wound area. Attracted by the chemotactic stimulus of bacterial toxins and further activation by neutrophilic granulocytes, the cells migrate in dense rows from the circulating blood into the wound. During their phagocytic activity, which represents the highest degree of activation of the cells, the macrophages are not limited only to a direct attack on micro-organisms but they also assist in presentation of antigen to the lymphocytes. Antigens captured and partially constructed by macrophages are presented to the lymphocytes in a recognisable form.



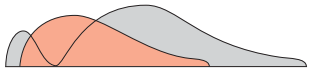
Macrophages during the phagocytosis of E. coli bacteria

Furthermore, macrophages secrete inflammation-promoting cytokines (interleukin-I, IL-II and tumour necrosis factor α, TNF-α) as well as various growth factors (BFGF = basis fibroblast growth factor, EGF = epidermal growth factor,



The role of macrophages in wound healing

PDGF = platelet-derived growth factor and TGF-α and -β = transforming growth factor α and β). These growth factors are polypeptides which influence the cells involved in wound healing. They attract cells and promote their influx into the wound area (chemotaxis), subsequently stimulating the cells to proliferate.



The proliferative phase

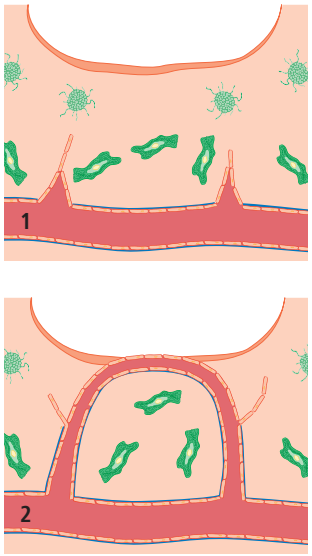
During the second phase of wound healing, cell proliferation predominates aimed at new vessel formation and filling of the defect by granulation tissue. The phase begins on approximately the 4th day after trauma, but the conditions for tissue growth were created in the inflammatory exudative phase: Fibroblasts from the surrounding tissue migrate into the clot and fibrin mesh formed during coagulation, using it as a provisional matrix for incorporation of collagen. The released cytokines and growth factors stimulate and regulate the migration and proliferation of the cells responsible for the formation of new tissue and blood vessels.

New blood vessel formation and vascularisation

Without new vessels which will ensure an adequate supply of blood, oxygen and nutrients to the wound area, wound healing can not proceed. New vessel formation starts from intact blood vessels at the edge of the wound.

Through stimulation by growth factors, the cells of the epithelial layer lining the vessel walls (here called endothelium), are capable of breaking down their basement membrane, mobilising and migrating to the surrounding wound area and into the blood clot. By means of further cell division, they create a tube-shaped structure which continues to divide at its budlike end. The individual vascular buds grow towards one another and combine into capillary loops which again continue to branch until they encounter a larger vessel into which they can empty. However, circulating endothelial stem cells were recently discovered in the blood which may call into question the current teaching.

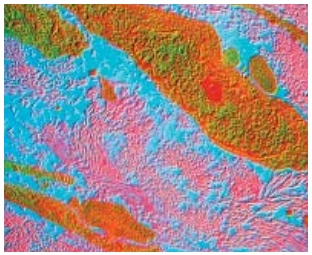
A well-perfused wound is extremely rich in vessels. The permeability of newly formed capillaries is also higher than that of other capillaries which takes account of the increased metabolism in the wound. However, the new capillaries are not very resistant to mechanical stresses which is why the wound area has to be protected against trauma. With the maturing of the granulation tissue to scar tissue, the vessels regress.



The model of angiogenesis: breakdown of the basement membrane of intact blood vessels by various substances, with resulting release of endothelial cells, formation of vascular buds by cell division (1), which develop further into capillary loops (2).

Granulation tissue

Depending on the time of new vessel formation, filling of the defect with new tissue begins on about the 4th day after the occurrence of the wound. So-called granulation tissue develops, the formation of which is initiated primarily by the fibroblasts. They produce collagen which matures into fibres outside the cells and gives the tissue its strength. They also produce proteoglycans which form the gel-like ground substance of the extracellular space.



Electronmicrograph of a fibroblast which, as the most important cell type of the skin connective tissue, is responsible for the synthesis and secretion of collagen, elastin and proteoglycans.

Fibroblasts

The spindle-shaped fibroblasts are not transported into the wound with the blood circulation but migrate mainly from the local tissue which has been injured. They are attracted by chemotaxis. Amino acids serve as substrates, supplied by the breakdown of the blood clot by the macrophages. At the same time, the fibroblasts utilise the fibrin mesh derived from the blood clot as a matrix for the ingrowth of collagen.

The close relationship between fibroblasts and the fibrin mesh led in the past to the assumption that the fibrin is changed into collagen. In fact, however, the fibrin mesh is broken down as incorporation of collagen progresses and the sealed vessels are recanalised. This process, controlled by the enzyme plasmin, is called fibrinolysis.

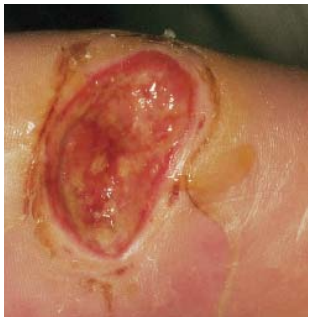
Fibroblasts thus migrate into the wound area when amino acids from the dissolved blood clots are available and necrotic tissue has been cleared away. However, if haematomas, necrotic tissue, foreign bodies or bacteria are present, both the growth of new blood vessels and fibroblast migration are delayed. The extent of the granulation formation corresponds directly with the extent of the blood coagulation and the inflammatory process and the body's own wound cleansing process with the help of phagocytosis.

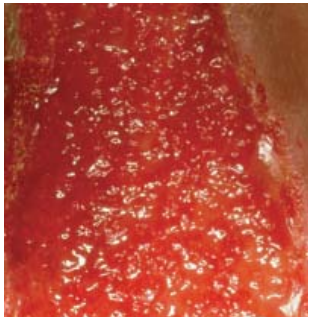
Even if fibroblasts are regarded as a "uniform cell type", it is especially important for wound healing that they differ in function and reaction. Fibroblasts of various ages are found in a wound, differing both in their secretory activity and also in their reaction to growth factors. In the course of wound healing, some of the fibroblasts change into myofibroblasts in order to produce contraction of the wound.

Features of granulation tissue

Granulation tissue may be called a temporary primitive unit of tissue or an organ which "finally" closes the wound and serves as a "bed" for the ensuing epithelialisation. After fulfilling its tasks, it is gradually changed into scar tissue.

The name granulation was introduced by Theodor Billroth in 1865 and is derived from the fact that the developing tissue exhibits a surface of bright red, glassy-transparent granules. A vascular branch corresponds to each of these granules with numerous fine capillary loops as they develop due to the new vessel formation. The new tissue is deposited beside the loops. When there is good granulation, the granules increase in size with time and also increase in number so that finally a salmon red moist shiny surface is formed. Such granulation indicates good healing. Stagnating healing pro-cesses are present if the granulation is covered with sloughy deposits, looks pale and spongy or has a bluish tint.





The structure of the granulation tissue is an important indicator in assessing the healing tendency and the quality of wound healing. The above illustration shows spongy granulation tissue where wound healing is inadequate; the fresh red granulation (below), on the other hand, is a sign of a good healing process.

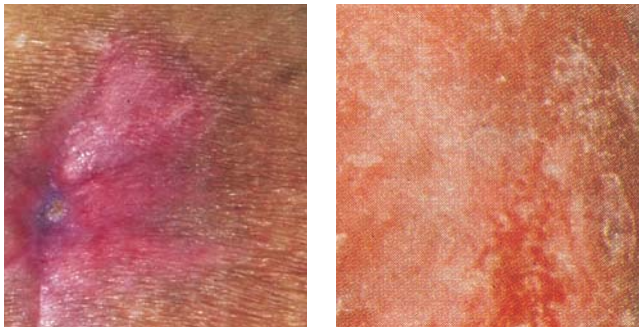
The differentiation and remodelling phase

Maturing of the collagen fibres begins between about the 6th and 10th days. As the wound contracts, the granulation tissue contains less water and fewer vessels, becomes continuously stronger and organises into scar tissue. Epithelialisation then concludes wound healing. This process consists of the formation of new epidermal cells by mitosis and cell migration, mainly from the edge of the wound.

Wound contraction

Wound contraction brings the undestroyed tissue substances closer so that the area of "incomplete repair" is kept as small as possible and wounds close spontaneously. Wound contraction is aided by mobility of the skin.

Contrary to the earlier belief, that wound contraction takes place by shrinkage of the collagen fibres, it is now known that this shrinkage plays a subordinate role. It is rather the fibroblasts of the granulation tissue which are responsible for the contraction. After these fibroblasts have completed their secretory activity, they change partly into fibrocytes (the resting form of fibroblasts) and partly into myofibroblasts. The myofibroblast resembles the cells of smooth muscle and like them contains the contractile muscle protein actomyosin. The myofibroblasts contract pulling the collagen fibres taut at the same time. This makes the scar tissue shrink and pulls the skin tissue together at the wound margin.



Wound closure by clearly visible contraction and epithelialisation (left), epithelial margins still fragile (right)

Epithelialisation

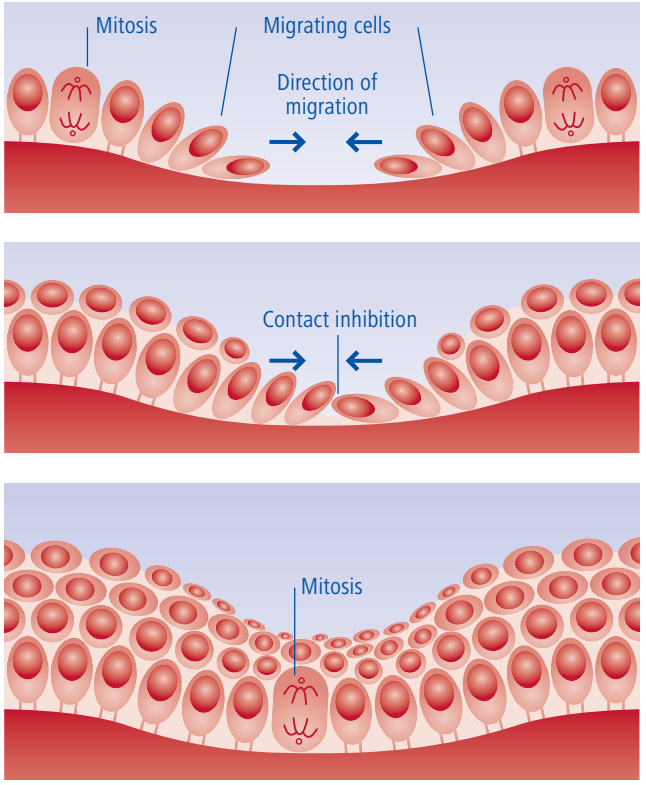
Epithelialisation of the wound marks the conclusion of healing with the processes of epithelialisation being closely linked to the formation of wound granulation. Granulation tissue sends chemotactic signals for the migration of marginal epithelia on the one hand, while, on the other hand, the epithelial cells require a moist sliding surface for their migration.

Re-epithelialisation is also a complex process which is based on an increased rate of mitosis in the basal layer of the epidermis and the migration of new epithelial cells from the wound edge.

Mitosis and migration

The metabolically active cells of the basal layer which are capable of the wound healing reaction obviously have an unlimited potential for mitosis which is normally held in check by tissue-specific inhibitory substances, the so-called chalones, but which becomes fully active in the event of injury. Thus, if the extracellular chalone level falls greatly after an injury of the epidermis, as a result of the loss of numerous chalone-producing cells in the wound area, there is a correspondingly high mitotic activity in the cells of the basal layer. This initiates the cell multiplication required to cover the defect.

Cell migration also has its special features. Where as during physiological maturing of the epidermis, the cells migrate from the basal layer to the skin surface, the reparative cell replacement takes place by advancement of the cells in a linear direction towards the opposite wound edge. Epithelialisation from the wound edge starts with the break in continuity of the epidermis. The separated epithelial cells creep towards one another by active amoeboid movements, which are reminiscent of the ability of single-celled organisms, thus attempting to close the gap. These amoeboid movements however can occur only in slitlike superficial wounds. In all other injuries of the skin, the migration of the epithelia at the wound edge is linked to the filling of the tissue defect by granulation tissue. Epithelial cells do not descend into hollows or wound craters but require a smooth moist creeping surface.



Schematic diagram of re-epithelialisation by cell division and cell migration. The epithelial cells creep towards one another on the slippery surface of the granulation tissue. When the defect is closed, the epithelial cells become heaped up so that the epithelial cover becomes stronger.

Migration of the marginal epidermis cells does not take place uniformly or inexorably but probably takes place intermittently, depending on the structure of the wound granulation in each case. The first outgrowth of the marginal epithelium is followed by a phase of thickening of the initially single-layered epithelial cover by a heaping up of the cells. In addition, the epithelial layers are soon several layers thick and are stronger and denser.

Features of re-epithelialisation

Only superficial abrasions of the skin heal according to the pattern of physiological regeneration, and the regenerated tissue is accordingly complete and uniform. All other skin wounds replace the lost tissue, as described already, by cell migration from the edge of the wound and from preserved skin appendages. The result of this re-epithelialisation does not represent a complete skin replacement but a thin substitute tissue which is poor in blood vessels and which lacks important constituents of the epidermis such as glands and pigment cells, and adequate innervation.

Quantitative classification of wound healing

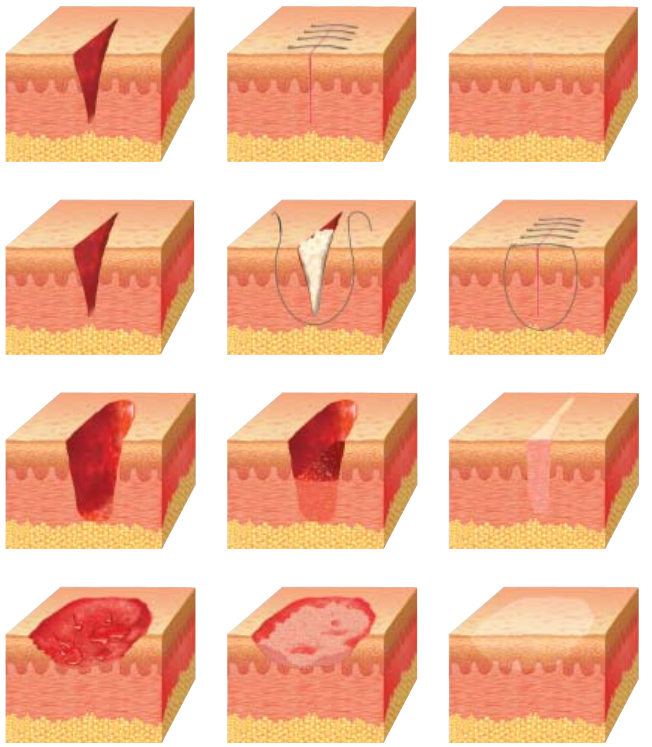
Since the time of Galen (A.D. 129 – 199), wound healing has been classified into healing by primary intention and by secondary intention. The word "intention" in Galen's understanding does not refer to the physiological nature of the healing processes but to the intention of the physician to achieve primary wound healing with wound edges close together and with few gaping wound edges. The classification thus is above all of quantitative significance (more replacement tissue has to be produced in the case of secondary healing) and is important in prognosis. In order to take into account the therapeutic problems which result from the extent and type of the tissue destruction, a further classification is made today into primary delayed healing, regenerative healing and chronic wounds.

Primary wound healing (per primam intentionem)

The conditions for wound healing are more favourable the less tissue has been damaged. The prospects of healing are best in the case of a smooth, closely apposed wound surfaces of an incised wound with negligible loss of substance and without inclusion of foreign bodies in a well-perfused area of the body. Primary wound healing occurs in such cases if there is no wound infection.

Healing by primary intention usually occurs after surgical incisions or accidental wounds made by sharp-edged objects. With a respective type of less tissue destruction by other mechanisms (e.g. tearing or bursting wounds), surgical debridement may create the conditions for primary healing.

Wounds capable of primary healing are closed with sutures, staples or wound closure strips. During blood coagulation, fibrin ensures temporary loose adhesion of the wound surfaces while the phase of inflammation and exudation takes place almost unnoticed. The repair processes supervene, characterised by the migration of fibroblasts, the formation of ground substance and incorporation of collagen fibres. Numerous newly sprouting capillaries feed the young connective tissue and restore the connection with the circulation. Both wound surfaces are firmly united after about 8 days. However, the wound only attains its final tensile strength in the course of several weeks. The result of the primary healing is a narrow linear scar which is initially red



Primary wound healing of non-infected, closely apposed wound surfaces

Delayed primary healing when the wound is at risk of infection

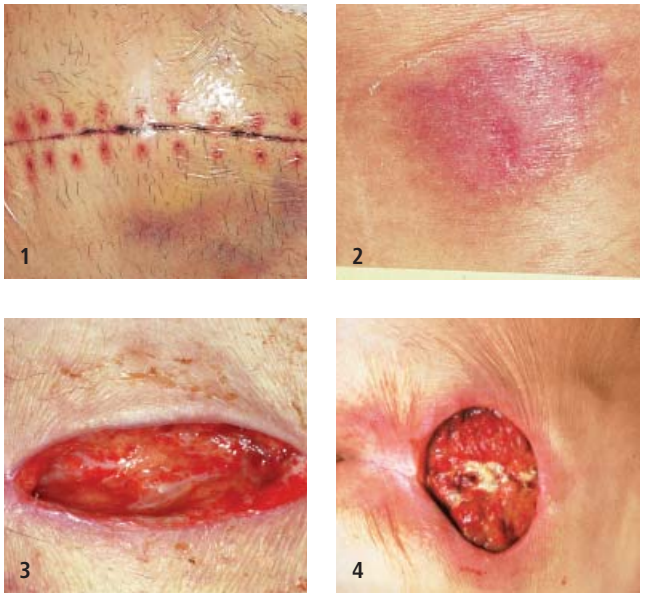
Secondary wound healing with filling of the defect by granulation tissue which changes to scar tissue in the course of healing

Regenerative or epithelial healing of injuries affecting only the epidermis

due to the large number of capillaries. Later it becomes lighter in colour as the number of vessels is reduced. Finally, the scar is whiter than the surrounding normal skin.

Delayed primary healing

Delayed primary healing occurs when an infection is anticipated. In this case the wound should not be closed with sutures or wound closure strips. To observe for the development of infection, the wound is loosely packed and held open. If no infection occurs, the wound can be sutured approximately between the 4th and 7th days. It then heals by primary intention. If an infection becomes apparent, the wound is classified as healing secondarily and receives treatment as for an open wound.



Examples of the different types of wound healing:
1) Primary wound healing, in the case of closely apposed wound edges, which is normally possible with surgically produced wounds.
2) Regenerative or epithelial healing, whereby the healed tissue hardly differs from the original condition.
3) Secondary healing with tissue formation – here after dehiscence of a thoracic wound. A secondary suture is made following appropriate wound conditioning.
4) Chronic healing process in the case of a sacral decubitus ulcer which, with the requisite formation of granulation tissue, corresponds to secondary wound healing.

Secondary wound healing (per secundam intentionem)

Secondary wound healing always occurs when tissue gaps have to be filled or when a purulent infection prevents direct union of the wound edges. The wound edges are now no longer closely apposed but gape. To close the wound, granulation tissue must be formed (as already described). The energy requirement of the organism is therefore greater than in the case of primary healing and likewise the formation of the granulation tissue is more subject to endogenous and exogenous influences.

Regenerative healing

Regeneration is defined as the replacement of destroyed cells or tissues by others of equal value and is only possible for cells which retain their capacity for mitosis. These include the basal layer of the epidermis. If only the epidermis is damaged by an injury, e.g. in a skin abrasion wound, it heals without a scar. The healing processes correspond to the wound healing phase of re-epithelialisation.

Chronic wound processes

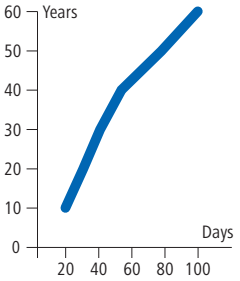
The chronic wound is one undergoing secondary healing which has to be closed by formation of tissue. If this process requires more than eight weeks, the wound is classified as chronic. The change from an acute to a chronic wound can occur in each of the wound healing phases. However, the majority of chronic wounds develop from progressive tissue destruction because of vascular disease from various causes such as disturbed blood circulation of arterial or venous origin, diabetes mellitus, local pressure injuries, radiation damage or tumours.

Influences on wound healing

The ability of the human organism to heal wounds is subject to great individual variation. How quickly and how well a wound heals depends on the general bodily constitution of the affected organism, on the aetiology of the wound and on the resulting specific circumstances.

Systemic influences

General influencing factors depend on the individual physical status of the person affected. Their relevance for the course of healing is very variable and some "influences" are themselves the cause of the wound.



Healing of a 20 cm² wound according to the person's age

Patient age

Clinical research suggests that physiological aging delays wound healing through the general reduction in cell activities which can lead to a loss of quality in wound healing. However actual disorders of wound healing result mainly from the effects of age-related co-morbidity such as poor immune status or deficient nutrition. Ulcer wounds as a result of metabolic disease, vascular disease and tumour inevitably also occur more frequently. Then a correspondingly poor healing tendency can be expected.

Nutritional status

Wound healing is impaired if the nutrients and nutrient components required for the increased wound metabolism (proteins and calories, vitamins and minerals) are not available in sufficient quantities. If, for example, not enough protein is provided, protein synthesis is interrupted and with it, the proliferation of granulation tissue cells, and of other cells of the immune defence system. A protein deficiency therefore impairs all the processes of wound healing, without exception.

Diseases, particularly infections and chronic ulcers, switch the metabolism via cytokine production to a catabolic state, which in turn leads to malnutrition. The body then has a shortage of nutrients available for energy production and good wound healing.

All vitamins, by their properties as coenzymes, positively influence wound healing and the deficiency of only a single vitamin may delay healing. Vitamins of the B complex, for example, participate in collagen synthesis and stimulate antibody formation and infection defence. Vitamin A also participates in collagen synthesis and cross-linking. Antioxidants such as vitamins E and C capture the free radicals that are toxic and therefore harmful to the epithelial cells. Furthermore, vitamin C plays a key role in the formation of collagen, as well as being significant for the formation of the intercellular substance, the basal membranes of vessels, complement factors, and gamma globulins.

Of the minerals, it is above all zinc and iron deficiencies that cause disruptions. Zinc is a central component of the so-called metalloenzymes and has significant biological

effects in the organism, which also extend to wound healing. Iron deficiency leads to anaemia and therefore hinders oxygen transport to the wound area.

Malnutrition, possibly leading to cachectic conditions, can often be observed in severely ill, multimorbid and elderly persons. It can be caused by diseases such as tumours, infectious diseases, organ disorders and severe pain. Nutritional causes such as inadequate intake of nutrition or disorders of absorption often play a major part.

Immune status

The processes of the immune response play an important role during wound healing. Accordingly, impairment or deficiencies of the immune system cause increased susceptibility to disorders of wound healing and infection. Acquired immune deficiencies can result from operative trauma, parasitic, bacterial or viral infections and also occur after periods of malnutrition, extensive burns, injury with ionising radiation, entero- or nephropathies and cytostatic immunodepressant treatment.

Underlying diseases

Diseases with an inhibitory effect on wound healing are again mainly those which impair the immune status of the affected organism such as tumours, autoimmune diseases and infections. Delayed or abnormal healing can be anticipated also in the case of connective tissue diseases (e.g. rheumatoid disease), metabolic disease (e.g. diabetes mellitus) and vascular diseases (e.g. peripheral vascular disease, venous insufficiency). Diabetes mellitus and arterial/venous vascular disease, in particular, are themselves the cause of ulceration.

Important systemic and local influences on wound healing

General (systemic)	Local
▪ Age ▪ Nutritional status ▪ Immune status ▪ Underlying illnesses ▪ Postoperative complications ▪ Acute traumas ▪ Medications ▪ Psychosocial situation	▪ Extent of injury (size, depth, etc.) ▪ Condition of wound bed (pus, necrosis, etc.) ▪ Condition of wound edges (smooth, jagged) ▪ Bacterial colonisation, contamination, infection ▪ Site of wound ▪ Age of wound ▪ Quality of wound management ▪ Surgical conditions and circumstances

Postoperative complications

Numerous postoperative complications have a direct effect on wound healing: thrombosis and thromboembolism, possibly due to the increased fibrinolytic activity, postoperative pneumonia, postoperative peritonitis, postoperative ileus and postoperative uraemia. The “intoxication” with degradation products before they are excreted in the urine apparently has an inhibitory effect on the course of healing.

Affects of acute trauma/shock

Trauma associated with blood loss or major fluid loss, such as in severe burns, causes a large number of mediator-induced reactions which can lead to a disorder of the microcirculation. Subsequent tissue hypoxia, increased capillary permeability and clinically detectable abnormal perfusion develop as symptoms of shock. The resulting imbalance between oxygen requirement and supply and the delay in removal of products of metabolism particularly affects the initial phase of wound healing and the immune response.

Medications

Different drugs have a direct negative effect on wound healing, especially immune suppressant, cytostatic, anti-inflammatory (mainly glucocorticoids) and anticoagulant agents. Depending on the inhibitory effect of the various drugs on coagulation, inflammatory processes and proliferation, formation of granulation and scar are particularly affected so that the wound's resistance to tearing can be expected to be reduced. However, the effects on the repair mechanism of the tissue depend on the dosage, the time of administration and the duration of therapy.

Patient's psychosocial situation

Wound healing, especially healing of chronic wounds of metabolic origin such as diabetic ulcers, require a large degree of collaboration of the patient. The individual's psychosocial situation, however, can create a wide variety of different starting conditions with regard to the patient's willingness and motivation for cooperating in the treatment. Above all, the number of elderly patients with chronic wounds who also suffer from dementia is constantly increasing, so that adequate compliance is no longer assured with these individuals. Self-harming tendencies also have to be taken into account.

Moreover, alcohol and nicotine abuse as well as illegal drugs may also have negative effects on wound healing. Apart from the vascular injury component of drug abuse (arteriosclerosis, severe perfusion abnormalities) this group of patients is often in poor general condition with reduced immune responses and a poor nutritional status.

Local influences

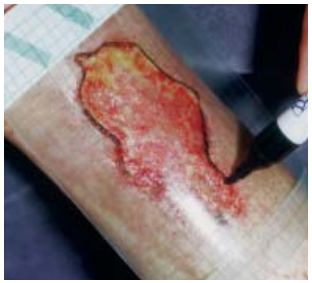
The condition of the wound and the quality of the wound management are other important influences on wound healing.

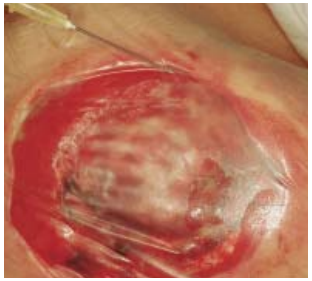
Condition of wound

A range of factors must be considered when assessing the condition of the wound and the resulting consequences for wound healing:

- aetiology/extent of injury (size, depth, involvement of deeper structures such as fascia, muscle, tendons, cartilage, bone)
- condition of wound edges (smooth, irregular, jagged, undermined, with pockets)
- condition of the base of the wound (proportion of necrotic tissue, composition of the necrotic tissue: closed black necrosis, crust, sloughy tissue, dirty, presence of foreign body, clean)
- composition of exudate (bloody, serosanguinous, purulent, dried out)
- extent of bacterial colonisation/signs of infection (see also chapter on "Wound infection")
- site of wound (in well or poorly perfused region)
- age of wound (acute trauma, time elapsed from injury to first aid/treatment, chronic wound conditions)

In the case of surgical wounds, the local influences include type of operation, site of operation, duration and type of operative preparation, and the quality of hygiene management in the operating theatre, operative techniques and duration of the operation.





Two methods for determining the size and volume of a wound: With a wound of large area (above), lay a transparent foil over it and mark the wound outline with a felt-tipped pen and calculate the area. The size and volume of a wound may be determined by measuring the fluid-holding capacity (below), in that the wound is covered with foil and sterile fluid injected into it. The quantity of fluid in ml. or cc. represents the wound volume.

Quality of wound treatment

The quality of wound management has a significant influence on wound healing. Depending on the type and cause of the wound, wound management requires a variety of therapeutic measures. These include surgical procedures to treat acute trauma and complex causal therapies to control chronic wound conditions or correct dressing treatment. Good wound management involves many medical disciplines and frequently the success in wound treatment is only possible with interdisciplinary collaboration.

The principles of wound treatment in acute (from page 80) and chronic (from page 96) wounds are described in the corresponding chapters.

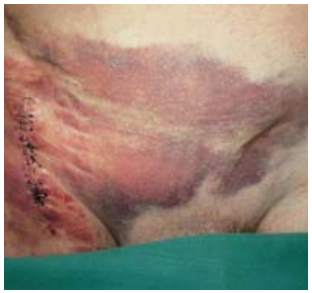
Disorders of wound healing

Disorders of wound healing occur in different manifestations and to a varying extent due to the effects of one or more of the influences described above. The more severe disorders arise from stagnating wound cleansing, poor quality or delayed formation of granulation tissue, absence of re-epithelialisation along with typical post-operative complications (seromas, haematomas, wound dehiscence and hypertrophic scar formation) and wound infections.

Seromas

Seromas are hollow spaces in the wound area in which blood, serum or lymph collects. They occur due to irritation in the wound area, e.g. caused by foreign bodies, coagulation necroses by excessive use of electrocoagulation or mass ligatures, but also in the case of tension in the wound while having tight sutures, or subclinical infection. Transudates during protein deficiency, generalised illnesses or impaired lymph flow are further causes.

Smaller seromas can be aspirated with a syringe, but a formal wound revision is necessary for larger ones. The wound is opened at the sutures and explored. Persistent lymph channels should be coagulated with diathermy. A Redivac drain is inserted and should only be removed when the skin has attached firmly to the underlying tissue. One complication can be that the primarily sterile seromas become infected due to the stagnant fluids which favour bacterial growth. Those seromas must then be treated as abscesses.

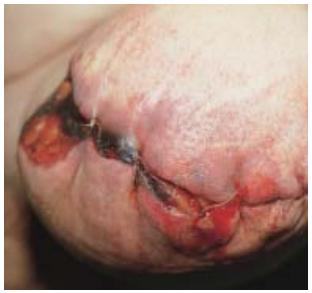


Extensive wound haematoma

Wound haematomas

Wound haematomas form in the wound crevice as a result of inadequate haemostasis of the vessels opening into the wound region. Postoperative rise in blood pressure and reperfusion of collapsed vessels are other causes. They often occur when coagulation is impaired due to anticoagulant therapy or in the event of pathological deficiencies in the coagulation system.

The clinical signs of a secondary haemorrhage are increased respiratory and pulse rate, fall in blood pressure, local swelling. A blood count and coagulation screen should be performed with monitoring of vital signs. For small haematomas, the application of ice and aspiration may be sufficient to limit them. Larger haematomas must be evacuated as potential foci of infection. The revision is usually undertaken in the region of the previous skin incision. All coagulum must be removed. After irrigation with Ringer's solution, a Redivac drain is inserted and the wound is closed.



Necrosis of wound edge in the area of suture of an amputation stump

Soft tissue necrosis

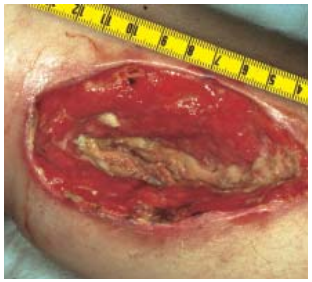
Soft tissue necrosis occurs when the supply of nutrients to the wound edges or soft tissues is reduced or interrupted by the injury or congestion of the supplying vessels, e.g. by inadequate type of incision, severe trauma of the skin or incorrect suture technique. As a rule, it can be recognised only in regions close to the skin wound and it should be observed for demarcation.

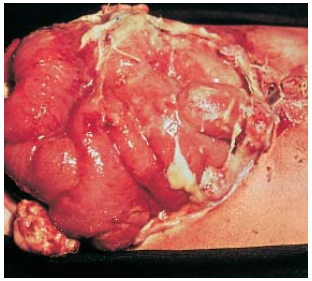
In the early days of wound healing, it is apparent as pale or cyanotic areas of skin which gradually become brown in colour. Skin necrosis must be kept dry and should not be removed prematurely as it functions as a sterile dressing. It is removed only after spontaneous demarcation. Moist necrosis, on the other hand, must be removed immediately because of the risk of deep retention of pus.

Wound dehiscence (rupture)

Wound dehiscence is a disorder of wound healing in which parts of the wound surfaces do not adhere and become bound by connective tissue despite apposing sutures. Examples of predisposing factors include: sutures causing ischaemia, sutures removed too soon, malnutrition, factor XIII deficiency, obesity, consuming neoplasms, postoperative coughing or diabetes mellitus. In addition, treatment with cytostatics, corticoids or antibiotics also increase the risk of rupture.

Postoperative wound dehiscence after laparotomy can be complete (affecting all layers), incomplete (intact peritoneum) or occult (skin suture still closed). The symptoms are a serosanguinous wound secretion commencing on the 3rd day, increase in wound pain, gastric atony, paralytic ileus or evisceration (bowel protruding through the wound). The dehiscence is treated by operation, if necessary inserting a plastic mesh. The prognosis is good when treated promptly and mortality is less than 10 %.



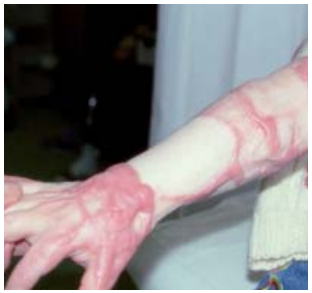


Complete rupture with muscle necrosis after a bypass operation in the knee (above), rupture after large bowel resection (below)

Hypertrophic scar formation

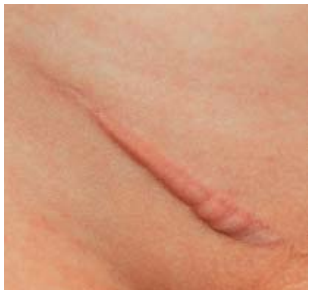
Some people tend to form excessive scar tissue, the causes of which may be disorders of collagen formation and cross-linking. Hypertrophic scars develop soon after operation, usually remain limited to the wound and demonstrate a spontaneous tendency to regress.

The wound site also plays a part in the formation of hypertrophic scars with regard to the skin's creases. If an incision runs vertical in the direction of Langer's lines, hypertrophic scarring can be anticipated. These circumstances gain particular significance in regions of the body where tensile forces act in the longitudinal direction of the scar because of strong muscle activity. The result is then not only a cosmetic failure. The scar crossing a joint restricts the range of motion with increasing scar contracture.



Hypertrophic scar formation after burn

When burns have healed in this manner, an attempt is made to prevent scar hypertrophy by compression with custom-made elastic clothing (pressure garments).



Keloid with typical collagen cords

Keloids

Distinguishing keloids from hypertrophic scars is difficult initially. There are also scar growths which are rich in fibres and which tend to recur even after subsequent excision. Their structure, which consists of thick glassy or hyaline cords of collagen embedded in a mucilaginous matrix, is crucial in distinguishing them from hypertrophic scarring. Even the smallest incisions can cause sizeable keloids which develop independent of muscle movement and rarely over joints. In contrast to hypertrophic scars, keloids often exceed the wound edges in their development and do not demonstrate any tendency to regress. Surgical correction often aggravates the situation.

Wound infection

Wound infection is the most serious disorder of wound healing. It is caused by a variety of micro-organisms which invade the wound and multiply, producing harmful toxic substances. The infection is usually limited locally, and leads through destruction of tissue to disturbances of wound healing which vary in severity. However, every wound infection can also extend systemically to become life-threatening sepsis.

Signs of infection

The signs of wound infection described by the Roman scientist Aulus Cornelius Celsus (1st century AD) such as rubor (erythema), tumor (swelling), calor (warmth), and dolor (pain), still serve as an aid to its recognition. They are an expression of the defensive struggle of the immune system against the invading micro-organisms. General signs and symptoms include fever, rigor, leucocytosis and lymph-adenopathy and leucocytosis. Fever, in particular, requires careful elucidation.

The earlier the diagnosis of infection is made, the better is the prospect of getting it under control in good time. However, recognising the onset of infections is associated with difficulties because unequivocal symptoms are still absent. The continuation of a local state of irritation, febrile temperatures, persisting leucocytosis and increasing wound pain are signs and symptoms which must be taken seriously.

Aulus Cornelius Celsus, who lived in the first century AD is considered, despite uncertainty about his precise dates, to be the author of the most important medical works of the ancient world.

Predisposing factors

The occurrence of infection is a complex process influenced by many predisposing factors. Of critical importance for the initiation of an infection are firstly the type, the pathogenicity, virulence and the number of micro-organisms involved. The micro-organisms then find a certain environment in the wound which more or less meets their living conditions. This explains why age, genesis and the condition of the wound (degree of contamination, extent of destroyed tissue and degree of perfusion), are other important predisposing factors. How quickly and effective the local defence mechanisms can form, depends on the wound condition.

This, in turn, is dependent on the general immune status of the involved organism. An already weakened immune system, reduced general condition, certain metabolic diseases, malignant tumours, advanced age, and malnutrition also have negative effects on the immune response. This means that penetrating micro-organisms find further favourable growth conditions.

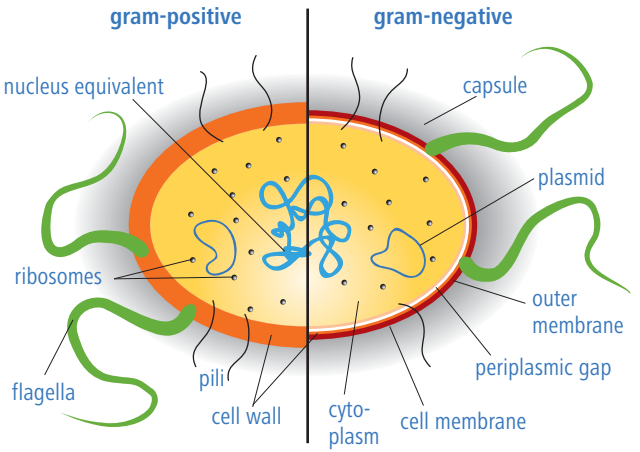
Pathogens

The causative agents of wound infections can be viruses, fungi and bacteria, with bacteria being implicated in the vast majority.

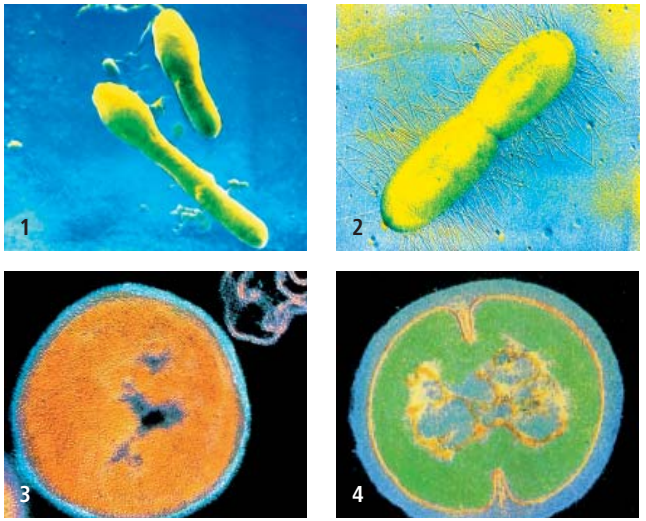
Bacteria are unicellular micro-organisms, whose cell interior is moderately differentiated. It consists of a "nucleus equivalent" containing genetic material as well as of cytoplasm containing ribosomes, various enzymes and plasmids (carriers of resistance genes). The outer cell wall can be found covered with a capsule of varying composition which can protect the bacteria against desiccation or against phagocytic cells.

Many bacteria form poisonous substances or toxins. The basis for toxin formation can be both exotoxin from the cytoplasm and endotoxin from the cell wall. The exotoxin is produced continually by the bacteria from the interior of the cell, e.g. in the case of gas gangrene organisms. Endotoxin is liberated only when the cell breaks up with destruction of the cell wall.

Bacteria are classified as obligate aerobic bacteria if they require oxygen to live, and as anaerobes if they need an oxygen-free environment. They are facultative aerobes or anaerobes if they can exist in both milieus. Differentiation of bacteria is by certain staining methods, for instance Gram's stain to distinguish them as gram-positive and gram-negative bacteria.



Structure and characteristics of gram-positive and gram-negative bacteria



Electronmicrographs of bacteria with differing pathogenicity:
1) Clostridium tetani, gram-positive, cause of tetanus, highly pathogenic
2) Escherichia coli, gram-negative, at early stage of division, facultative pathogen
3) Staphylococcus aureus, gram-positive, complete bacterium with a bacterium dissolved by effect of antibiotic in right upper corner, facultative pathogen
4) Staphylococcus epidermidis, gram-positive, during division; non-pathogenic

Pathogenicity

Bacteria only merit consideration as pathogens of infectious disease or of wound infections if they possess a disease-inducing potential (pathogenic).

Bacteria may already be highly pathogenic when they invade the wound. The human organism then has no time to activate the body's own defence mechanisms. Such infections are life-threatening. An example is tetanus caused by Clostridium tetani.

Other pathogenic strains are facultative, i. e. partially pathogenic. These are often bacteria from the physiological colonisation of the human organism which have left their natural location, penetrated into the wound and unleashed their pathogenic potential in the altered environment. This is the case, for example, when Escherichia coli from the bowel flora gets into the wound. In the case of Staphylococcus aureus, likewise an important causative agent of wound infections, the human carrier rate is about 30 %, with the main reservoir being the nose.

Another group of bacteria is classified as non-pathogenic. However, in the predisposed patient, with reduced immune defences, this can lead to opportunistic infections and wound infection. An example is Staphylococcus epidermidis which is normally found as an innocuous bacterium on the skin.

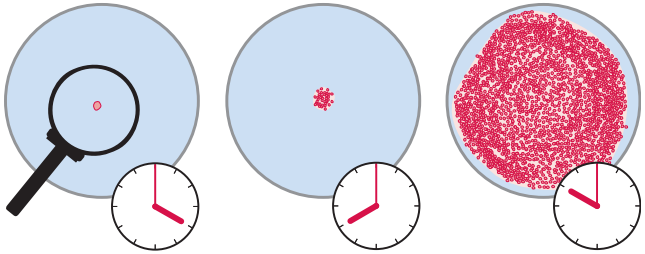
Virulence

The pathogenicity, or the potential of bacteria for causing illness, should be seen in close association with their virulence (infective force) which ultimately determines the degree of pathogenicity. Virulence is an acquired alterable characteristic: avirulent or slightly virulent bacteria can change genetically under the pressure of environmental influences and can become extremely virulent. This problem is particularly present in hospitals. Here new genotypes have developed due to the concentrated use of antibacterial therapy which are more virulent and more resistant to chemotherapeutic agents and disinfectants than, for example, the same type of bacteria in a domestic setting.

Dose of pathogen – manifest wound infection

Every wound, even so-called aseptic surgical wounds, are colonised by bacteria. The mere presence of bacteria in the wound, however, does not mean the same as a wound infection, but is designated as contamination. The body's defence mechanisms are often capable of removing bacterial colonisation so that infection does not occur at all. It is only when the bacteria penetrate deeper into the wound, multiply there, damage the tissue by their toxins and produce inflammatory reactions that infection can be said to be present.

Multiplication of bacteria is always by division. Apart from highly virulent bacteria, the reproductive activity of bacteria begins a few hours after adapting to the new nutritional situation. This incubation time is generally eight to ten hours. After that the bacterial count increases rapidly.



4 hours
4096 bacteria

8 hours
16777216 bacteria

10 hours
1073741824 bacteria

The speed of division (generation time) in a favourable environment and at the optimum temperature is about 20 to 30 minutes for many bacteria. The diagram shows the theoretical multiplication of a single bacterium with a generation time of 20 minutes after 4, 8 and 10 hours.

Logically, the number of invading bacteria, the pathogenic dose, is of critical importance. The more bacteria invade, the greater the probability that a infection will occur. Measurements of standardised samples have shown that 10^4 pyogenic Streptococci/mm^3 or $10^5 - 10^6$ Staphylococcus aureus/mm^3 have to be present to produce a wound infection. Depending on the clinical condition, a bacterial count of 10^5/mm^3 tissue is the approximate guiding principle for an infection requiring treatment.

When taking a wound swab, correct technique is crucial for a reliable result. The swabs should be taken from the depths of the wound and from the wound edges as the pathogens are concentrated at these sites.

Condition of wound and susceptibility to infection

The fresh wound is highly susceptible to infection. With increasing organisation of the defence mechanisms, the risk of infection diminishes so that a wound with well vascularised granulation tissue can already counter the pathogens with considerable resistance. Older chronic wounds also appear to have a lower susceptibility to infection. However, as long as the wound is not protected by a closed epithelium, the risk of infection persists.

The cells and substances important in local defence and antibody production and the oxygen required for phagocytosis depend on sufficient circulation. If perfusion in the wound area is reduced or absent, the risk of infection increases considerably.

Necrotic tissue has no perfusion and represents an ideal breeding ground for bacteria. All traumatic wounds with crushing, tearing and loculation of tissue are thus particularly at risk of infection. In the treatment of such wounds, infection should be assumed from the outset in order to create a "clean" wound situation in good time by comprehensive wound excision. If there are areas of closed necrosis (typical of decubitus ulcers) it should be borne in mind that there may be a purulent infection beneath the necrosis which can spread into deeper tissue layers.

Moreover, stagnant secretions loaded with bacteria, such as in deep and gaping wounds, are also dangerous. It may occur a so-called moist chamber where this negative effect may be reinforced by an unsuitable dressing with inadequate absorbency and moisture permeability.

Foreign bodies, such as suture material, plastic parts or implants may cause a local reduction of the body's defences. They may cause a marked ischaemia and a risk of infection. The site of the wound is also of significance with respect to the risk of infection as the individual body regions have both a variable perfusion and a variable level of bacterial colonisation.

Finally, the aetiology of the wound plays a major role in the risk of infection. In the case of surgical incisions, the risk of infection is always dependent on the type of operation with its specific hygienic risks (aseptic and partially aseptic procedures, surgery in a primarily contaminated and in a primarily septic wound area). Other risks emanate from operative preparation and performance and from post-operative wound care. Important factors confirmed by various studies include:

- duration of pre-operative stay on the ward, because with each day the patient´s colonisation by nosocomial organisms increases
- preoperative antibiotic regime
- preoperative shaving of the operative field
- hygiene status and quality of hygiene management in the operating theatre
- operative techniques, degree of tissue trauma (faulty incision, diathermy, suturing and ligating technique)
- duration of operation (the number of pathogens increases, exposed tissues are jeopardised more because of drying, circulatory disturbance, reactive oedema etc.)
- wound drains and their postoperative care

All wounds due to external force, such as stabbing, crushing and impalement wounds, should generally be considered as being infected, as organisms always get into the wound along with the object causing the injury. The same applies to bite wounds as very virulent micro-organisms are transmitted with animal and human saliva.

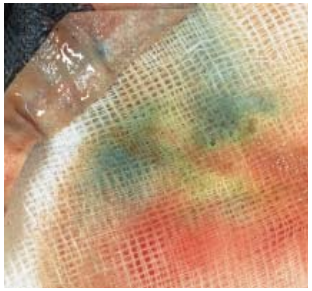
Types of infection

The different types of pathogens produce specific tissue reactions which characterise the clinical signs and symptoms of the infection.

Pyogenic infection

The causes of pyogenic, or pus-producing infections are above all the pygenic agents such as gram-positive staphylococci and streptococci as well as gram-negative pseudomonas and Escherichia coli. An experienced clinician can deduce the main type of pathogen from the appearance and smell of the exudate. Nevertheless, a culture swab with antibiotic sensitivity should not be omitted as the basis for adequate antibiotic treatment.

- Staphylococci: creamy yellow odourless pus
- Streptococci: runny yellow-grey pus
- Pseudomonas: blue-green sweet-smelling pus
- Escherichia coli: brownish faeculent-smelling pus

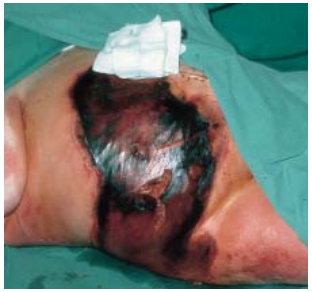


The blue-green sweetish-smelling pus is typical of a pyogenic pseudomonas infection

Putrid infection

Putrid infection or putrid faeculent tissue gangrene develops from mixed infections with Escherichia coli and the putrefactive organisms Proteus vulgaris and Streptococcus putrides. The putrefactive organisms destroy the tissue and foul-smelling gases form during the breakdown of the protein structures. The clinical appearance is of gangrenous inflammation with gas phlegmon in the surrounding tissue.

Emergency treatment with an antibiotic effective against both aerobes and anaerobes must be started without waiting for bacteriological diagnosis.



Gas gangrene with soft tissue necrosis which has already turned black; typically, there is crepitus on palpation.

Gas gangrene

The gas gangrene pathogens Clostridium perfringens, Clostridium novyi and Clostridium septicum which occur in soil and street dust are obligate anaerobes and find ideal growth conditions in gaping, necrotic and poorly perfused wounds. They rapidly give off tissue-dissolving and gas-producing exo- and endotoxins which quickly lead to general intoxication of the organism. Genuine gas gangrene (as opposed to gas phlegmon in putrid infections) occurs rarely and is usually fatal.

Tetanus

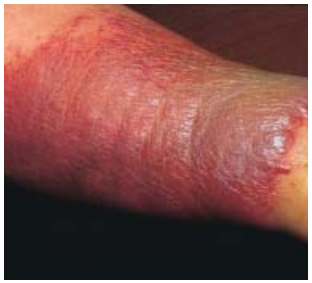
The aetiological agent is Clostridium tetani, also an obligate anaerobe occurring in soil and street dust. Once again, gaping, contaminated and poorly perfused wounds are particularly at risk, but every tiny injury of the skin can be the site of entry. The nerve toxins released by the bacteria migrate along the nerve tracks to the spinal cord and cause severe spasms which spread in a craniocaudal direction. Tetanus immunisation offers protection against tetanus which has without immunisation a fatal outcome in about 50 %. If immunisation is uncertain in the event of an injury, the patient is considered non-immune and receives active and passive immunisation protection.

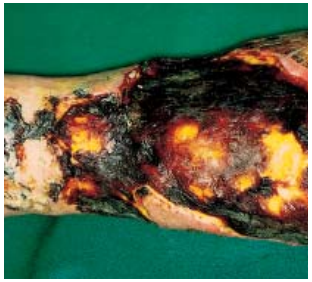
Rabies

Rabies, caused by rhabdovirus, is transmitted with the saliva in a bite from an infected animal. The virus enters the bite wound and ascends along the nerves to the central nervous system. Total paresis and death occur. When the condition is established, every therapy fails so that treatment must be given as soon as rabies is suspected (abnormal behaviour in the biting animal).

Erysipelas

Erysipelas is a relatively common bacterial disease, usually caused by β-haemolytic streptococci. It starts acutely with fever, rigor, swelling, erythema, and tenderness of the affected skin. Favoured sites are the lower leg and face. The diagnosis can be made easily from the typically sharp demarcation between healthy areas of skin and the fiery erythema. Tiny erosions of the skin or mucous membrane are sufficient as an entry portal; outflow obstructions in the lymphatic and venous system favour its occurrence. A rare form causing severe illness is necrotising erysipelas with symptoms of shock.





Erysipelas of the lower leg with typical sharp demarcation from the healthy areas of skin (above); advanced, already necrotising erysipelas, also of the lower leg (below).

Prevention and treatment of wound infections

The prevention of a wound infection means the greatest possible prevention of bacterial colonisation, while treatment concentrates on a corresponding reduction of the existing bacterial colonisation or on elimination of the invading bacteria. The measures which serve for prophylaxis and treatment should not be seen in isolation but as an overall concept, and require a disciplined approach by all involved in wound care.

An overriding measure is strict observance of asepsis. It is an essential requirement for preoperative preparation, intra- and postoperative activity and for the open wound treatment in all acute and chronic wound conditions.

Even wounds which are already clinically infected should be treated exclusively under aseptic conditions. Apart from the fact that further secondary infections must be prevented, such wounds represent a reservoir of extremely virulent organisms, spread of which can be prevented only by comprehensive asepsis.

Other measures to prevent and treat wound infections are in turn dependent on the condition of the wound and require an adequate procedure:

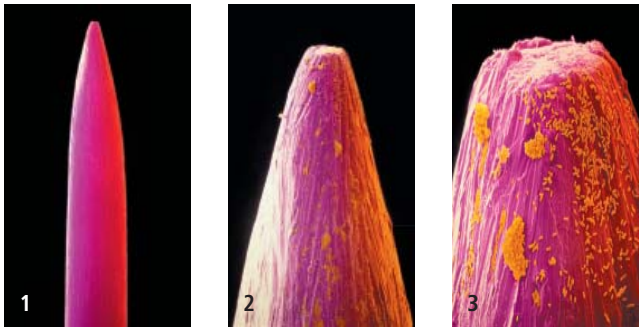
In the case of infected wounds which have had primary closure, rapid drainage of secretions should be obtained by opening the suture and using suitable wound drains. In all wounds healing secondarily such as traumatic or chronic ulcerative wounds, extensive surgical debridement is to the fore: necrotic and devitalised tissue must be removed generously, wound loculations opened up, sloughy deposits, foreign bodies and infected areas excised. At the same time, tissue perfusion is ensured with oxygen delivery which is essential for the body's local defences.

If surgical debridement is not possible because of certain circumstances, physical wound cleansing with moist dressing treatment and possibly the topical application of enzymatic preparations is indicated.

Antiseptics

According to the general definition, the prophylactic/therapeutic aims of antisepsis consist in killing or deactivating microorganisms, or preventing them from multiplying by using locally acting chemical substances designated antiseptics or antiinfectives. Since wound antiseptics have a more or less cytotoxic potential, the most suitable wound antiseptic needs to be chosen in each treatment case. The following basic requirements should be met by the preparation:

- reliable germ-killing (microbicidal) or inactivating effect against a wide spectrum of microorganisms
- no protein-related faults, i.e. no loss of effectiveness of the antiseptic when affected by proteins (since during open wound treatment, the antiseptic is always in contact with proteins, e.g. in the blood and wound secretions, particular attention should be paid to this point)
- rapid onset of effect
- no development of microbial resistance or gaps in effectiveness
- no pain caused
- greatest possible cell and tissue tolerance and toxicological safety
- simple use and storage

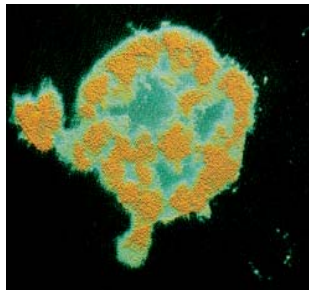


Infective agents are present everywhere, even if they are not visible to the naked eye. The illustrations show an apparently clean needle tip (1). The magnifications (2) and (3), however, reveal heavy bacterial colonisation (yellow).

A reduced-risk application of antiseptics in open wound areas therefore always presupposes that the user is thoroughly informed about the particular properties of the selected substance and, in particular, about its effects on the immunologically active cells.

In general, the principle applies that treatment with antiseptics should be carried out as briefly as possible. Antiseptic application should be stopped as soon as the clinical signs of infection cease (e.g. when secretion and swelling subside). The progress of treatment should be assessed daily and possibly checked with microbiological diagnostic methods.

Above all in the case of chronic wounds, it is not infrequently observed in clinical practice that antiseptic treatment may be continued for weeks or months without problems and without regard to any treatment success. Whereas during the infection stage, the disruption of sensitive wound healing processes by relatively cytotoxic antiseptics can be disregarded, since they are already severely disrupted by bacteria, long-term use carries the potential for significant damage. The undesirable effects of these substances significantly worsen the poor healing tendency of chronic wounds, and can trigger contact allergies. Added to this is the fact that long-term use of antiseptics is often regarded as a sufficient and safe wound treatment method, so that nothing is done to diagnose and treat the actual causes of the poor wound healing.



Left: Escherichia coli, resistant to two antibiotics (no halo).

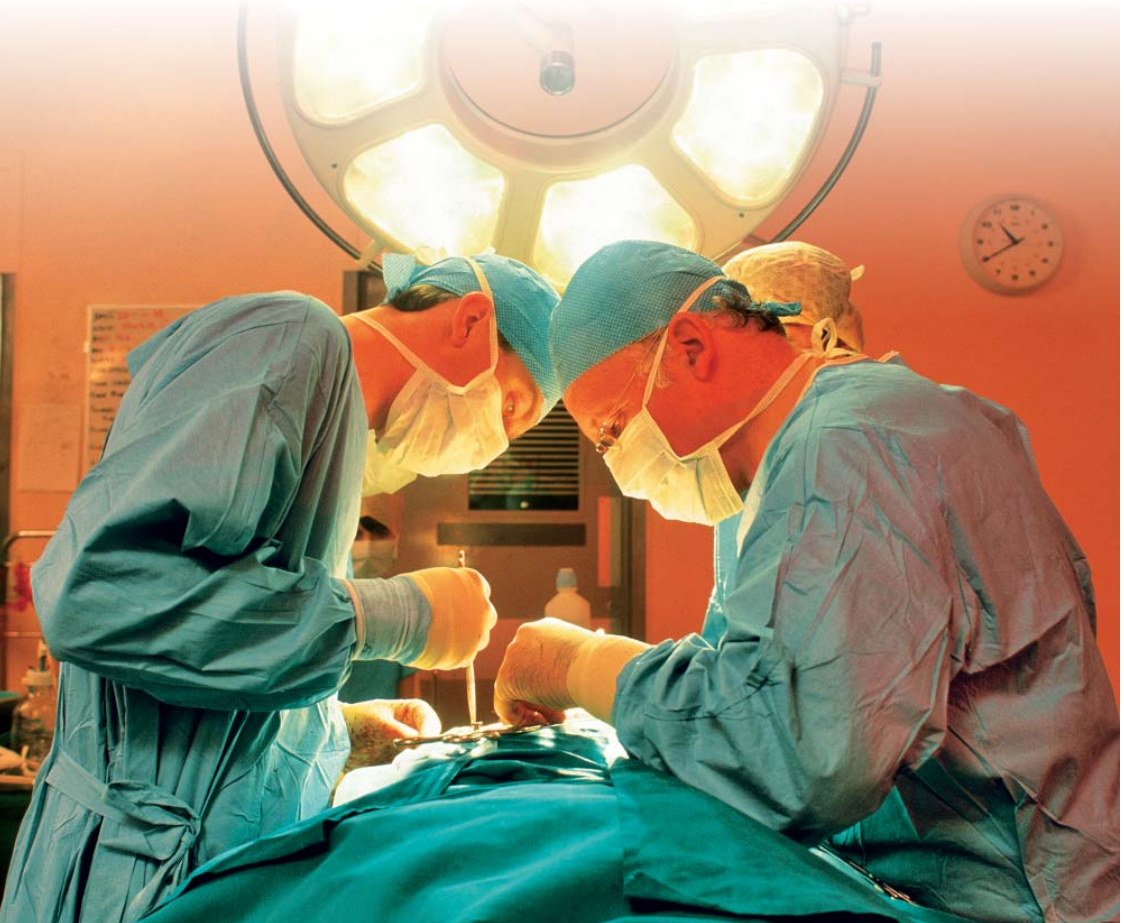
Right: Staphylococcus aureus during destruction by an antibiotic: destruction of the outer cell wall with release of intercellular material into the surroundings.

Antibiotics

Treatment with local antibiotics is a controversial subject and is generally regarded today as obsolete. The reasons for this lie in their selection of resistant germs, sensitisation of the patient and the consequent loss of a potential antibiotic for systemic treatment, as well as the danger of superinfection with fungi. Contrasted with this, the systemic administration of antibiotics for local progressive infections (phlegmons, lymphangitis, etc.), deep infections (emphysema, osteomyelitis, etc.) and generalised infections (sepsis) is absolutely necessary. When choosing an antibiotic, the pathogen spectrum needs to be taken into account in accordance with the germ identification and resistance testing. In the event of a dramatically developing infection process, an empirical initial treatment should be begun, and broad-spectrum antibiotics have proved valuable for this. The treatment is then assessed following performance of an antibiogram and a resistogram and adjusted accordingly if required.

Principles of treatment of acute wounds

The treatment of acute traumatic wounds was probably the earliest medical activity. Pioneering successes were achieved towards the end of the 19th century when, with the discoveries of antisepsis and asepsis and the development of anaesthetic techniques, the restricting factors of surgery were cleared away. Today, the surgical treatment of traumatic wounds has reached a high level. Particularly helpful are the possibilities of plastic surgery, which offer survival and acceptable wound healing results to some severely injured people.



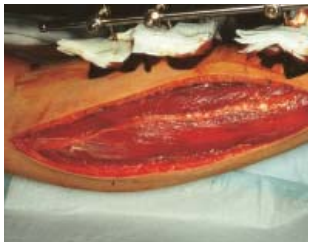
The goal of every wound treatment is to assist the organism to achieve as soon as possible functional regeneration or repair of the injured tissue. Fundamental measures include:

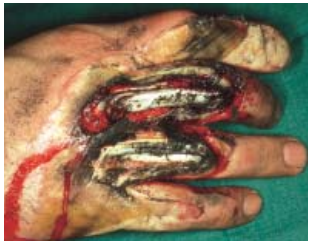
- evaluation of the wound with regard to aetiology, site, age and condition along with any concomitant injuries and underlying illnesses
- elimination of bacterial colonisation and factors favouring it by means of thorough debridement
- wound closure by primary or secondary suture or by skin or flap transplantation

The degree and extent of the individual measures differ according to the wound findings and the healing that can be expected. The principles and techniques of wound treatment in the case of acute traumatic wounds are summarised in brief below. However, it must be borne in mind that a rigid treatment plan is ruled out by the variety of individual patient circumstances. Ultimately, the skill of the person treating the patient is of fateful significance for the patient.

The acute traumatic wound

Related to the mechanism and the circumstances of the accident, traumatic injuries cause a wide spectrum of damages. They range from the incised wound to complex defects with involvement of tendons, muscles, nerves, vessels, bones or internal organs. Apart from trivial injuries, treatment is classified for practical reasons as provisional or definitive wound treatment.





Lower leg fracture (above), severe finger trauma due to injury on a conveyor belt (below).

Provisional wound treatment includes:

- first aid measures for haemostasis
- application of an emergency dressing to protect against infection and for transport
- if necessary, immobilisation of the injured body parts and limbs

However, in the case of severe injuries accompanied by shock, immediate initiation of shock therapy and stabilisation of the vital parameters, always takes precedence over provisional wound care.

Definitive treatment or primary care follows basic surgical principles. With the exception of superficial skin defects, all other wounds are explored surgically with adequate analgesia and under aseptic conditions. Inspection of the outer wounds suffices only in very rare cases. Further radiological or neurological investigations may be necessary to confirm suspected foreign bodies in the depths of the wound, fractures or nerve an head injuries.

Prompt debridement has the aim of rendering a wound low in bacteria and well perfused. Tissue with impaired perfusion such as obvious necrosis and crushed soft tissues is excised in order to obtain a smooth and clean wound, thus removing the breeding ground for wound infections. Nerves, tendons and muscles should be protected and preserved as far as possible. Injured vessels should be treated immediately by vascular surgical techniques.

Particular care is warranted in the case of deep and gaping wounds. Foreign bodies such as particles of dirt, fragments of cloth or glass splinters may be detected by soft tissue X-rays only with difficulty but must not remain in the wound because of the associated high risk of infection. Superficial

Management of traumatic wounds

Provisional wound treatment

- treatment of shock, where necessary
- haemostasis
- emergency dressing
- immobilisation
- transport to hospital

Definitive wound treatment

- operative wound exploration
- debridement
- decision on wound closure

Wound closure

primary / primary delayed

dressing to protect wound

secondary / open

moist dressings to condition the wound

later closure by secondary suture,
spontaneous epithelialisation,
skin graft, plastic surgical procedure

epithelial wounds are cleaned by irrigation. Finger and facial injuries are not excised provided the wound edges have not been crushed.

In general, debridement of the wound is a demanding surgical operation and requires meticulous care on the part of the surgeon, based on solid anatomical knowledge.

The decision on wound closure depends on the extent and the result of the debridement. Primary wound closure by

suture, staples or wound closure strips is possible if the wound edges can be approximated without tension and if it can be ensured that the wound is sufficiently clean.

A wound must never be closed under tension as every forced wound closure endangers wound healing because the sutures cause ischaemia resulting in a disturbance of tissue perfusion leading to necroses and infections. In case of doubt, the wound should be left open for secondary wound healing.

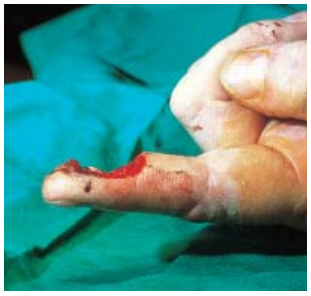
To ensure low levels of micro-organisms when primary wound closure is attempted, apart from correct debridement, the following conditions must be met:

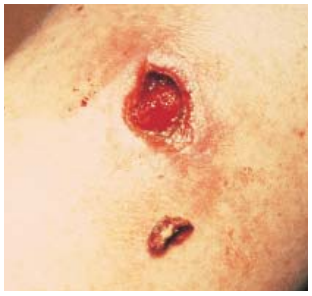
- the wound must not be older than 6 – 8 hours and
- it must not be due to causes which from the start involve a high probability of primary infection

This includes all bite wounds, including human bites, tearing and scratch wounds from animals, stab and shooting injuries. In addition, it includes injuries in persons who have come in contact with infectious material such as human or animal pus or excrement.

Knowledge of the mode of injury and accompanying circumstances are thus of crucial importance for assessing the risk of infection and the procedure to be adopted.

Bite injuries from animals (at left, a dog bite wound) or gunshot wounds (right) have to be classified as infected from the start and treated accordingly.





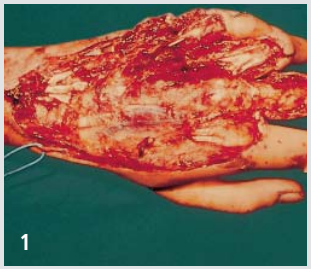
Postponed primary care is often considered in the case of wounds which are unsuitable for primary closure. The wound is debrided but is then kept open for observation with sterile moist dressings or by packing for a few days. If no signs of infection appear, the wound can be closed by suture, usually between the 4th and 7th days. The sutures for wound closure are usually inserted during the initial treatment but left untight.

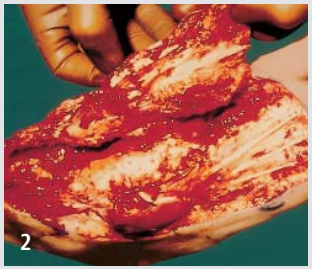
The question of wound closure in the case of wounds which are healing secondarily with their varying degrees of tissue destruction is much more complex. Simple skin defects with or without exposed muscle can usually be closed by secondary suture after operative revision, adequate debridement and conditioning of the wound with skin substitute materials or can be covered by split skin grafts. If there are complex defects, reconstruction of the soft tissues using plastic surgical procedures is essential.

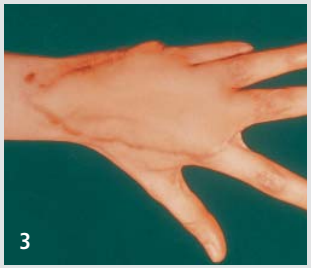
Complex traumatic defects

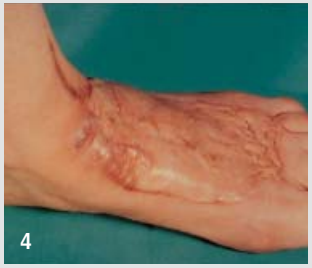
In complex defects, several functionally important structures of the limb are injured. This may occur in various combinations. In the case of compound fractures, muscle tears and zones of contusion are often found in addition to nerve, tendon or vascular injuries. The involved structures are exposed and cannot be managed adequately and definitively with simple skin grafting.

Covering of defect by plastic surgical procedure:
1) Complex defect on the dorsum of the hand after a motorcycle accident with loss of all soft tissues and extensor tendons of the middle fingers.
2) A tendo-fasciocutaneous flap is raised from the dorsum of the foot.
3) Function after 8 weeks.
4) Acceptable defect at donor site.

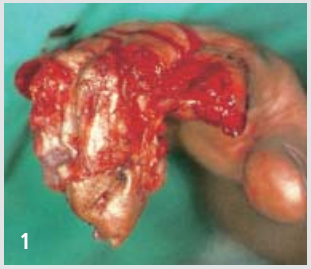


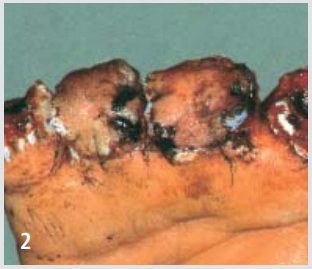


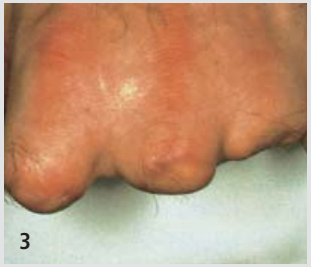


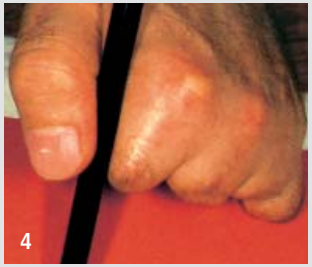


Treatment of a traumatic finger injury:
1) Partial amputation of 2nd to 5th fingers on admission.
2) Subsequent completion of amputation with two free skin flaps. Revision was necessary because of increasing necrosis of the ring finger.
3) Findings after 8 weeks.
4) Good functional result.









Later in the course of such an injury after inadequate primary treatment, a soft tissue or bone infection can occur which then complicates the local situation. The emphasis is then on treatment of the infection by means of stable soft tissue cover. Reconstruction of defective structures under such conditions has to be carried out secondarily, as the risk of infection is too great for the reconstructed structure if done primarily.

The same basic principles of treatment apply to all stages of soft tissue injury. Securing and stabilising the vital parameters are followed by evaluation of the patient, in interdisciplinary collaboration whenever possible. During the primary operative exploration, the fracture is stabilised, the wound is debrided and where possible all damaged structures are reconstructed.

If definitive debridement is possible during the initial management, the wound can receive its final primary covering. If there are doubts about the vitality of the remaining tissue, definitive optimum closure can be attempted within 5 – 7 days following a planned "second look".

The guiding principle of treatment must be to offer the patient the "optimum" solution. This means that the procedure at the correct stage to reconstruct the soft tissues is guided by the size and nature of the defect, the local situation, the patient's overall condition, and also the medical and social profile of the patient. The wound should receive its definitive treatment as soon as possible. In the case of complex defects, where immediate reconstruction is not possible, an interim soft tissue cover is required.

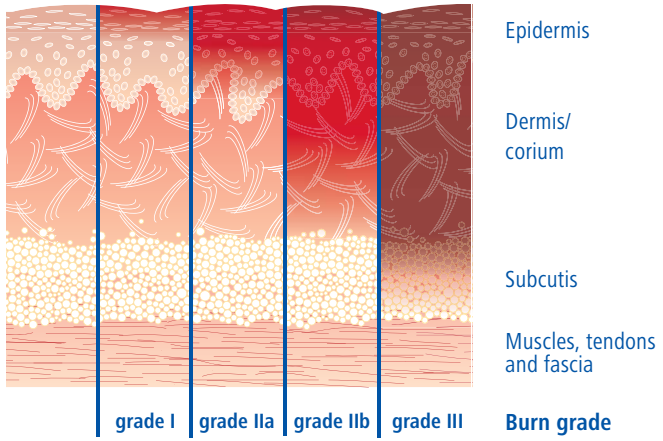
Principles of treatment of complex defects
- Stabilisation of the patient
- Interdisciplinary evaluation of the patient
- Operative exploration of soft tissue and bone situation
- Fracture treatment
- Radical debridement
- Primary reconstruction of injured structures
- Possible second look
- Definitive differentiated cover/ closure within 5 – 7 days

Thermal injuries/burns

Depending on the intensity and type of the thermal medium acting on the skin, a burn wound of varying severity is produced. Its appearance ranges from superficial erythema to total skin necrosis. Extensive severe burns are among the worst injuries which a human being can sustain.

At the accident site a decision is made, based on findings, as to whether treatment should be undertaken on an outpatient or an inpatient basis. For superficial first-degree burns and second-degree burns of type a, covering less than 10 % of the body surface area or third-degree burns over less than 0.5 % of the body surface area and located on the trunk, upper arm or thigh, outpatient treatment is recommended. For deep second-degree of type b or third-degree burns, covering more than 10 % of the body surface area, the patient is admitted to the nearest hospital regardless of the location of the burns. A decision must then be made whether transfer of the patient to a centre for burned persons is necessary.

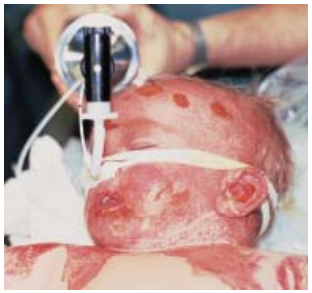
The severity of burns is classified into three degrees for prognosis and treatment, with second degree further subdivided into grades IIa and IIb. The classification refers to the depth of the injury, i.e. which parts of the skin are burnt.



During emergency treatment, cooling of burn wounds as soon as possible with tap water for approximately 30 minutes is a priority measure. However, care should be taken with babies and young children to avoid hypothermia. Cooling can lessen pain and reduce or avoid “afterburning”, which is caused by energy storage for almost an hour in the well heat-insulated skin. This heat, together with continuing intravascular coagulation in the injured skin, lead to further tissue damage, so that a primarily superficial burn can turn into a deep burn.

In the case of a first-degree burn, which is characterised as damage to the uppermost epidermal layer and manifests itself as erythema, healing takes place spontaneously in a few days without scar formation.

A second-degree burn of type a, involves the entire epidermis and is extremely painful. Vesiculation, caused by plasma escaping from the injured capillaries, occurs after a delay of between 12 and 24 hours after burns. Since in the papillary cones and in the intact skin appendages, enough vital cells are still present for rapid re-epithelisation, spontaneous healing without scar formation generally occurs within about 14 days. Of great importance is sterile treatment of the wound by disinfection and covering with suitable wound dressings (e.g. ointment dressings, such as Atrauman, or cooling hydrogel dressings, such as Hydrosorb). Large area injuries of this degree of severity, e.g. scalding in children, can induce a shock reaction.



Grade IIb: Deep dermal burn of the epidermis and almost the entire dermis with the skin appendages. The wound base is red or whitish at the more deeply burned skin areas. There is always an acute risk of increasing the thickness to that of a third degree burn.

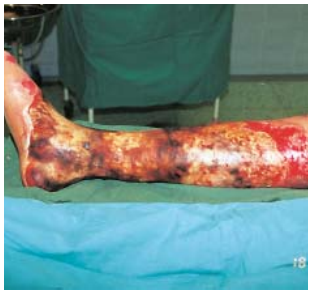
In second-degree burns of type b, the epidermis, almost the entire depth of the dermis and, to a large extent, the skin appendages are destroyed. In such cases, spontaneous healing takes several weeks and frequently leaves a hypertrophic scar. Often, despite all efforts, the injury deepens to become a third-degree wound. In general, second-degree, type b wounds resemble third-degree burns in their clinical appearance, so that treatment by necrosis removal and subsequent covering of the defect (with the patient's own skin or a skin replacement) is also similar to that for third-degree wounds.

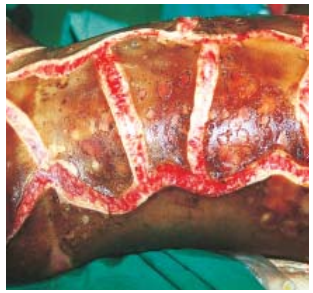
In third-degree burns, the epidermis, dermis – and often partially the subcutis also – are irreversibly destroyed (full thickness burn). Spontaneous healing with very slight extension of the wound edges is possible only with scar tissue. Otherwise, the coagulation necrosis of the skin causes massive contractions. The patient does not feel any more pain and the nails and hair fall out. Treatment of such burn wounds is purely surgical.

On principle there is a high infection and sepsis risk with all open burn wounds. Wound infections represent the commonest cause of death in burned persons. A badly burned person is exposed to additional risk of burn shock and consequent sickness. Careful observation of the clinical appearance, informed decisions on individual treatment steps and adequate intensive care are therefore of decisive importance to the survival of the patient.

Wound conditioning of deep burns

The aim is the creation of a vital wound base into which a variety of skin transplants or skin replacement materials can become incorporated for definitive or temporary wound closure. A variety of techniques are available for removing necroses, depending on the depth and extent of the burn.





Degree III:
Necrosis of epidermis, dermis and parts of the subcutaneous tissue (left); the skin is brownish, black, leathery and insensitive to pain, hairs and nails fall out. Circumferential burns on the trunk with relieving incisions to facilitate breathing (right).

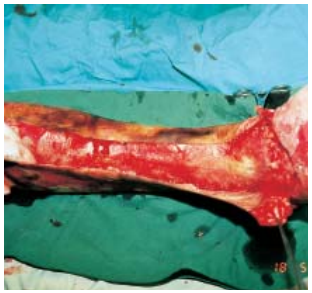
With the so-called granulation method, in a phased process (about every 3 or 4 days), the eschar is thinned on the surface with a knife or removed with hard brushes. Between debridements, the burn wounds are protected against infection usually by antimicrobial (ointment) dressings. This "closed treatment" also prevents drying out of the wound surfaces, so that the danger of secondary necrosis is reduced.

In the case of third-degree circumferential burns on the neck, trunk and extremities, relieving incisions (escharotomy) in the eschar are necessary. Otherwise, the contaminated, necrotic skin coupled with the excessive oedema formation lead, during the course of the burn disease, to suffocation symptoms and to disturbed blood circulation and compression of the neurovascular bundles.

A further technique for removing necrotic skin tissue is tangential excision. This comprises area removal of destroyed skin with a dermatome, layer by layer until a bleeding, vital wound surface is reached, onto which the split-skin graft can grow. A disadvantage of this method is the poorly controlled capillary bleeding and the difficulty of finding exactly the right excision depth at the transition to the healthy tissue. Following the excision, wound closing is immediately carried out with patient's own skin transplantation.

Treatment of burn wounds

- Degree I
  Spontaneous healing in a few days
- Degree IIa
  Spontaneous healing within about 14 days
- Degree IIb
  Partly conservative, partly surgical depending on clinical appearance
- Degree III
  Surgical with necrosectomy and skin grafting



Deep epifascial excision with radical removal of destroyed skin and fatty tissue down to the fascia.

Using the technique of total or deep epifascial excision, however, the destroyed skin is radically removed down to the healthy muscle fascia. Bleeding can be controlled better than in the case of tangential excision and healing of the grafts is generally good, since a healthy wound base is reliably produced by the deep excision. The cosmetic result cannot be regarded as optimum, however. This method is indicated, above all, for life-threatening third-degree burns whereby survival is a higher consideration than function and aesthetic result.

Less well-known is necrosectomy with 40 % benzoic acid in white vaseline (salicylic acid was used in the past) for bloodless removal of necrosis, e.g. in elderly patients, in the case of burns on the dorsum of the hand and wherever subcutaneous structures are directly beneath the skin surface.

Temporary coverage of burns

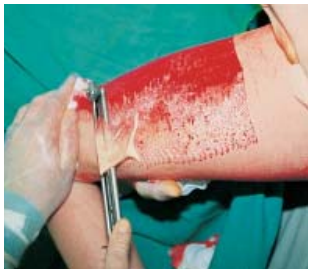
After removal of necrotic skin tissue by the various excision methods, the wound surface is usually immediately transplanted. In cases where the wound cannot be grafted or there are not enough donor sites because of the extent of the burns, the wound still has to be covered temporarily. Biological wound covering materials are used, the most suitable of which is an allograft of human skin. This is either fresh skin or preserved cadaver skin. Apart from its massive stimulatory effect on wound healing, allografts stem the loss of secretions and protein, reduce pain and contribute markedly to microbial reduction.

A further option for temporary coverage is xenotransplantation with porcine skin. While the allografts heal for about 14 days, the xenografts have to be removed after 3 – 4 days. The xenografts have the basic effect as human skin replacement even if not to the same extent. Not least for cost reasons, synthetic wound dressings such as Syspur-derm are often used for temporary cover. The use of allo- or xenografts is primarily limited to cases of critical burns.

Methods of autografting

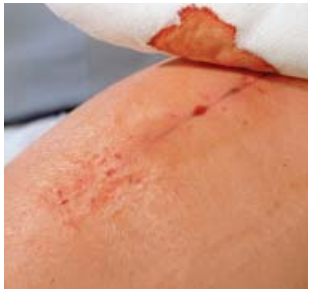
Split skin is taken initially for autografting using a special knife or dermatome. If there are enough donor areas, it can be left in this form for grafting. Usually, however, because of a lack of donor sites, the skin has to be processed into a mesh graft with a mesh dermatome. For both aesthetic and functional reasons, use of mesh transplants is contraindicated on the face, neck and hands.

The mesh transplant is laid on the well-prepared wound, fixed with sutures and staples and covered by a slightly compressing, non-adhering and absorbent dressing. Depending on the secretion, the dressing is changed at intervals of a few days, about every 2 – 5 days.



Autografting split skin graft: removal of donor skin (left); coverage of well-prepared wound surface with the mesh graft and fixation with staples (right).

With critical burns affecting 80 % of body surface, the lack of donor sites is dramatic. A solution for this situation could be the method of culturing keratinocytes in vitro, by means of which up to 1 – 2 m^2 of autologous epithelium can be grown from 2 – 4 cm^2 of the burn patient's skin. To do this, the keratinocytes are isolated from the piece of the patient's skin using certain processes and brought to division in a nutrient medium until a layer of cells capable of being grafted has formed.

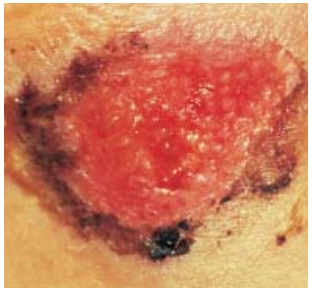


Incisions closed with sutures are predestined to heal rapidly by primary intention provided there are no wound infections or other disorder of wound healing.

Incisions/surgical wounds

Surgical wounds usually involve negligible tissue loss and are predestined for rapid healing by primary intention, if there are no wound infections or other disorders of wound healing. According to the circumstances of operation, wound drains are inserted to remove serous secretions and blood in order to avoid seromas and haematomas.

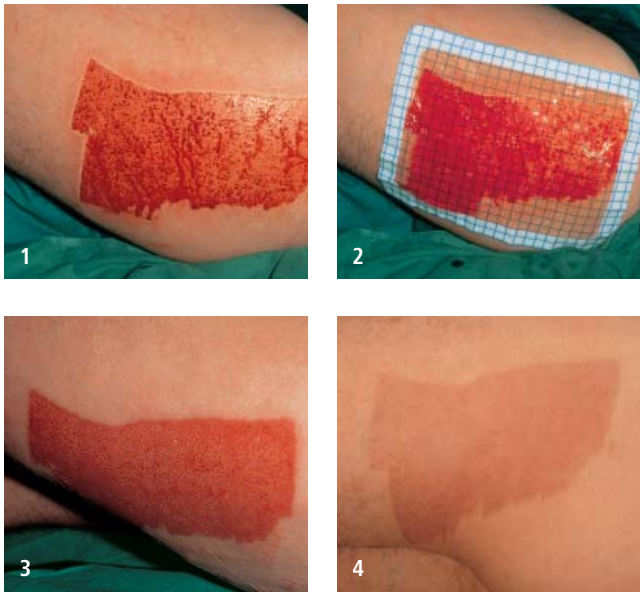
The surgical wound is covered with an absorbent and air-permeable dressing pad which has the function of absorbing any secondary bleeding and protecting the wound against secondary infection and mechanical irritation.



Epithelial wounds affecting only the avascular epidermis heal without scarring.

Epithelial wounds

Epithelial wounds or superficial wounds affect only the avascular epidermis. They re-epithelialise spontaneously and heal without scarring because replacement tissue does not have to be produced. However, because the fine capillaries lying directly under the germinal layer are also opened, superficial wounds can bleed and secrete a great deal and therefore tend to adhere to the dressing. The wounds are also often quite painful because many nerve endings are exposed. Epithelial wounds are produced by accidental skin abrasions or by split skin removal.



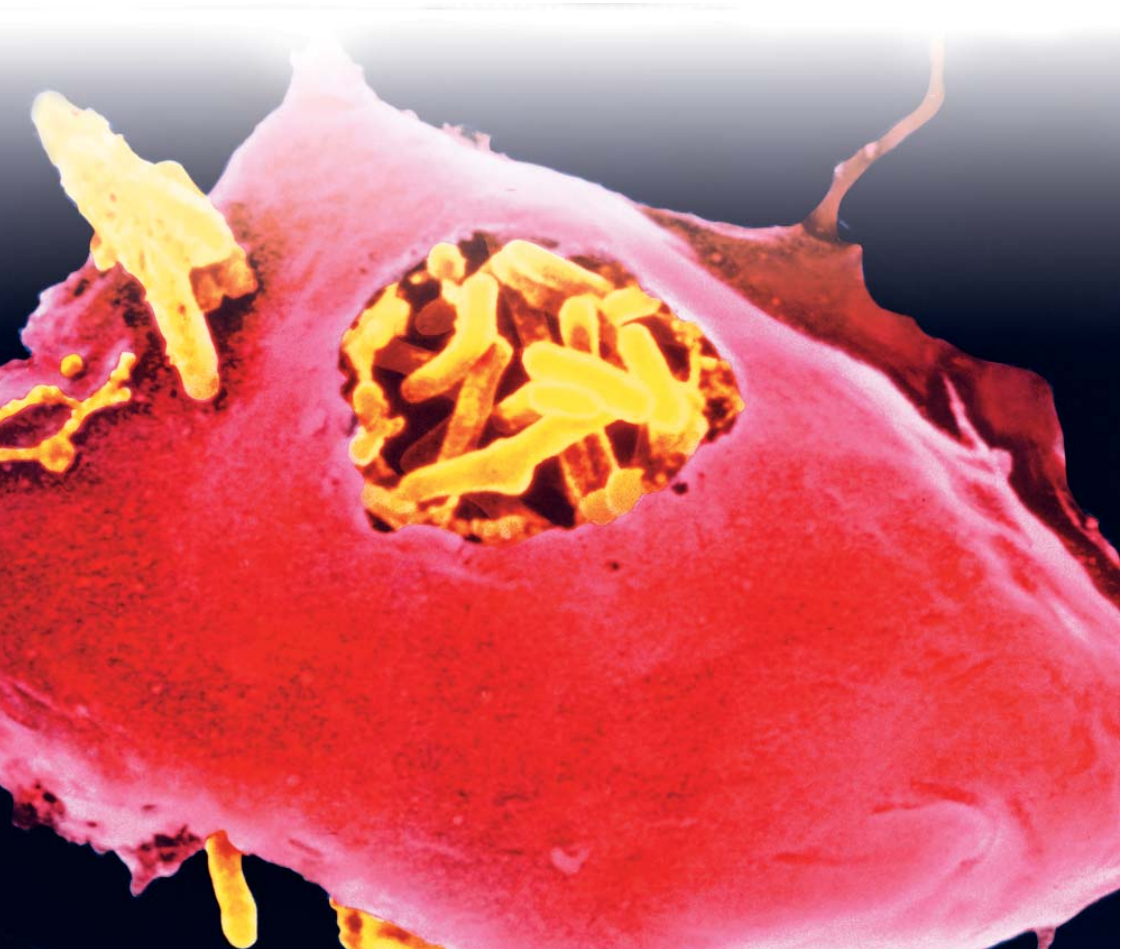
Moist wound treatment is of crucial importance for a cosmetic result of wound healing of epithelial wounds:
1) Primary aseptic wound after removal of split skin from the thigh.
2) Application of Hydrosorb plus* on the split skin donor site.
3) Re-epithelialisation is complete on the 5th day.
4) Condition 5 months after the start of treatment, the donor area has regenerated almost completely.
*Hydrosorb plus is also available as Hydrosorb comfort with a transparent continuous adhesive edge.

Abrasions are cleaned mechanically, and haemostasis of the exsudate with warm moist compresses may be required. A dressing is then applied which protects against infection but, if selected correctly, can also promote the process of epithelialisation. The dressing has to keep the wound moist and supple and must not dry out or adhere. Drying results in formation of a scab which delays healing. If the dressing adheres, newly formed epithelial cells are removed when the dressing is changed and the change of dressing is painful. Suitable dressings for treating epithelial wounds are ointment dressings such as Atrauman, absorbent dressings with a non adhering gel coating such as Comprigel or hydrocolloid and hydrogel dressings such as Hydrocoll and Hydrosorb.

Split skin and Reverdin donor sites are like skin abrasion wounds and are treated accordingly. Removal is followed by haemostasis and the wound surface is dressed with a moist dressing in the operating theatre.

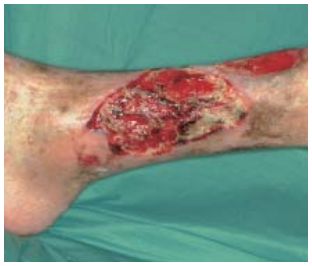
Principles of treatment of chronic wounds

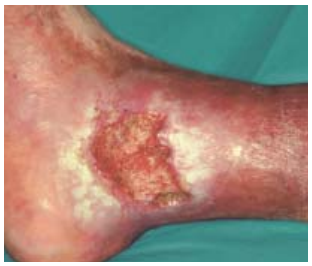
The treatment of chronic wounds of varying genesis places the greatest demands on therapeutic management. After all, by no means all the processes that can adequately explain faulty cell mechanisms are known. It is, however, increasingly possible, based on the actual knowledge about physiological wound healing mechanisms to intervene actively and correctively even in disrupted healing processes.



By definition, a wound healing secondarily which shows no tendency to heal after 8 weeks, despite correct local and causal treatment, is designated as chronic. Chronic wounds can develop at any time from an acute wound, because of an undetected persisting infection or inadequate primary management. In the majority of cases, however, chronic wounds represent the final stage of progressive tissue destruction, produced by venous, arterial or metabolic vascular disorders, pressure injuries, radiation damage or tumours.

As might be expected from the causes, it is mainly elderly people who are affected by chronic wounds and the alteration in the age structure of the population with an increased proportion of elderly persons will lead to a further marked increase in chronic wounds. In order to meet this requirement, it is urgently necessary, on the one hand, to enhance the prophylactic efforts and, on the other hand, to introduce a scientifically based and effective wound management with corresponding quality controls.



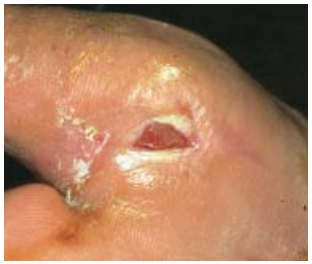


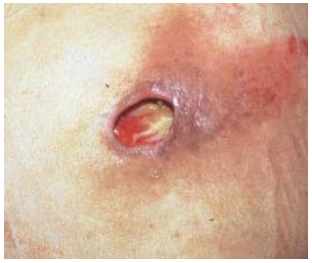
Examples of chronic wounds: mixed ulcer due to chronic venous insufficiency and peripheral arterial occlusive disease (above), venous ulcer as a result of post-thrombotic syndrome (below).

General principles of therapy

Although the appearance of chronic ulcers is very heterogeneous, the pathophysiological mechanisms leading to chronicity are very similar. All underlying vessel damage, even if of different origin, results ultimately in disorders of nutrition of the skin tissue, with increasing hypoxia and ischaemia which then results in cell death (necrosis).

This situation is the worst conceivable starting point for wound healing which, as with acute wounds, takes place in the three known phases of cleansing, granulation formation and epithelisation.





Examples of chronic wounds: trophic ulcer in diabetes mellitus (above), decubitus ulcer due to effect of pressure (below).

The repair work of the cells has to be started in a skin area which is extremely metabolically damaged, so that from the start it cannot be guaranteed that the "right cells do the right thing at the right time". However, regular wound healing is only possible if the involved cells appear in proper chronological order.

During chronic wound healing, the continuing tissue damage maintains the influx into the wound area of inflammatory cells such as neutrophilic granulocytes and macrophages. These in turn secrete inflammation-promoting cytokines which synergistically increase the production of certain proteases (matrix-metalloproteases, MMP), while the rate of synthesis of the MMP inhibitor (tissue inhibitor of metalloprotease, TIMP) is reduced. Because of the increased activity of the MMPs, extracellular matrix is broken down, and cell migration and laying down of connective tissue are disturbed.

Moreover, growth factors including their receptors are degraded on the target cells, so that the wound healing cascade cannot be continued because the mediators for the corresponding stimulation are missing. The inflammation persists. At the same time, toxic breakdown products from the tissue and also bacteria infiltrate the surrounding wound area, causing further tissue destruction and sustaining the chronicity of the wound.

According to this hypothesis, the wound healing cascade can only be restored when the vicious circle of persisting inflammation with its increased protease activity is interrupted. Two interdependent conditions appear to be essential for this:

- The blood supply and microcirculation in the affected area of skin must be normalised, in order to put an end to the defective nutritional situation which has led to the tissue destruction. In practice, this means treatment of the cause, i.e. the causes of the ulcer must be diagnosed exactly and treated adequately.
- By thorough cleansing of the wound bed, the chronic wound should be converted into the condition of an acute wound. This gives an opportunity of starting the processes required for healing in the physiologically correct cellular and temporal sequence which can then take place in an orderly fashion.

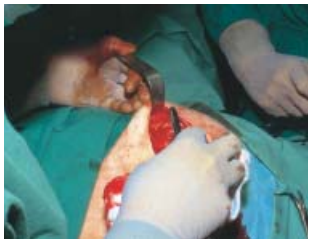
The possibilities for causal therapy such as vein surgery, compression therapy, recanalisation of lumen narrowing by dilation techniques, optimal diabetic control and relief of pressure will be listed when describing the most important types of ulcers.

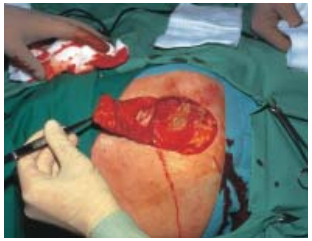
Local therapeutic methods

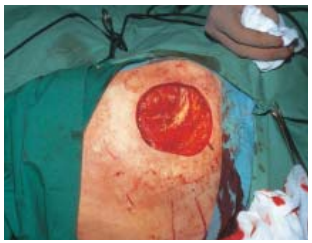
Wound cleansing

The treatment of choice to clean the wound bed is surgical or sharp debridement. It means excising of necrotic tissue exactly at the border with healthy tissue using a surgical instrument such as a scalpel, scissors, sharp curette or laser. This method is classified as selective, since healthy tissue is not damaged when it is carried out properly, or – if required for prophylactic reasons – is excised only in minimal quantities.

The advantages of surgical debridement lie, among other things, in its life-saving speed when combating severe infections. It also saves time during wound treatment. Through surgical debridement, all the factors that hinder local wound healing, such as necroses, coatings, foreign bodies, germs, etc., are thoroughly cleared from the wound. It is particularly indicated in ulcers with thick, adherent, necrotic deposits and is necessary when there is advanced cellulitis or sepsis.







Sharp debridement is best performed in the operating theatre, especially when extensive debridement is required or it is not yet clear how deeply it must extend.

Surgical debridement is a task for the doctor in both the inpatient and outpatient setting. Depending on the wound situation, a decision should be made on an individual basis as to whether the necrosis should be removed in a single procedure by operation under general anaesthetic or if a removal step by step in several treatment sections should take place. In the case of clinically apparent infection, a single-stage procedure is advisable, in order to remove the nutrient medium as quickly as possible from the infection.

Should surgical debridement not be possible due to specific situations (patient refusal, multimorbidity and poor general condition, Marcumar or heparin treatment, fever, metabolic disturbances, etc.), moist wound treatment in order to soften necroses and, if necessary, enzymatic debridement with proteolytically active substances offer an alternative. Both methods may be indicated in addition to the surgical debridement to loosen thin superficial layers of necrosis, which are impossible or difficult to remove by mechanical excision.

A range of hydroactive wound dressings are available for wound cleansing using moist wound treatment, which are effective and can be used without problems. They absorb bacteria-laden exudate, by providing moisture they promote the loosening of coatings and overall they create a physiological cell-preserving microclimate that effectively promotes the body's own autolytic cleansing mechanism.

Algorithm of treatment of chronic wounds

History and baseline diagnosis

for exact clarification of the cause of ulcer including differential diagnostic measures

→ **Causal therapy**

to restore or compensate the circulatory situation as much as possible in the diseased area of skin

Measures according to the causes, e.g.
- vein surgery
- compression therapy
- angiosurgical dilation techniques
- optimum diabetic control
- relief of pressure

→ **Wound diagnoses/assessment**

→ **Wound bed treatment/cleansing**

if possible by surgical debridement, otherwise wound cleansing by means of moist wound treatment, possibly enzymatic also

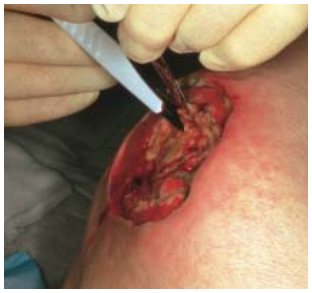
→ **Wound conditioning/promotion of granulation**

with the use of moist wound treatment

→ **Wound closure**

- by contraction and spontaneous epithelialisation
- closure by split skin grafting
- by plastic surgical procedures (myocutaneous flap)

Moist wound treatment is also regarded as selective, since only devitalised tissue is softened and cleared. Healthy tissue is not traumatised. The method is safe, free from side-effects, and simple to carry out in all the treatment domains, for example also during wound treatment at home. It must always be considered, however, that this type of wound cleansing requires more time than surgical debridement. The mode of action of the individual wound dressings is described in detail in the section headed "The wound dressing".



Necroses removal and refreshing of the wound edge with a scalpel in the case of a decubitus

In the case of very difficult infectious wounds, additional continuous irrigation with Ringer's solution through an in situ catheter has a good cleansing effect. If necessary, irrigation just at every change of dressing can be sufficient.

With the initial debridement, however, the process of cleansing and treating the wound bed in the case of chronic wounds is usually not complete, as the improvement in the nutritional state of the tissue cannot be improved promptly. Depending on the development of further necrosis or the formation of fibrin deposits, fine debridement, careful freshening of the wound edges or removal of the fibrin deposits may become necessary just as bacterially contaminated and excessive exudate still has to be removed from the wound. Correctly performed moist wound treatment is once again an adequate means for this.

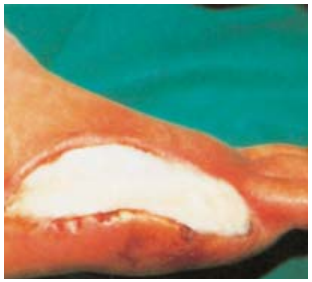
An attempt is often made precisely in the treatment of chronic wounds to curtail the cleansing and healing process by scientifically unproven use of a wide variety of measures.

It should be pointed out explicitly that disorders can arise because of the cytotoxicity and other side effects of substances used for wound treatment. Antiseptics, antibiotic-containing ointments, dyes, stain solutions, metal-containing pastes, etc., all have a more or less marked potential for wound-healing impairment. When such substances are used for short periods, it may be assumed that local damage is slight, whereas with long-term use on chronic skin ulcers, the situation is different. Healing may be significantly delayed or impaired by their undesirable effects, quite apart from the fact that the various substances may be trigger of contact allergies and the development of resistance.

Wound conditioning (wound bed preparation)

If direct surgical closure of the defect, e.g. by various flap procedures is not possible following surgical debridement, the wound has to be conditioned. That means all treatment measures which are suited to promote granulation tissue until the defect has filled up almost to the level of the skin. In the case of successful conditioning there is a fresh and clean granulating surface which represents the basic precondition for subsequent spontaneous epithelialisation or coverage by a skin graft.

The most important measure for promoting granulation growth is keeping the wound bed permanently moist by treating it with hydroactive wound dressings. This prevents the cells from dying by drying out and creates a microclimate in which the necessary proliferative cell activities can take place.

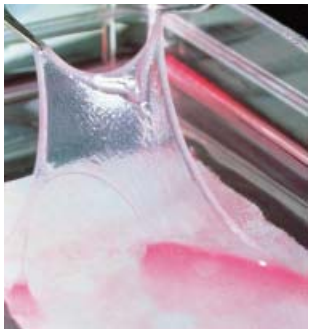


Conditioning of the wound takes place by means of a moist dressing treatment. The examples show conditioning with the calcium alginate compress Sorbalgon, which is packed in dry and then changes into a moist gel when it absorbs secretions.

Wound closure

Epithelialisation concludes wound healing. However, chronic ulcers usually epithelialise poorly. As Seiler et al. demonstrated in 1989 for decubitus ulcers, epithelial cells at the immediate edge of the ulcer demonstrate a greatly limited migration. The rate of outgrowth was only 2 – 7 %, while the rate of outgrowth of healthy skin in controls was about 80 %.

The current standard in the treatment of the epithelialising wound surface is a moist and atraumatic wound therapy. Every drying and every injury of epithelial cells during dressing changes result in the destruction of cells and thus a further reduction of this already scanty cell population, delaying the wound healing.



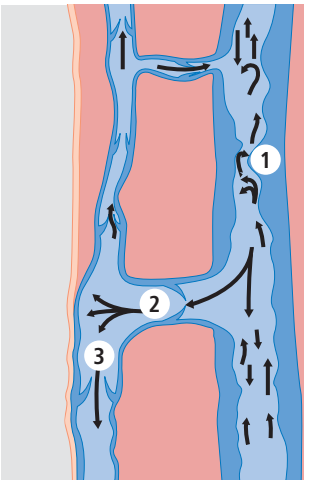
Transplantation of autologous keratinocytes cultured in a suitable nutrient solution seems to have a variety of stimulating effects in the treatment of chronic wounds.

If the tendency to spontaneous epithelialisation is poor, especially with large wound surfaces, wound closure with a split skin graft or Reverdin graft should be considered. Another possibility is grafting of autologous keratinocytes cultured in vitro. However, a prerequisite for all procedures is an adequately conditioned, well-perfused and infection-free wound base. To prepare the base for grafting or when there is no tendency to heal despite correct therapy, local application of growth factors can sometimes be worthwhile.

The venous leg ulcer

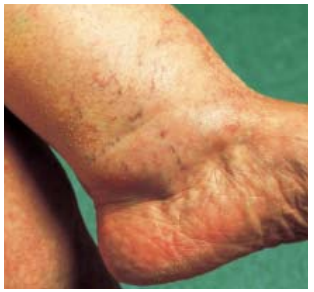
Vein abnormalities and vein disorders are among the commonest disorders of health and well-being and about 70 % of leg ulcers are caused due to vein disorders. Many ulcer patients have suffered for decades because of inadequate and frustrating attempts at therapy.

The venous leg ulcer reflects the worst metabolic disturbance of the skin and subcutaneous tissue due to chronic venous insufficiency. If the venous return of blood to the heart is disturbed (venous insufficiency), less blood is transported out of the involved venous segments and the venous pressure does not fall sufficiently (venous hypertension). This overstretching of the veins acts as a backward decompensation back to the capillaries of the final circulation. The low pressure values required for a regular metabolism can not raise, and the circulation in the vessels is slowed or even at a standstill. Metabolism, particularly in the skin and subcutaneous tissue, is impaired. In the long term, the lymphatic system is also affected by this, since it is only able to compensate in the early stages of an impaired drainage situation for fluid build-up in the intercellular spaces (interstitial fluid) by increased lymph flow.



Post-thrombotic vascular and flow situation: the deep vein is scarred and recanalised after the thrombosis (1). Blow out by dilated communicating veins (2), resulting in development of secondary varicosities (3).

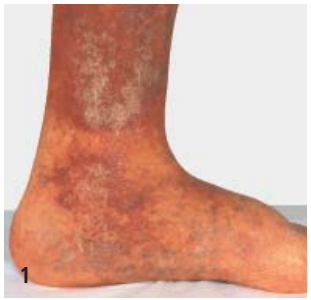
The earliest identifiable result of the disturbance in venous return is oedema, which in turn results in further rises in pressure and deposition of fluid, thus increasing the metabolic disturbances. Perivascular fibrosis and degenerative and inflammatory processes occur with trophic skin changes. Through further obliterative inflammatory processes in the venules and arterioles, a leg ulcer finally develops first in areas with poor venous haemodynamics (ankle area), as the now visible sign of decompensated venous hypertension an the metabolic disorder.

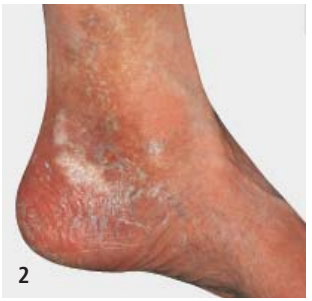


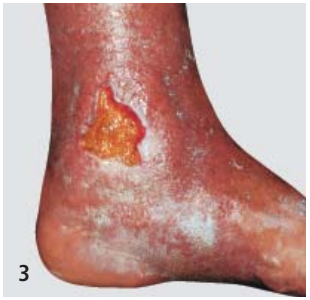
Lymphatic ankle oedema

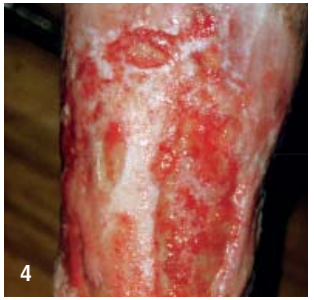
The severity, site and duration of the disorder in venous return and the degree and duration of the load on the leg vein system determine the various clinical symptoms which gradually and continually increase. They are summarised under the concept of chronic venous insufficiency (CVI) and usually classified into three degrees of severity:

- CVI grade I is characterised by venous flare around the ankles and above the arch of the foot. There is also ankle oedema.
- Grade II is expressed by hyper- and depigmentation of the skin, oedema of the lower leg and dermatoliposclerosis extending to white atrophy (also called capillaritis alba).
- Grade III is manifested as a florid or healed venous leg ulcer. It occurs mainly in the area of the ankle but can also occur at other sites on the lower leg.









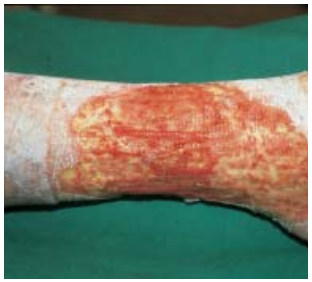
1) Marked dermatoliposclerosis in CVI grade II, which can be attributed to increasing fibrosis of the skin and subcutaneous tissue.
2) White atrophy with white atrophic skin changes.
3) Florid venous leg ulcer in grade III.
4) "Gaiter ulcer" involving the entire lower leg.

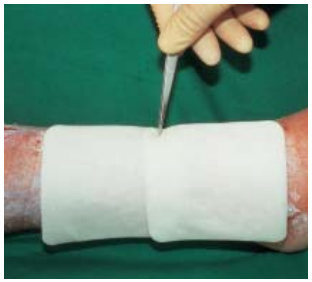
CVI can result both from primary varicose veins when the dilated lumen and valve insufficiency of the superficial leg veins extend to the perforating veins and subfascial veins, and can also be the sequela of a postthrombotic syndrome with decompensated subfascial veins. It also represents the sequela of a postthrombotic syndrome (PTS), which usually occurs as a secondary consequence of a deep leg vein thrombosis. PTS is the commonest cause of a leg ulcer (ulcus cruris postthromboticum), whereby the anatomical location of the flow impediment is a decisive factor for the clinical prognosis. In the case of primary varicose veins when the valve apparatus of the perforating veins is still functioning, ulcerations can nearly always be attributed to injuries, blunt trauma or rupture of a varix. Its prognosis is accordingly more favourable.

Diagnosis of a venous leg ulcer (ulcus cruris venosum) includes reliable anamnesis, clinical and laboratory examination with recording of the venous and arterial status as well as differential diagnostic measures to exclude factors of non-venous origin.

Ulcus cruris venosum (venous leg ulcer) is a chronic wound which heals poorly or not at all and which, because of its cause, cannot be induced to heal by local treatment alone. The venous hypertension causing the ulcer must effectively be rectified in order to improve the nutritional situation in the damaged skin area. An ulceration can only heal when the oedema has ceased and the venous outflow in the leg has again reached a compensated condition (Hach).

These therapeutic aims may be essentially achieved by compression treatment and possibly by invasive treatment methods. In modern phlebology, sclerosing therapy and operation offer mutually complementary invasive methods.





A range of hydroactive wound dressings are available for trouble-free moist wound treatment. The example shows treatment of an extensive venous ulcer with TenderWet, which brings about rapid cleansing of wounds with its "absorbing and rinsing" effect.

Which method is used depends finally on the anatomical location of the disorder in venous return and the extent of chronic venous incompetence.

Local ulcer therapy is based on proper wound treatment which is appropriately geared to the individual healing phases. During treatment, wherever possible, all factors that have a generally impairing effect on wound healing, such as infections, the influence of concomitant diseases, the side-effects of other treatments and negative psycho-social factors, must be removed.

Proper wound treatment includes, phase-adapted, thorough cleansing and conditioning of the wound and promotion of epithelisation. If the patient's medical condition allows, the most complete possible removal of necrotic tissue and inadequately perfused tissue by means of surgical debridement should be attempted. If surgical debridement is not practicable, cleansing should be carried out with moist wound treatment, which is continued in order to condition the wound bed until complete epithelisation has been achieved. Continuous compression treatment to improve the haemodynamic situation is also important.

Uncertainties in the treatment often arise with regard to prevention and treatment of infection. Bacterial colonisation of the ulcer may usually be assum, though the contamination leads – particularly in the case of purely venous ulcers – rarely to a clinically manifest infection. The generally observed low infection susceptibility of older chronic wounds also appears to apply to venous leg ulcer (ulcus cruris venosum). Prophylactic disinfection of the ulcer or topically used antibiotic therapy may therefore generally be considered as not useful, especially with regard to the potential for inhibiting wound healing of many of these substances as well as the high risk of sensitisation. In the case of severe infections and markedly increased C-RP

Diagnosis

- clinical examination
- diagnostic investigations
- differential diagnosis (arterial ulcers, venousarterial mixed ulcers, diabetic ulcers, exogenous infectious ulcers, ulcers due to blood disorders, neoplastic ulcers)

Treatment

Compression therapy

- prolonged bandaging with zinc adhesive dressings
- changes of dressing with elastic bandages
- general: the patient should move as much as possible with the bandage

Invasive therapy

- to compensate the CVI: sclerosing therapy, phlebosurgery
- to treat the ulcer: possibly paratibial fasciotomy or endoscopic ligation of perforating veins

Local ulcer therapy

- surgical debridement
- physical cleansing by moist dressing treatment
- continuation of the moist dressing treatment during the production of granulation tissue until spontaneous epithelialisation or skin grafting

Aftercare

- compression stocking to preserve the result of therapy
- avoidance of risk factors with as much movement as possible and elevation of the legs, weight reduction if indicated
- possibly drug support by anti-oedema medications

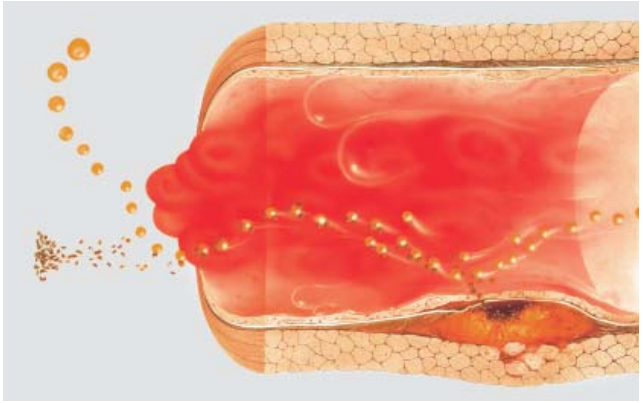
Treatment algorithm in venous leg ulcer

(C-reactive protein; indicator of inflammation) and with problem ulcers, on the other hand, systemic antibiotic therapy can be necessary.

With treatment-resistant ulceration, a vascular reconstruction may be required. Procedures with good success rates have proved to be the paratibial fasciotomy and the endoscopic ligation of perforating veins, in particular.

The arterial leg ulcer

The cause of the arterial leg ulcer is predominantly arteriosclerosis obliterans of the large and medium vessels with resulting tissue ischaemia. Sketched in outline, it originates from a lesion of the intima of the vessel wall, which produces platelet aggregation at the damaged site in reaction and in turn results in increased proliferation and migration of smooth muscle cells from the media into the intima of the vessel wall. The muscle cells produce large quantities of fibre proteins (collagen and elastin) and proteoglycans (important constituents of the extracellular matrix), which change into the so-called atherosclerotic plaques by accumulation of lipids. These plaques then lead to stenosis or



Typical of arteriosclerosis is the formation of plaques at particular foci. These arise after damage to the vessel inner wall when blood fat and calcium compounds which are transported in the bloodstream, become deposited at the site of damage.

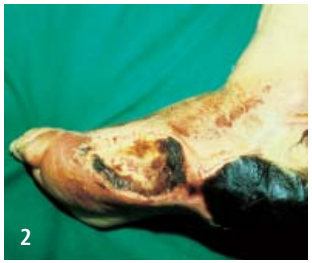
Atherosclerotic plaques (grey deposits) in an arterial wall

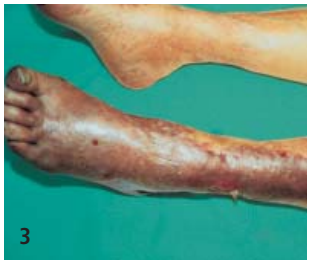
complete closure of the affected vessel, while the extent of the resulting underperfusion depends on the degree of stenosis and on the available collateral circulation.

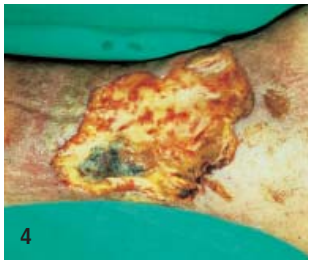
Circulatory disorders of the legs can result both from obliterative processes of the aorta itself and also of the peripheral arteries. Depending on the site of the obstruction, the site of obliteration is classified according to Ratshow, as aortic bifurcation type, pelvic type, femoral type and peripheral leg type with combinations of these being possible.

Arteriosclerosis as such is not purely a disease of old age. While there is a rapid increase in severity between the ages of 45 and 60, there is a significant range of contributory risk factors implicated in the occurrence of the illness. Apart from constitutional disposition, hypertension, diabetes mellitus, hypothyroidism, nephrosis, abnormalities of lipid metabolism, thrombophilia, respiratory insufficiency, a faulty life style with a diet high in fats and calories, overweight, stress and above all smoking are important risk factors.

Examples of arterial ulcers:
1) Toe necrosis
2) Necrosis on the lateral border of the foot and region of calcaneus and Achilles tendon
3) Complete gangrene of the lower leg
4) Mixed leg ulcer of the lower leg







Men suffer from obliterative arteriosclerosis about 5 times more often than women, though the sex differences level off in older age groups.

Furthermore, it is of importance for the occurrence of the illness that a conjunction of several risk factors causes the risk of the disease to rise nearly exponentially when a single risk factor such as diabetes mellitus can multiply the probability of developing arterial obstruction of the lower limbs. It is thus a very complex disorder that demands treatment of all the factors which have a negative influence.

Sites of predilection for arteriosclerotic ulcers on the foot are the distal phalanges of the toes and nails, the nail bed and the heads of the 1st and 2nd metatarsals. They often arise due to pressure of the shoe on bony prominences and are apparent as haemorrhages which appear dark blue to black. Another frequent cause of ulcers are lesions from incorrect pedicure or trivial injuries of the toes.

Necrosis due to severe circulatory disorders are situated mostly on the lateral foot edge, the heel, in the interdigital space and on the extensor sides of the lower leg. In the differential diagnosis of the venous ulcer, there is pain in the area of the ulcer. In diabetics, the ulcer is also distinguished as an angiopathic or a neuropathic form (see from page 117). The degree of wound severity can be divided into six grades according to the classification formulated by Knighton for chronic wounds into stage I to VI.

In the initial stages, prompt recognition facilitates therapy and improves the prognosis, while a detailed history should pay attention to the typical features of claudication pain. The clinical staging of occlusive arterial disease is modified after Fontaine:

- Stage I: asymptomatic, possibly slight fatigability
- Stage IIa: onset of pain after walking 200 m
- Stage IIb: walking distance less than 200 m
- Stage III: rest pain
- Stage IV: continuous pain, necrosis, ulcer, gangrene.

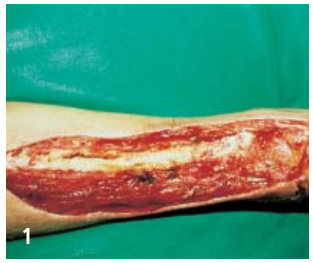
After making the diagnosis and localising the site of obliteration, a plan of treatment must be drawn up, taking into account the various pathogenetic factors where possible. It contains:

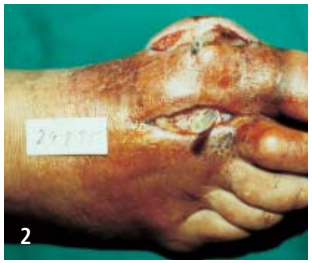
- elimination of risk factors
- treatment of concomitant illnesses (e.g. achieving normal blood sugar levels in diabetes mellitus)
- measures to restore or improve the circulation by vascular surgery, angiology and interventional radiology
- local wound treatment

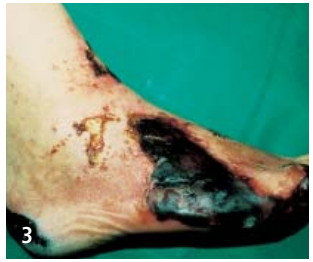
In the hierarchy of treatment measures, reconstructive arterial procedures and angioplasty are the most important methods of treating the primary cause of the arterial leg ulcer. The choice of the surgical procedure should be guided by the location and extent of the arterial occlusion as well as by the general condition of the patient. Apart from revascularisation, drugs may be considered to improve perfusion, which can influence in particular the hyperproliferative cell processes and the flow characteristics of the blood, e.g. prostaglandin E1.

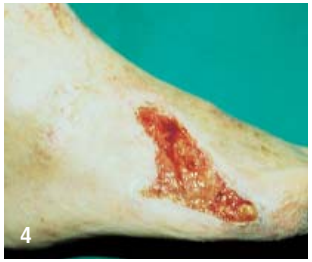
During local wound treatment, the basic risk must be borne in mind that the tiniest injuries in a patient with occlusive disease, ignored or trivialised at first, can extend rapidly within a few days.

A further central problem is the high risk of infection of arterial ulcers. Accordingly, surgical debridement serves for rapid treatment of infection. Necrotic areas must be removed, loculations opened up widely, sloughy deposits removed and infected areas excised. Free flow of secretions is ensured by drainage (osteomyelitis suction/irrigation drain).









Examples of treatment:
1) Occlusive arterial disease of femoral-lower leg type stage IV, condition after surgical debridement, posterior tibial saphenous vein bypass.
2) Diabetic gangrene with occlusive arterial disease of femoral-lower leg type stage IV, condition after incision and drainage.
3) Dry gangrene in the area of the 4th and 5th rays, of the lateral edge of the foot, in the calcaneus and in the dorsal area of the foot.
4) 4 months later following removal of necroses and amputation of the 4th and 5th rays.

Treatment algorithm in arterial leg ulcer

Diagnosis

- determine severity of peripheral arterial disease and site of obstruction
- evaluation of concomitant illnesses/risk factors (hypertension, diabetes mellitus, abnormalities of lipid metabolism, smoking, overweight etc.)

Treatment

Causal therapy

- removal of risk factors (avoid smoking, alcohol consumption)
- treatment of concomitant illnesses (reduce high blood pressure, obtain normal blood sugar etc.)
- measure to restore or improve the circulation (angioplasty/vascularsurgery; medical procedures, leg lowering, vessel training)

Local ulcer treatment

- surgical debridement
- treatment of infection (systemic antibiotic therapy)
- moist dressing treatment for further wound cleansing, conditioning and epithelialisation
- if amputation is indicated:
  - heal infection as much as possible
  - convert wet to dry gangrene
  - attempt maximum possible revascularisation

Aftercare

- train patients, reinforce their own responsibility
- orthopaedic shoes with appropriate distribution of pressure
- inspect feet daily for changes (callosities, fissures, fungal infections of the nails)
- do not use any cutting implements for foot care, foot baths at body heat only, do not go barefoot, use no outside heat sources to promote perfusion (hot water bottles, heated pads) but only body warmth (socks, fleece boots)

After surgical debridement, wound cleaning and conditioning are continued using moist wound treatment. Antiseptic dressings may be indicated until the infection subsides.

If amputation is necessary, this should be performed if possible after resolution of the concomitant infection, after conversion of a wet gangrene into a dry gangrene and achievement of maximum revascularisation in the necrotic border zone.

The diabetic ulcer

Diabetes mellitus is a chronic disruption of the carbohydrate metabolism and has reached almost epidemic proportions worldwide. In Germany currently, some 300,000 people suffer from type I diabetes and about 4 or 5 million are affected by type II diabetes. Since type II diabetes is partially age-dependent, increasing numbers of diabetics can be expected, purely due to changing age patterns.

Among the complications of diabetes, diabetic foot syndrome (DFS) takes a prominent position. According to epidemiological surveys, it may be assumed that approximately 15 % of diabetes mellitus patients suffer from foot lesions of differing severity and characteristics during the course of their disease, and these all too often end in amputation.

The basic precondition for the occurrence of diabetic foot lesions is the presence of diabetic (poly)neuropathy and/or peripheral arterial disturbed blood circulation. Although the statistical surveys differ somewhat, the following distribution can be taken to apply: in about 45 % of cases, diabetic neuropathy is the cause, whilst in a further 45 %, the aetiology is a mixture of neuropathy and disturbed blood circulation and, in 10 % of cases, isolated peripheral disturbed blood circulation alone.

	Neuropathic foot	Angiopathic-ischaemic foot
Anamnesis	Diabetes mellitus for many years, possibly additional alcohol consumption, further delayed damage from diabetes	Diabetes mellitus for many years, possibly lipid metabolism disruption, heart diseases, nicotine abuse and arterial hypertension
Clinical presentation		
Skin colour/temperature	rosy, warm	pale to livid (position-dependent), cool
Sweating/secretion	disturbed; dry, cracked skin	atrophic skin, loss of skin appendages (hair loss)
Sensibility	reduction or loss of perception of vibration, pain, pressure, temperature, touch; reflexes impaired	unaffected, sensation present
Pain	pain at rest or at night	present, claudicatio intermittens; pain symptoms
Foot pulse	palpable	not palpable
Hyperkeratosis	frequently at sites exposed to pressure	relatively minor
Bone deformation	frequently changed bone structure, early osteolysis	rarely
Predisposed sites of lesions	foot sole, in particular in region of metatarsophalangeal joints	acral necroses

Occurrence of a neuropathic lesion

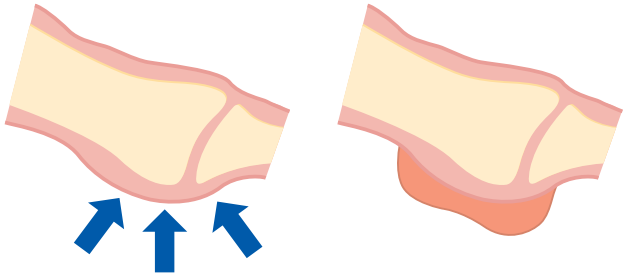
Diabetic neuropathy, characterised as increasing saccharification of the nerve cells and progressive damage to the nervous tissue affects autonomous, sensory and motor fibres equally. Clinically, these types of damage lead, alone or together, to the characteristic changes in the foot of a diabetic person:

- Damage to the autonomous fibres causes a reduction in sweat secretion with atrophic dry warm skin.
- Sensory function impairment brings about reduced pain and temperature sensations or loss of pain sensations.
- Reduction in motor neural activity leads to atrophy of the foot internal musculature with static changes and defective regulation of the foot motor function.

This produces the conditions for development of a neuropathic ulcer, whereby callus formation on the foot sole is a possible indicator of impending ulceration. The reason for this is that enhanced cornified skin formation (hyperkeratosis) leading to formation of a callus results as a reaction to the effect of increased pressure on the foot sole (with the preferred localisation being the metatarsophalangeal joints). The callus then conducts the pressure forces into deeper tissue layers lying beneath the skin.

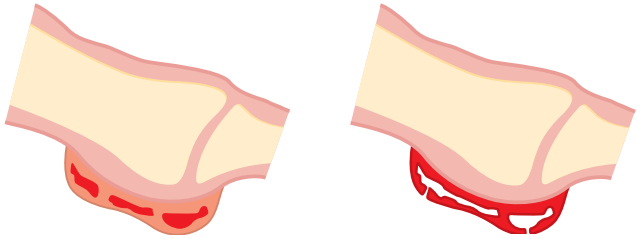
At the same time, due to increased pressure and shearing forces, detachment of the cutis and subcutis occur in the hyperkeratotically changed skin, with formation of fissures, haemorrhages and haematomas, which later become colonised by bacteria. As a result, a central, infected tissue defect, "mal perforant du pied" (Malum perforans pedis) develops.

Ulcer formation can also be triggered by other traumas. Among these is the unphysiological pressure loading due to ill-fitting footwear, also pressure points due to in-growing toenails, minor injuries, such as, from cutting, sharp devices used for foot care or thermal trauma, e.g. from excessively hot footbaths.



Excessive pressure and shear forces due to changed static forces in the foot and changed motor function...

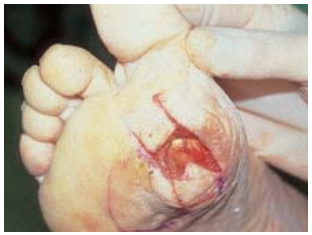
lead to hyperkeratosis and callus formation,...



cracks, haemorrhage, haematoma and bacteria colonisation,...

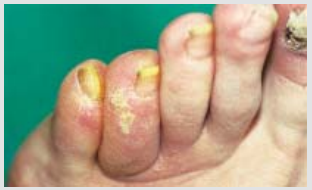
and finally to an infected defect, known as a "mal perforant"

The formation of a "mal perforant"

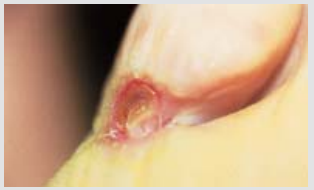


Added to this is the difficulty that the processes of ulcer development are often barely noticed by the person affected, since pain perception is impaired. This therefore often brings about a risky temporal delay since, with the diabetic patient's generally weakened infection defence, the initially localised infection can rapidly spread in depth and jeopardise the main anatomical structures (tendons, muscles) and bones (bacterial osteitis). Inflammation of the bones can even lead to the total collapse of the foot skeleton. The consequence is that Charcot's foot, or deep inflammation of the foot tissues (pedal phlegmon) arises, which endangers the blood circulation to the toes, so that finally, diabetic gangrene threatens to develop.

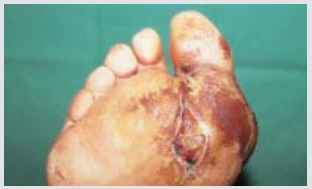
In order to plan treatment and assess the prognosis, a precise description and classification of the various lesion types is indispensable. This also serves to ensure clear communication between the different individuals involved in the necessarily multidisciplinary treatment. A variety of classifications are available for describing the lesions, of which the so-called Wagner classification is the most widespread classification of diabetic foot lesions throughout the world. With its six stages (0 to 5), it has the advantage that is simple to use in clinical practice.



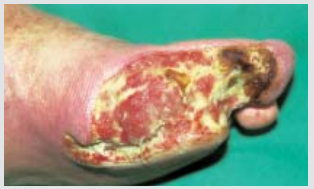
Degree 0: no lesion, possibly deformation of foot or cellulites



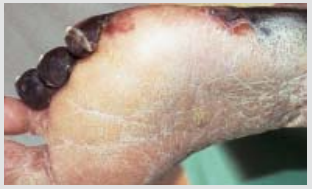
Degree 1: superficial ulceration



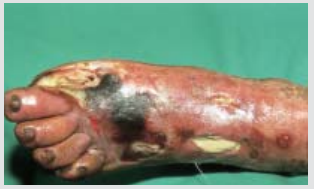
Degree 2: deep ulcer reaching to joint capsule, tendons or bones



Degree 3: deep ulcer with abscess formation, osteomyelitis and infection of joint capsule

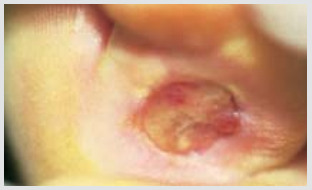


Degree 4: limited necrosis in forefoot or heel region

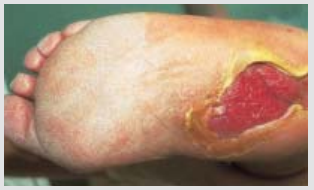


Degree 5: necrosis of entire foot

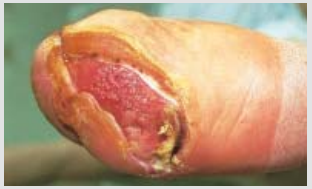
Stages of a malum performans, according to Arlt



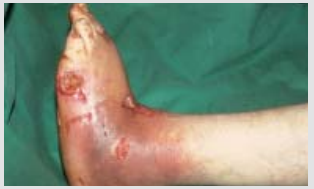
Stage 1: necrosis of epidermis (pressure point)



Stage 2: malum perforans extending subcutaneously as far as the bones or joints, but without lesions in these



Stage 3: malum perforans with bone and/or joint involvement



Stage 4: infection no longer restricted to a region spreading from a malum perforans

The first signs of neuropathic disruptions in the legs are dry skin, burning and tingling sensations and pains at rest, particularly at night. However, there is hardly any pain sensation from injuries.

Occurrence of an angiopathic-ischaemic lesion

Reduced blood circulation in the tissue due to micro- or macroangiopathy is a serious risk factor for the development of a diabetic foot ulceration and impairs the healing of existing ulcerations.

The macroangiopathy of the diabetic person, which has no independent existence either from the histological or from the histochemical standpoint, can be characterised as an advanced, particularly severe type of arteriosclerosis. This sclerosing of the arteries ages an individual by 10 to 15 years relative to a person of healthy metabolism, with the result that diabetic person suffer cardiac infarctions, strokes and occlusions in the legs earlier and more often than persons of healthy metabolism.

Microangiopathies are diseases of the terminal vessels and are grouped together as microcirculation disruptions. They involve, in particular, vessel wall restructuring, blood flow properties and conditions, and metabolic processes at the interstitium and in the peripheral portions of the lymphatic system. The aetiology is still unclear, although the metabolic theory remains at the forefront of pathogenic observations.

The preferred sites for ischaemic diabetic ulcer are similar to those for arterial ulcer: the end phalanges of the toes and the nails, the nail beds and the heads of metatarsals I and II. Necroses resulting from the most severe blood circulation insufficiency are usually located on the lateral foot edge, the heel, in the interdigital spaces, and on the extensor sides of the lower leg. Not uncommonly, traumatic events, such as pressure points from shoes, improperly

performed pedicures and other minor injuries of the toes also contribute to the development of ulcers.

Before ulcerations ever develop, inspection can reveal trophically disturbed nails, mycoses, reddening, marbling and loss of hair, which illustrates how regular inspections can contribute to prevention.

The principles of treatment

The aim of treatment for diabetic foot syndrome is primarily a reduction in amputations, preserving function in the extremities and maintaining the quality of life of the diabetic patient. Treatment is an interdisciplinary task and success is only possible with broadly spread measures. The specialists involved are internal medicine specialists, vascular surgeons, orthopaedic specialists, neurologists and dermatologists.

A basic measure in the treatment of all diabetic lesions is optimal adjustment of the diabetes (normoglycaemia), which is also the best treatment for the neuropathy. Further conservative treatment is focused on improving the central haemodynamics (treatment of cardiac insufficiency or ventilation disruption, blood pressure regulation), the haemorheology and vasodynamic (blood flow/conditions) as well as the anticoagulation.

A primary and central problem in the treatment of diabetic ulceration is the extraordinarily high risk of infection. Only very few angiopathic lesions show no sign of surrounding infection. Mixed forms of neuropathic and angiopathic foot and purely neuropathic ulcers, however, can generally be assumed to be infected. The opportunities for spreading of an infection in the foot are particularly favourable, due to the differentiated connective tissue apparatus, so that consistent systemic antibiotic treatment is always worthwhile.

Diagnosis

exact verification:
- of underlying cause (according to symptoms of neuropathy and angiopathy, mixed ulcer)
- of the immediate trigger of the lesion (injury, infection, etc.)
- of the metabolic situation of the diabetes
- of the inflammatory parameters

Treatment

Causal treatment

- optimum adjustment of diabetes

Local ulcer treatment

- treatment of infection (systemic antibiotic therapy)
- absolute relief of pressure on ulcer until healed (walking aids, wheelchair, bed rest)
- adequate surgical debridement
- moist dressing treatment for further wound cleansing, conditioning and epithelialisation

Aftercare

- train patients, reinforce their own responsibility
- orthopaedic shoes with appropriate distribution of pressure
- inspect feet daily for changes (callosities, rhagades, fungal infections of the nails)
- do not use any cutting implements for foot care, foot baths at body heat only, do not go barefoot

Treatment algorithm for neuropathic ulcer

Treatment algorithm for angiopathic ulcer corresponds to that for Ulcus cruris arteriosum (arterial leg ulcer)

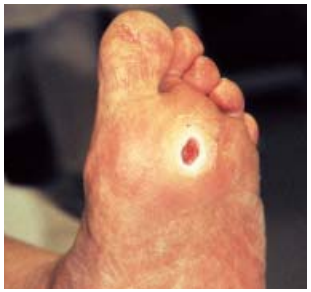
The following therapeutic principles may be formulated for the local treatment of the neuropathic ulcer:

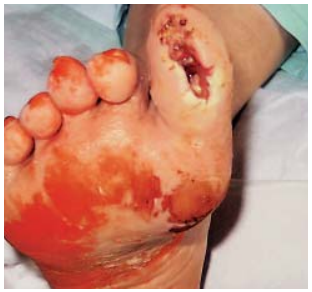
- absolute removal of pressure on the lesion (walking aids, wheelchair, bed rest)
- correct wound treatment with adequate debridement and moist dressing treatment until there is complete wound closure by strong epithelium
- treatment with suitable orthopaedic footwear
- specialised aftercare, training of the patient and prevention of recurrence

Despite all difficulties, a neuropathic lesion always implies a prospect of wound healing, so that, where possible, after surgical debridement, a conservative procedure is primarily indicated for conditioning of the wound area. The most frequent local surgical measure for correcting pressure points that hinder wound healing is resection of the metatarsal head.

The angiopathic gangrene in the presence of arterial occlusive disease requires a different approach, which depends essentially on the vascular status and on the result of revascularisation. In contrast to the neuropathic foot lesion, amputation are not easily avoided.

Diabetic ulcers of neuropathic genesis under conservative treatment





The methods for treating the wound ground, in principle, are surgical removal of necrosis, border zone amputation with extensive secondary wound healing as well as amputations through the classical amputation lines with primary wound closure. Determination of the treatment measures in each case requires clinical experience. The decision should be made after careful consideration and should not be hasty. The supreme goal of treatment is preservation of the extremities.

If surgical removal of necrosis is sufficient, this should be regarded as the method of choice. Even if secondary healing can possibly take months, the result obtained in this manner is still the best. With good prophylaxis against infections, the patient can put pressure on the foot – in contrast to the neuropathic foot – in the case that the wound is free of necrosis. The so-called vascular training favours revascularisation and wound healing.

Border zone amputations are always required when bony parts of the foot lie within the necrotic area. Yet the point of time for amputation should only be determined if an extensive demarcation of the findings is clear. Demarcation denotes the clearly visible border between black (dead) and healthy tissue. Operations in inflamed tissue often result in secondary necrosis due to wound oedema when blood circulation is reduced. When determing the amputation line, the subsequent possibilities for prosthetic or special shoe provision should be kept in mind.

The Decubital ulcer

A decubitus is defined as damage to the skin as a result of continuous local pressure. Its occurrence can be sketched out schematically as follows: when sitting or lying, the human body exerts pressure on its contact surface, which in turn exerts a counterpressure on the skin area of contact. The size of the counterpressure depends on the hardness of the contact surface, although normally it is above the physiological arterial capillary pressure of ca. 25 – 35 mm Hg.

Pressure / pressure rest time

↓

local blood circulation insufficiency

↓

oxygen shortage / build-up of toxic metabolic products

↓

increase of capillary permeability, vascular dilatation, cellular infiltration, oedema formation

↓

vesiculation

↓

total ischaemia, irreversible death of skin cells

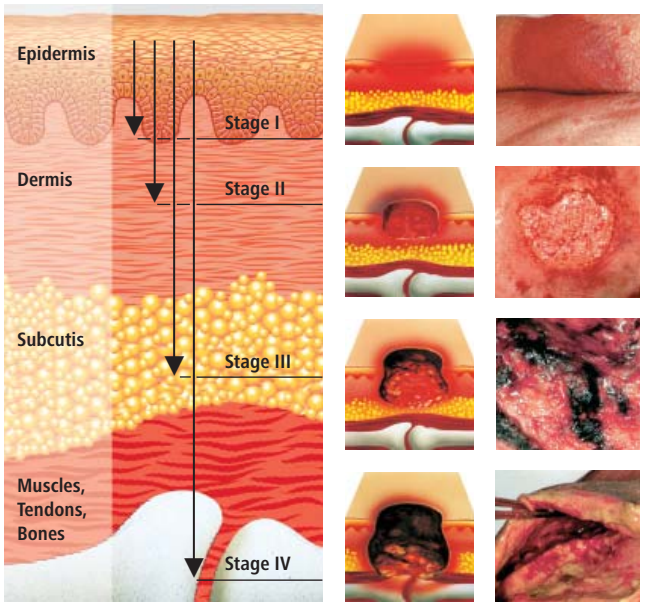
↓

Ulcer / necrosis

In the short term, the skin itself can tolerate the application of relatively high pressures. However, if the pressure is maintained, due to compression of the capillaries conveying the blood in the affected skin area, blood circulation insufficiency and oxygen shortage (hypoxia) come about. The body reacts to this nascent damage in the form of a warning pressure pain. In a healthy person capable of movement, this is the trigger to relieve the compressed skin areas by a positional change.

If a person is not able to perceive this pressure pain due, for example, to complete immobility from unconsciousness or narcosis, or due to relative immobility as a consequence of severe pain, fever, dementia, age-related weakness, etc., then the compression of the skin area is sustained. The blood circulation insufficiency worsens and leads to a build-up of toxic metabolic products in the tissues and increased capillary permeability, vascular dilatation, cellular infiltration and oedema.

Assuming the affected skin area is completely relieved of the pressure, the cells are still able to regenerate at this time, since the inflammatory reactions favour the removal of toxic metabolic products. However, if the pressure is sustained, as a consequence of the further increased ischaemia and hypoxia, irreversible skin cell death with necroses and ulceration formation take place.



Classification of decubital ulcer:
Stage I: Sharply defined reddening of intact skin, which does not blanch on pressure. As a guide: overheating of the skin, hardening or oedema.
Stage II: Partial skin loss of the epidermis up to the dermis. This is a superficial ulcer which appears clinically as an abrasion, blister or flat crater.
Stage III: Damage to all skin layers (epidermis, dermis, subcutis) which can extend to fascia, although these are not yet involved. The ulcer appears as a deep, open ulcer with or without undermining the surrounding tissue.
Stage IV: Skin loss over the entire thickness of the skin with extensive tissue necroses and damage to muscles, tendons and bones. Even undermining and pocket formations occur often. In stage III and IV risk through septic complications!
(according to "National Pressure Ulcer Advisory Panel", 1989)

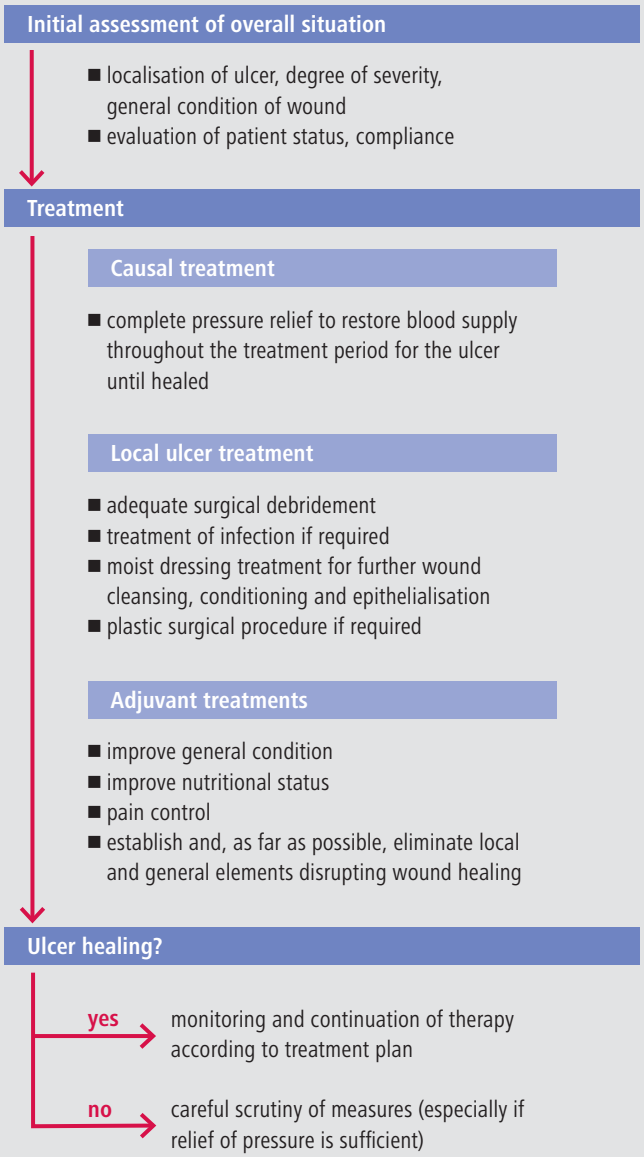
The time for which the skin tissue can survive under ischaemic application of pressure without damage is about two hours. However, this tolerance range is subject to great variations from one patient to the next. It is influenced by the severity of the applied pressure, and the general condition of the skin. A young, elastic skin is more resistant to pressure than the thinner aged skin. Furthermore, any disease associated with acute or chronic hypoxic conditions of the skin cells, or external damage to the skin is significant.

Decubital ulcers can in principle develop in any sites of the body. However, the greatest risk occurs when the pressure on the body, and the counterpressure from the surface below, act on a skin area lying over a bony prominence which is not cushioned by subcutaneous fat tissue. Therefore the classical sites are: the sacral region, heels, ischia, greater trochanters and lateral malleoli. About 95 % of all decubital ulcers occur at these sites.

Apart from perpendicular application of pressure on a skin area, a risk also exists from shearing forces. The term shearing implies tangential displacement of the skin layers over one another, by means of which blood vessels are also narrowed and compressed. It is above all the region of the buttocks that is subject to tangential shearing forces, for instance when the patient is pulled into a new position instead of being lifted, or if he slips when sitting up in bed, due to insufficient support for the feet.

The treatment of the decubitus is based on three therapeutic principles: The supreme requirement of every decubitus treatment is restoration of the blood supply of the damaged skin area by a complete relief of the pressure. Without relieving the pressure, healing is not possible and all other measures may fail. Therefore the relief of pressure must be maintained throughout the entire period of treatment. Every pressure, even if only for a few minutes, causes fresh damage and leads to relapses in the healing process.

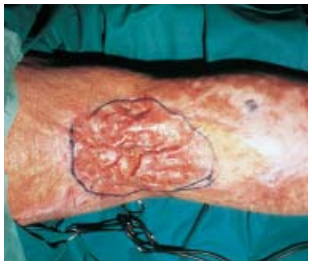
Local wound treatment includes thorough debridement, surgical if possible, as well as continuing wound cleansing with hydroactive wound dressings. This is followed by conditioning of the wound with formation of granulation tissue as well as eventual epithelialisation by moist wound treatment.

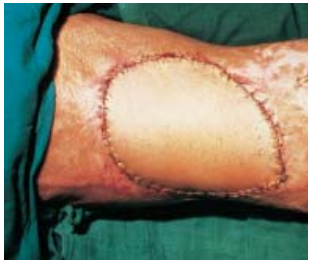


Treatment algorithm in decubital ulcer

As the case may be the long lasting and stressing healing times can be reduced by plastic surgical covering of the decubitus. But even in this case previous wound conditioning is recommended to ensure the surgical result and avoid recurrences.

As the third therapeutic principle, adjuvant therapies are indicated to improve the patient's general condition and the nutritional status as well as the pain control. Cachexia with conditions of protein deficiency, which inhibit wound healing, is often observed in elderly patients so that adequate nutritional intake and a sufficient supply of vitamins and minerals must be ensured.





Unstable scar in the popliteal fossa after burn as a special form of chronic post-traumatic wound (above) and its treatment by microsurgical scapula flaps (below)

The chronic post-traumatic wound

The chronic post-traumatic wound occurs as a result of inadequate primary treatment of an injury or due to complications during primary management which were not rectified in the immediately following phase of treatment. Typical causes of post-traumatic wounds taking a chronic course are soft tissue contusions, degloving injuries, skin necrosis, osteitis, infected implants, joint arthroplasty infections, joint infections or deep soft tissue infections. Frequently, this development can be attributed to an initial underestimation of the soft tissue injury underlying the initial trauma. Among primary injuries, the compound fracture is particularly difficult. Contamination can cause soft tissue and bone infections which often prove to be severe.

The unstable scar, such as that found in areas subject to mechanical stress after wounds have undergone secondary healing or after split skin grafting occupies a special position. With this type of scar, the integrity of the skin is not broken, but ulceration easily occurs with a corresponding risk of infection, so that treatment of the soft tissues is required in this situation also.

	Post-traumatic ulcers	Radiation damage	Tumour wounds
Cause	inadequate primary therapy, e.g. of soft tissue contusions; complications of wound healing which were not dealt with immediately, such as necrosis, infections, unstable scars etc.	ionising radiation, sometimes in association with other risk factors such as trauma, chemical factors infections etc.	benign, malignant or semimalignant cell growth
Causal therapy	removal of focus of infection where necessary and antimicrobial treatment	adequate tumour therapy and/or therapy of concomitant risk factors as needed	tumour therapy as needed
Treatment	cleansing as early as possible by radical debridement and plastic reconstructive procedure	cleansing as early as possible by radical debridement and plastic reconstructive procedure	if radical therapy is possible, the wound caused by the tumour is converted into a surgical wound and treated as such

Overview of causes, causal treatments and treatment of other chronic skin ulcers

The goal of all measures to treat a chronic post-traumatic wound is stable soft tissue cover. Debridement and the removal of all areas of necrosis and foci of infection in turn represent the first step. It can sometimes be impossible to take into consideration functional structures such as tendons, fascia and even nerves and vessels. A soft tissue situation must be created in which it is possible to cover the defect without a risk of persisting necrosis and thus persistence and spread of infection.

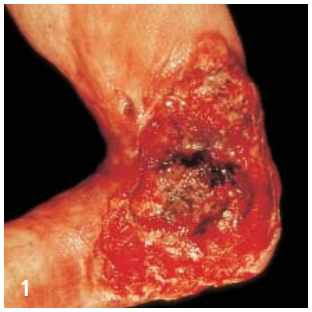
Planning of later reconstructive procedures must be included during the debridement operation. A decision must be made early as to whether both soft tissue and bone defects can be closed in a single session or whether individual reconstruction steps should be postponed, to be performed later when the soft tissues have healed.

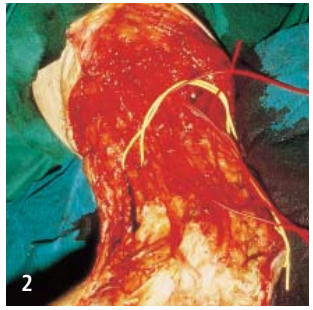
Overall, the time factor should not be overlooked in planning. Bones and tendons exposed after debridement can become infected secondarily or can dry out. As a rule, definitive wound closure can be undertaken two days after the first debridement during a planned second look. Plastic reconstructive methods are required for soft tissue cover, ranging from simple split skin graft to free microsurgical flap transfer. Skin grafts always need a clean granulating surface with covered functional structures and no mechanically stressed regions.

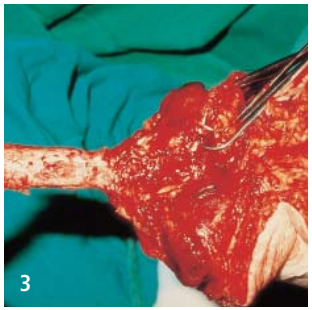
Chronic radiation damage

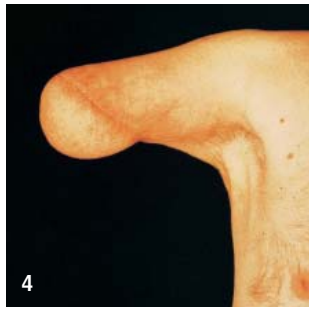
Treatments with ionising radiation lead to inevitable damage to the skin and the underlying tissue. Even though this damage does not have to be visible macroscopically, the first sign of the chronic sequelae of radiation is telangiectasia, which can be interpreted as re-generation of destroyed capillaries.

The skin and subcutaneous tissue are not as well perfused after exposure to radiation and undergo secondary atrophy. The skin becomes thinner and is bound firmly to the underlying structures due to the loss of the subcutaneous fat. In addition, there is general tissue fibrosis and direct cell damage with chromosomal changes. Local lymphoedema, increasing hyalinisation at the expense of elastic fibres and thrombosis in the arterioles and venules lead ultimately to local disturbances of nutrition and thus to a poorly healing ulcer. These ulcers, in the worst case, may undergo malignant transformation after a latency of 4 to 40 years.









A 64-year-old patient developed a squamous cell carcinoma after irradiation of a haemangioma in his youth, which led to amputation of the arm.
1) Preoperative appearance
2) and 3) In order to obtain a stump capable of taking a prosthesis, a pedicled latissimus dorsi flap was wrapped around the upper arm...
4) ...and a stress-stable amputation stump was obtained.

If an initially stable area of irradiated skin suddenly becomes unstable, recurrence of the primary tumour or a malignant neoplasm due to the radiation can be the reason. Skin metastases occur preferentially in irradiated areas of skin. Other causes for such a development are trauma such as injections, biopsies, insect bites or chemical factors such a topical therapy, local long-term irritation or occupational exposure to hazardous chemicals. Skin infections, osteomyelitis and non-infectious skin diseases, such as varicose vein disease and varicose dermatitis can also produce chronic injury, as can internal illnesses such as diabetes mellitus or arteriosclerosis.

The indications for surgical treatment consist of the resection of local recurrences, resection of an unstable scar or resection of the radiation-damaged skin to relieve pain, facilitate nursing and improve the patient's quality of life.

The surgical treatment of the consequences of radiation firstly requires radical debridement with histological examination of the resected tissue, the resection edges and depth. This can also require resection of bone, including ribs, sternum or the entire chest wall. Without such debridement, osteoradionecrosis in particular cannot be treated. As direct wound closure should usually not be attempted and cover with a split skin graft is also often insufficient, well-vascularised skin (muscle) flaps should be considered to cover the often large defects.

It must be noted that many radiation injuries are often treated conservatively and therefore undergo surgical treatment too late. Experience has shown and it should be borne more in mind that ulcers in irradiated areas as a rule do not heal with conservative treatment and that chronic ulcers in this situation can all too easily become the site of secondary malignancies. Quite apart from the fact that the patient's suffering can be cut short, early surgical treatment in many cases obviate the need for complex reconstructions.

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