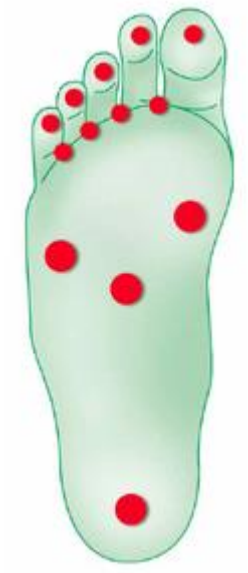
DIABETIC FOOT

Aetiology of diabetic foot

There are many factors contributing to the development of a diabetic foot ulcer. The main causes, however, are nerve damage due to impaired metabolic control in the diabetic patient (so-called polyneuopathies* , PNP* , or impaired arterial blood flow, peripheral occlusive arterial disease, pOAD*) to the lower extremity. A combination of pOAD* and PNP* presents a particularly serious clinical picture with a poor prognosis.

Impaired arterial blood flow in the large vessels (also called macro-angiopathy) and the small vessels (micro-angiopathy) is caused by deposits on the walls of the vessels (arteriosclerosis) which may lead to narrowing or even occlusion of these vessels. Occlusive arterial disease in the pelvic and leg arteries occurs in diabetics at an early age and is 6 times more prevalent in diabetics than in non diabetics[4]. 24% of diabetic patients develop pOAD* within the first 11 years of the disease[1]. This results in disturbed blood circulation to legs and feet although the arteries of the feet as such are frequently unaffected.

Other pathogenicity factors to be mentioned in this context are osteoarthropathy (changes to bones and joints caused by neuropathy) and infections.



Polyneuropathy*

With nerve damages a distinction is made between sensory, motor, senso-motor, and autonomous neuropathy. Bascially, a combination of these forms is regularly encountered in routine daily practice.

Sensory polyneuropathy

Damage to the sensitive parts of nerves can lead to unpleasant sensation deficits in the diabetic foot such as formication, pins and needles or stabbing pain especially at rest or during the night. On the other hand the perception of actual stimuli such as temperature, vibration, touch, pressure, in-depth sensitivity and wound-related pain is either diminished or disappears completely. This results in a high risk of foot trauma in such patients. These deficits irradiate upwards from the toes to the lower leg. Footwear that is too narrow yet frequently felt to be comfortable because of the missing sensation of pressure on the foot as well as small wounds are often overlooked or identified at a late stage. Non-painful wounds are classed subjectively by the patient as a trivial injury. Delayed onset of treatment or limited patient compliance are therefore common complaints. Neuropathic ulcers often develop on the heels, soles of the feet, the head of the 1st and 5th metatarsal bones*, between the toes and on the tips of the toes *(see graph).*

Motor neuropathy

Nerve damage to the muscular section of the foot and the lower leg is frequently associated with sensitive trauma as described above. In this case reference is made to motor neuropathy. The small foot muscles consequently become crooked resulting in loss of foot stability and foot deformation. The head of the metatarsal bone* bulges out over the sole of the foot.

Flexion contracture occurs in the middle or end joint of the toes, and is associated in some cases with overstretching of the phalangeal joint (digitus malleus*). Another aspect in this context is the effect of the limited joint mobility* caused by diabetes. These foot deformities persistently change the walking and pressure profile of the sole of the foot. The skin attempts to counteract locally raised pressure with increased callus formation. Thus calluses* or corns (clavus*) tend to develop on the foot in those areas exposed to pressure, and they do not increase but rather reduce the skin‘s padding capacity. Blisters form beneath the calluses due to the pressure build-up. Left untreated, these blisters may result in bleeding and ulcer formation. Such typical, painless tissue defects with keratinized wound edges are known as ulcera malum perforans.

Autonomous neuropathy

The most obvious change in the diabetic foot with this type of nerve damage is the lack of activity or reduced activity of the sweat glands manifesting in a dry, flaky skin. It should be noted that the skin on the soles of the feet as on the inner surface of the hands has hardly any sebaceous glands. The moisture content of the skin is maintained mainly through foot perspiration. Skin that has dried out loses its suppleness and thus its ability to compensate pressure and shearing forces to a certain extent, resulting in fissures in the skin (rhagades*). The barrier function in

general and against microorganisms is adversely affected. Further consequences of autonomous neuropathy include dilatation of the arterial vascular system and in connection with this an arteriovenous shunt in the region of the lower leg and foot leading to increased blood flow in the foot. This is associated with redness and overheating throughout the entire foot. Oedema formation at the back of the foot can also be seen. The skin is dry and flaky. Defective sweat production and overheating exacerbate the drying out of skin. At the same time reduced mobility is observed in the foot joints which is presumably due to a structural change of proteins (protein glycation) in joints, tendons, soft parts, and skin (limited joint mobility* , see "**Motor neuropathy**").

These serious structural changes considerably inhibit the foot‘s biomechanical function to atraumatically absorb pressure on walking and running. On the one hand, the various changes in the shape of the foot trigger increased pressure at exposed sites and, on the other hand, the ability of the soft tissue and foot bones to offset pressure is compromised. In the case of advanced denervationin the foot, the formation of blisters or ulcers is also accompanied by foot fractures that go unnoticed by the patient due to the absence of any warning signs such as pain. The subsequent course is characterized by massive bone changes to the foot arch and the instability of various joints, possibly culminating in the onset of the full picture of diabetic neuropathic oesteoarthropathy* (DNOAP; Charcot‘s foot*).

Peripheral occlusive arterial disease

Arteriosclerosis either diminishes or disrupts blood flow (ischaemia*) due to the narrowing or occlusion of the arteries. These changes are much more common in diabetics than in nondiabetic patients. Younger patients are affected and the disease advances more rapidly. pOAD* of the lower leg is characteristic of diabetes mellitus.

In routine daily practice the classification of pOAD* into grades according to Fontaine, a French surgeon, has established itself *(Table 1).* It is based on the fact that pOAD* generally manifests by pain while walking or at rest. The patient‘s mobility is thus reduced and the distance that can be walked without experiencing pain is shortened. It should be noted, however, that pOAD* often occurs in combination with sensory neuropathy in diabetic patients, so that pain while walking is not perceived and an existing pOAD* is disguised. A yearly examination of the vascular status is therefore required.

One specific feature of arteriosclerosis which occurs in 5 – 10 % of diabetic patients is the tubular calcification of the vascular wall in the lower leg region, known as medial sclerosis or Mönckeberg‘s sclerosis*. A rigid channel is formed which does not completely restrict arterial blood flow but which nevertheless seriously affects the indirect measurement of arterial blood flow in the ankle region. This is evident from the very high malleolus pressure values recorded in Doppler pressure measurements.

There is a high risk of critical Ischaemia* if the arterial blood supply to the foot is reduced to such an extent that the foot pulses (arteria tibialis posterior, arteria dorsalis pedis) are no longer palpable and values of less than 50 mmHg are recorded on Doppler measurement of arterial pressure in the foot. Unproblematic wound healing is unlikely in this case unless action is taken to improve the blood flow. The wound healing processes are significantly disrupted and patients are especially prone to wound infection and – in the worst case – amputation[5].

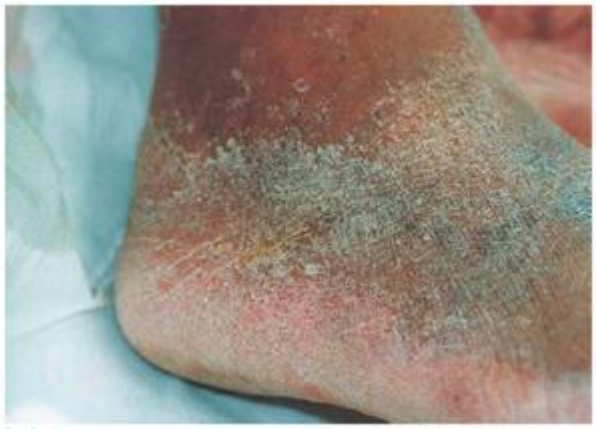


Fig. 1: Dry skin

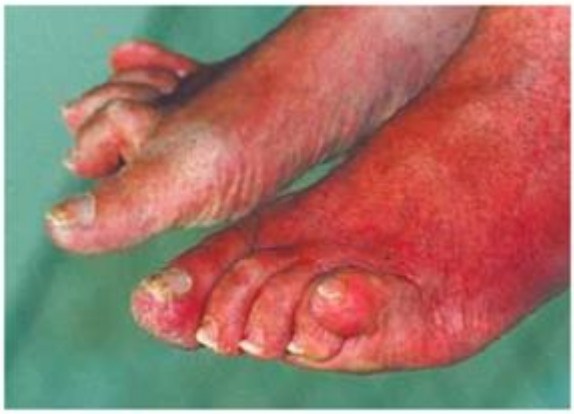


Fig. 2: Hammer toe

Fig. 3: Gangrene*/toe necrosis with pOAD*

PNP* with concomitant pOAD*

This form of diabetic foot – polyneuropathy* in conjunction with circulatory disturbances in the lower extremities – makes early diagnosis difficult because of the absence of the cardinal symptoms of pain on exercise and resting. The Fontaine classification *(Table 1)* does not apply. For this reason, patients presenting with PNP* and pOAD* often do not attend for consultation until they have reached Stage IV with necroses.

The diagnostic approaches listed under pOAD* and PNP* are implemented. There is a high risk of amputation due to the adverse effect on the physiological wound healing processes and the related risk of infection. Treatment should mainly be based on the circulatory situation which presents the most restrictive risk.

Fontaine classification of impaired blood circulation with pOAD	
Stage I	Asymptomatic with objectively diagnosed arterial occlusive disease
Stage II	Pain after exercise, claudicatio intermittens* II a: walking distance > 200 m II b: walking distance = 200 m
Stage III	Rest pain (persistent for a minimum of 2 weeks) in the affected extremity when placed in a horizontal position due to depleted blood flow to the muscles; often transient relief on lowering the leg
Stage IV	Acral ischaemic necrosis

Table 1 *Fontaine classification[6] of impaired blood circulation with pOAD**

Diagnosis

The clinical findings of diabetic foot vary depending on the aetiology. The clinical picture is mixed if polyneuropathy* and peripheral occlusive arterial disease* are both present.

Clinical features and findings with PNP*

Clinical features and findings with PNP*	
Medical History	• no claudicatio intermittens* • poor diabetes management (hyperglycaemia*, HbA1c >7*) • alcohol • duration of diabetes
Foot examination	• reddish, warm, dry, predisposed to oedema • formation of calluses and corns in areas exposed to pressure • restricted joint mobility • osseous and muscular foot deformities (hallux valgus*, digitus malleus*, Charcot's foot*
Pain	• sensitive clinical signs of irritation such as stabbing, pins and needles, formication and/or • absence of pain on sustaining injuries and wounds • bands of discomfort, manifesting mainly at night
Achilles tendon reflex Knee-jerk reflex	• diminished / eradicated
Foot pulses	• often palpable
Localization of skin defects	• at pressure-prone sites • malum perforans (painless ulcer on the sole of the foot with keratinized wound edge)
Basic diagnosis (always examine both feet)	• sensitivity to vibration (e.g. 128-Hz graduated Rydel-Seiffer tuning fork) • perception of touch (e.g. 10 g Semmes-Weinstein-Monofilament) • perception of temperature (Tip-Therm) • perception of pain (tooth pick or Neurotip) • proprioceptive reflexes (Achilles tendon and knee-jerk reflexes) The results of these examinations can be quantified by means of the neuropathy deficit score* [8].

Table 2 [8]: *Polyneuropathy**

Clinical features and findings with pOAD*

Clinical features and findings with pOAD*	
Medical History	• insulin resistance, nicotine, hyperlipoproteinaemia*, hypertension, coronary heart disease (CHD), claudicatio intermittens*
Foot examination	• skin: bluish pallor, like parchment, cool, dry; shape of the foot: narrow, atrophic*
Pain (no discomfort in conjunction with sensory neuropathy)	• on load-bearing or at rest (claudicatio intermittens*, see table 1) • chill • severe pain when moving necrotic toes • pain-sensitive wound edges (and frequently wound surface)
Achilles tendon reflex Knee-jerk reflex	• normal
Foot pulses	• weak or not palpable
Localization of skin defects	• tissue damage (necroses*, gangrene*) irradiating mainly from the toes (acral localization)
Orientational angiologic examination	• Palpation of foot pulses (a. tibialis posterior and a. dorsalis pedis) • Doppler ultrasound – based measurement of brachial and ankle systolic pressures and calculation of the ankle brachial pressure index (ABPI) • transcutaneous O2 partial pressure

Table 3 [8]: *Peripheral occlusive arterial disease**

Doppler pressure measurement is a straightforward and reliable method for detecting pOAD*. With the patient lying down, the systolic blood pressure quotient in the ankle and arm provides an index that allows evaluation of pOAD* (so called ankle-brachial-index, ABI). Normally this index is equal to or slightly higher than 1. Values < 0.9 are indicative of pOAD*. If the value is > 1.3 it is indicative of Mönckeberg‘s sclerosis*. This makes the test unsuitable for these patients. An X-ray image of the leg arteries is obtained with the assistance of angiography (various established techniques). This is essential in all cases of critical leg ischaemia* especially prior to amputation and surgical techniques to improve the blood supply. An X-ray is taken to facilitate the evaluation of changes in the foot bones. If necessary, further examinations such as a magnetic resonance tomography (MRT) (osteolysis*, osteomyelitis*, DNOAP, facture, false position) must be carried out.

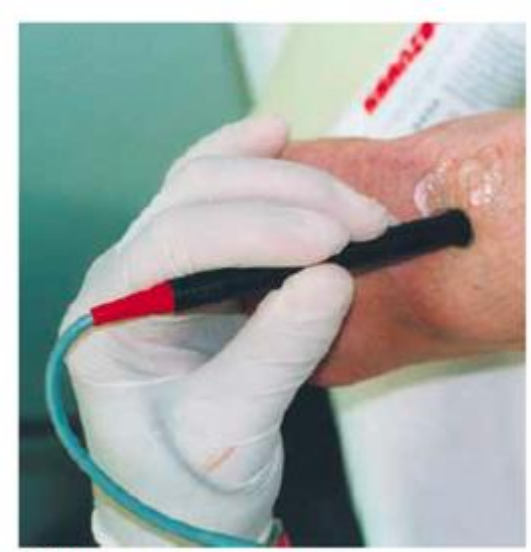


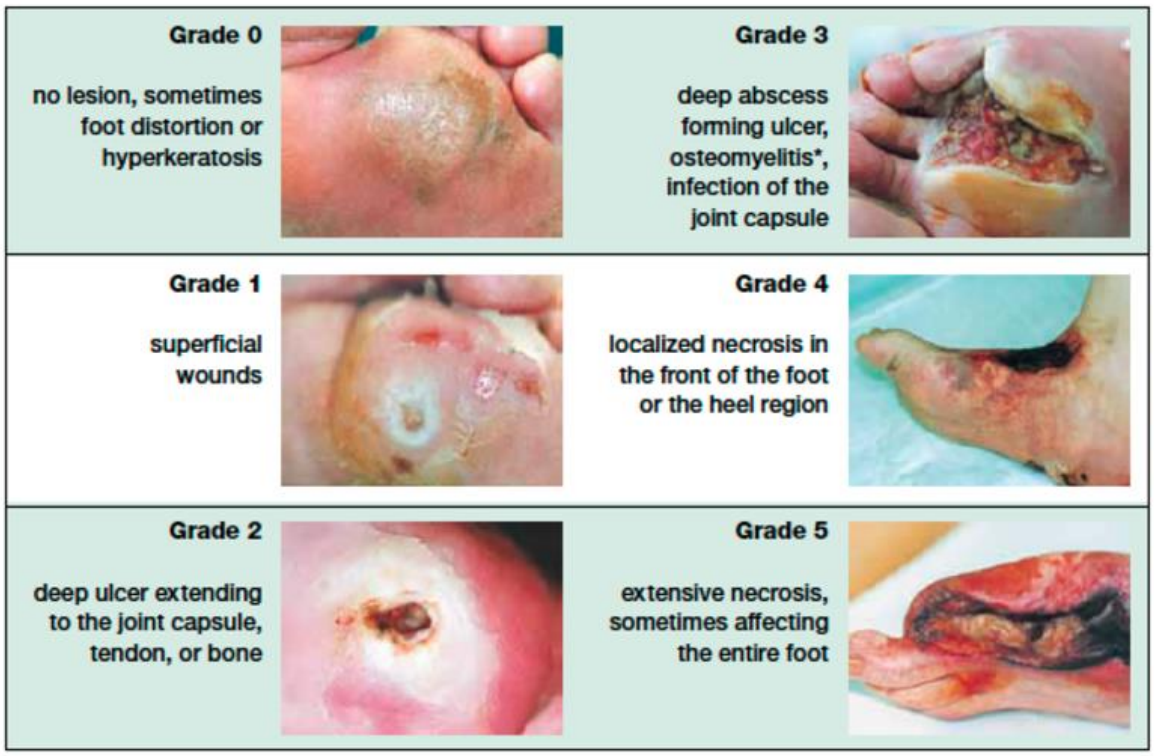
Fig. 4: Doppler pressure measurement

Classification of diabetic foot lesions

There is yet no unlimited internationally binding standard classification for the various foot lesions associated with DFU. The Wagner classification is the most widely used and – known as Wagner-Armstrong classification – is also binding in Germany. This system classifies foot lesions depending on their depth into six categories of severity (0-5).

The „University of Texas Wound Classification System“, also known as „Armstrong Classification“ which was presented in 1996 accounts for the aspects of „infection“ and „ischaemia“ besides the size of the lesion[9]. Meanwhile, a combination of these two systems, the so-called Wagner-Armstrong classification *(Table 4)* is becoming increasingly common. In this instance infection and ischaemia* – key factors in treatment and healing – are allocated varying to the different grades of severity and may result in therapeutic consequences.

Wagner classification



Wagner-Armstrong Classification

Wagner-Armstrong Classification							
		Wagner-Grade					
		0	1	2	3	4	5
Armstrong Grade	A	Pre- or post ulcerous foot	Superficial wound	Wound involving tendons or capsule	Wound involving bones and joints	Necrosis on part of the foot	Necrosis on the entire foot
	B	With infection	With infection	With infection	With infection	With infection	With infection
	C	With ischaemia*	With ischaemia*	With ischaemia*	With ischaemia*	With ischaemia*	With ischaemia*
	D	With infection and ischaemia*	With infection and ischaemia*	With infection and ischaemia*	With infection and ischaemia*	With infection and ischaemia*	With infection and ischaemia*

Table 4: *Wagner-Armstrong Classification. Ways of describing diabetic foot lesions by means of a combination of the Wagner-Armstrong classification*[1].

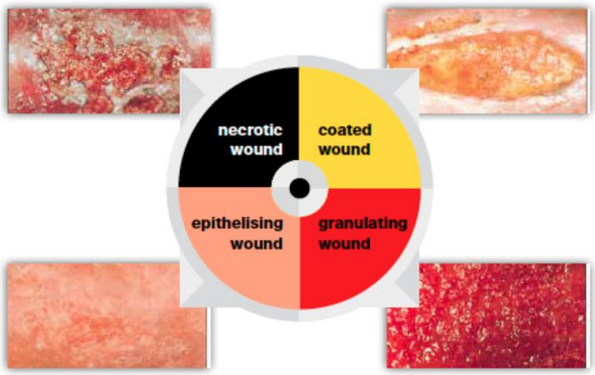
Wound condition

Infection with existing DFU is a serious complication that must be detected and treated as early as possible. Poorly managed diabetes is associated with reduced endogenous defence mechanism against infection. The otherwise typical signs of inflammation (redness, overheating, swelling, pain and impaired functions) may not

appear despite massive microbial invasion. The microbial spectrum is determined in the laboratory from a wound swab or tissue sample. Bony structures detected at depth in the wound are indicative of an increased risk of osteomyelitis*. Particular caution is warranted in the presence of necroses*, gangrene* of the toes, foot- and nail fungus with concomitant skin lesion, dry skin fissures (rhagades*), bleeding beneath calluses, and in the event of crusty deposits on superficial, non-irritant foot wounds. These foot injuries as well as wounds caused within the scope of foot care and existing skin blisters may be the starting point for wound infection extending along the main structures of the foot (tendons, bones) rapidly spreading to the entire foot and warranting amputation of the extremity in the worst case scenario. In order to facilitate a phase-oriented use of wound dressings, wounds are assessed according to their appearance and are colour-coded:

The extent of wound exudation can only be estimated. A wound can be subjectively allocated to one of the following degrees of exudation:

- heavy
- moderate or
- light exudation



Treatment

Control of blood sugar levels to near-normal values is imperative for the successful healing of diabetic foot. According to the literature, foot ulcer therapy following diagnosis of diabetic foot is fundamentally always based on the same procedure[1,7]. In addition to consistent off-loading, wound infection must be treated, any circulatory defects must be improved and the wound healing process promoted by local measures such as wound debridement and a phase-oriented wound dressing. Because of the complexity of the clinical picture it is strongly recommended to treat patients in a specialized foot care centre which offers interdisciplinary (internist, vascular and general surgeons, orthopaedist, neurologist, dermatologist and others) and interprofessional care (physician, nurse, orthopaedic technician, podiatrist).

Pressure relief

Ulceration on the sole or edge of the foot can heal only if pressure is consistently relieved and the wound is given time to heal. Hospital admission and complete relief from any pressure are essential in the case of deep, infected wounds where the loss of the extremity is imminent.

Ways of local off-loading are numerous. They range from bed rest, crutches and wheelchairs, footwear to provide partial relief, and special insoles to one- and two-shell relieving orthoses which are manufactured by specialists using various materials.

Total Contact Cast with Cellacast® TCC*

One method which until recently attracted little attention in Germany is the total contact cast. Total contact casts in the form that created full contact with the sole of the foot were originally used in India in the thirties in the treatment of patients suffering from leprosy with neuropathic foot ulcers[10]. This method was introduced to the USA in the treatment of neuropathic ulcers and has established itself in the US as the gold standard for relieving pressure in non-infected neuropathic ulcers[11]. With this method of offloading, a rigid cast made of plaster of Paris, synthetic or glass fibre casting material is applied to the lower leg and foot. By means of a special moulding technique at the sole of the foot, the patient‘s weight is removed from high pressure areas and redistributed to other regions of the foot. Additionally, the firm lower leg cast acts as a bootleg which takes off parts of the load from the lower leg and reduces undesired shearing forces, i.e. the foot slipping back and forth inside the cast, by holding the lower leg in place.

To this day, this classic total contact cast has seen numerous modifications. For example, the plaster material has been replaced by fiberglass material (synthetic cast) to reduce weight. Using fiberglass also allows an interesting alternative of TCC* construction: the TCC* in two-shell technique *(Fig. 12).* The circular cast is cut,

forming two shells. These shells are then replaced and affixed. With this method, dressing changes are possible at any time and usually a single cast can be used until the ulcer is fully healed.

Fig. 12: Total contact cast made from the Cellacast TCC* treatment system

A TCC* must only be applied by experienced specialists who have attended expert training. Knowledge of the pathopysiology of diabetic foot and experience in treating diabetic foot ulcers are imperative.

A TCC* should not be applied to patients suffering from clinically relevant ischaemia*. An ankle-brachial-index of < 0.8 or the absence of palpable foot pulses are indicative guidelines of ischaemia*. Acute infection with clinical signs of infection is another contraindication against a TCC*.

Good patient compliance, i.e. the patient‘s understanding of the disease and his/her corresponding cooperation in the treatment are crucial. Experience shows that it is best not to allow patients to remove the TCC* themselves, as even a nighttime walk to the bathroom in bare feet may ruin weeks of attempt at wound healing. The TCC* should be removed only in a specialized foot care centre or by a wound management expert (e.g. outpatient nursing service, home carer) who has been appropriately trained in the application and removal of the TCC*.

All materials needed for the construction of a total contact cast are included in the Cellacast TCC* treatment system. Meanwhile, scientific data on higher degrees of evidence on the successful application of TCC* on patients with neuropathic foot ulcers are also available in Germany[13]. The efficiency and suitability of the TCC on patients with diabetic foot was tested in a multi centric study in six foot care centres certified by the German Diabetes Association (DDG). In total, 50 patients with neuropathic foot ulcers (max. Wagner Grade 2, foot pulse at least palpable, or ABPI > 0.8, no infection) were treated. 46 ulcers had healed within an average of 37 days (SD: +/- 27 days; min. 7 days, max. 154 days).

The fast healing rates with TCC were highly convincing since some of the ulcers had previously persisted over a period of 4 years.

Eradication of infection

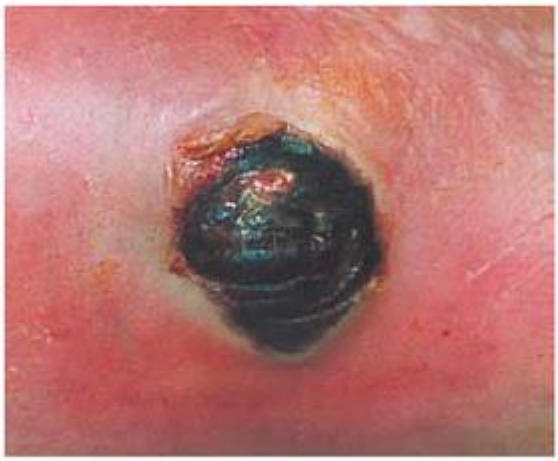


Fig. 13: Infected wound

Soft tissue infections in the foot will disturb the healing process for diabetic patients and place the extremity at risk. This is all the more relevant when a combined ischaemic-/neuropathic lesion is involved. The most trivial skin defect in the foot may be a trigger factor. It is therefore always necessary, even with nondescript skin injuries, to first treat the wound antiseptically. It should then be bandaged and medical advice must be sought immediately. A soft tissue infection at the front of the foot may lead to compression of the blood vessels and the formation of septic thrombi due to tissue swelling. This will disrupt the blood supply from the individual toes, triggering necroses* even in the absence of pOAD*.

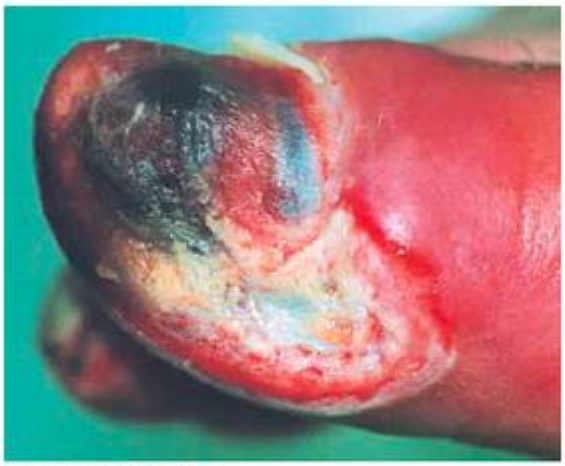


Fig. 14: Wound with pOAD*

The situation takes a dramatic turn if microorganisms penetrate and proliferate in the limit zone between necrotic and viable tissues. Within a short space of time a wound infection can spread along the principal anatomical structures, thus extending to the

entire foot. This can be fatal if, due to a lack of pain perception, the patient has a delayed reaction to changes in the foot and only then seeks medical advice.

A wound infection is frequently a combined infection with anaerobic* and aerobic* pathogens. It should be noted that the spectrum of pathogens affecting deep tissue layers often differs from that on the wound surface. Systemic antibiotic therapy, if possible based on an antibiogram*, should generate a sufficiently high level of efficacy in capillary free tissue such as cartilage. Surgical debridement is essential in cases of existing wound infection in order to remove necrotic and devitalized tissue. Retentions and abscesses must be opened.

The insertion of wound drains will ensure that any microbe contaminated exudate is discharged from the depths of the wound. Infected bone sections are surgically removed. Control of the wound infection and sufficient blood flow for wound healing are essential conditions for amputation with the formation of a load bearing amputation stump. The sequence of the IRAS principle should be followed: infection control - revascularization - amputation - shoe provision/patient training. The consequent immobilization of the affected extremity and strict monitoring of the treatment course can only be guaranteed under hospital conditions. At the same time, control of blood sugar levels to near-normal values* should be attempted in order to prevent the endogenous defense situation from deteriorating as a result of blood sugar imbalance[7].

With critically colonized and infected wounds, the focus is on the reduction of the microbial load as well as exudate management. Antimicrobially active silver and polyhexamethylene biguanide (PHMB) fight microorganisms in the wound area. Because of its very high tolerability PHMB is particularly suitable for long-term use. In the treatment of wounds with a particular risk of infection and infected deep and superficial wounds, the choice of a therapeutically appropriate wound dressing depends on the degree of wound exudation.

Besides reducing the pathogen load it is important to support the wound healing process. Wound dressings with an occlusive mode of action have a defined water vapour permeability and are impermeable to liquids and microorganisms. Such dressings pose the risk of promoting wound infection in high-risk wounds. However, care must be taken that the wound will not dry out either and possibly form crusty deposits. Antimicrobially active HydroBalance wound dressings (Suprasorb X + PHMB) are suitable for keeping the wound surface moist in wounds with light to moderate exudation, and for the reduction of the microbial load, for example in combination with special dressings (e.g. Solvaline N). With heavily exuding wounds, highly absorbent calcium alginate dressings with antimicrobially active silver (e.g. Suprasorb A + Ag) guarantee the absorption of exudate and the reduction of the microbial load, for example in combination with absorbent dressings (e.g. Vliwazell). With malodorous wounds, dressings containing activated charcoal and

antimicrobially active silver (Vliwaktiv Ag) will specifically fight odours as well as bacteria.

Treatment of vascular disease

In cases where pOAD* has been diagnosed, the attending physician will decide whether vascular treatment is appropriate and necessary. These are usually bypasses in case of extended vascular occlusion. In patients with short occlusions, the vascular lumen may be successfully distended using, for example, a balloon catheter[5]. In the event of existing wound infection, effective eradication of the infection is a pre-requisite for the blood supply to be surgically corrected. If an amputaion is unavoidable, this is carried out once the disrupted blood flow has been corrected in order to prevent potential post-amputation healing problems. Medical treatment to improve the outcome in patients with critical ischaemia* is being discussed[1].

Local treatment by means of moist wound management

Foot ulcers must be cleansed before a dressing is applied. Necroses* and deposits should be surgically removed where possible. Physical cleansing is carried out by rinsing with physiological solutions or special wound rinsing solutions. Wound antiseptics* help to reduce the number of microorganisms on the wound. Pronounced callus formation in the marginal region of the wound is regularly removed on changing the dressing.

Modern wound dressings should ensure a physiologically moist wound climate around the clock, with a temperature kept constant as far as possible. Gas exchange, protection against mechanical influences and the creation of a barrier against penetrating or released microorganisms are important requirements on modern wound dressings. Depending on the degree of exudation, the focus will be either on the absorption or the liquid release capacity. With expert application there will be no adhesion to the wound bed. Newly formed tissue is thus always protected against damage.

On the last pages of this brochure, you will find a detailed overview of the various stages (healing phase, wound depth, wound infection, degree of exudation) of wound dressings and their possible combinations.

Cleansing	Cleansing antimicrobial	Intelligent	Intelligent antimicrobial	Active	Easy	Versatile	Transparent	Moistening
Suprasorb® A Calcium alginate dressing	**Suprasorb® A +Ag** antimicrobial Calcium alginate dressing	**Suprasorb® X** HydroBalance wound dressing	**Suprasorb® X +PHMB** antimicrobial HydroBalance wound dressing	**Suprasorb® C** Collagen wound dressing	**Suprasorb® P** PU foam dressing	**Suprasorb® H** Hydrocolloid dressing	**Suprasorb® F** Film wound dressing	**Suprasorb® G** Gel dressings
for deep or superficial wounds with heavy exudation, in the exudation and granulation phase	for infected deep or superficial wounds with heavy exudation, in the exudation and granulation phase	for deep or superficial wounds with light to moderate exudation, in all three phases of healing	for infected deep or superficial wounds with light to moderate exudation, in all three phases of healing	for deep or superficial wounds with light to moderate exudation, in all three phases of healing	for superficial wounds with moderate exudation, in all three phases of healing	for superficial wounds with light to moderate exudation, in all three phases of healing	for superficial wounds with light exudation, in the epithelisation phase	for deep or superficial, necrotic and dry wounds
								
Controls exudation, conforms ideally to the contours of the wound bed and protects the wound against drying out by forming a gel.	The combination of the properties of Suprasorb A with silver ensures safe management of critically colonized and infected wounds by a wide anti-microbial spectrum and fast action against MRSA and VRE.	Identifies whether moisture has to be released to the wound or exudate has to be absorbed or both are necessary at the same time. Reduces wound pain during treatment and conforms excellently to the wound bed.	The combination of the properties of Suprasorb X with PHMB ensures safe management of critically colonized and infected wounds by a wide anti-microbial spectrum and fast action against MRSA and VRE. PHMB is welltolerated, especially gentle to cells and thus also suitable for long-term use.	Is very versatile – from burns to complicated defect wounds. The wound dressing binds exudate and inflammation-inducing proteases and radicals very quickly and efficiently. It promotes collagen synthesis and the proliferation and migration of epidermal cells.	The carrier layer made of polyurethane ensures reliable gas exchange yet repels microorganisms and water and ensures an ideal moist wound environment.	Is self-adhesive and ensures a moist wound environment and good exudate absorption at the same time.	Supports a moist wound environment and allows visual wound monitoring.	Ensures a persistently moist wound environment and gently dissolves necroses*.
					"Tip: for malodorous wounds with all degrees of exudation: **Vliwaktiv® Ag**"			

Concomitant measures to support basic treatment

A near-normal blood sugar level* is imperative for a diabetic foot ulcer to heal properly. The treatment of concomitant diseases and the avoidance and/or elimination of nutritional deficits play an important role. Another point of high significance is patient training. Without patient compliance and an understanding of the required therapeutic measures there are always considerable risks that the health condition may deteriorate. Nail and foot fungus must consistently be treated.

Dry skin requires continuous care. Creams and foams containing, for example, urea, are suitable for this purpose.

Surgical approaches

The significance of surgical intervention in the diabetic foot treatment has previously been discussed within the scope of the chapters of eradication of infection, local therapy, and the treatment of vascular disease. In the presence of irreversible extensive tissue damage or if revascularization is not feasible, the formation of load bearing amputation stumps falls into the domain of surgery. This also covers preventive surgery. The eradication of foot deformitis such as hallux valgus* and hammer toe (digitus malleus*) may reduce the risk of pressure ulcer formation at these sites and thereby possibly help to prevent amputation.

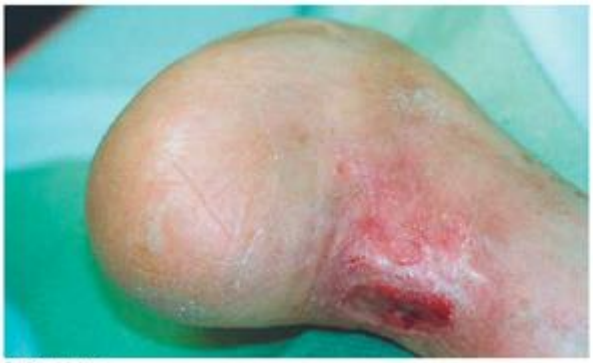


Fig. 15: Amputation stump