

ABC of wound healing: Wound assessment

Causes of ulceration

- Vascular (venous, arterial, lymphatic, vasculitis)
- Neuropathic (for example, diabetes, spina bifida, leprosy)
- Metabolic (for example, diabetes, gout)
- Connective tissue disease (for example, rheumatoid arthritis, scleroderma, systemic lupus erythematosus)
- Pyoderma gangrenosum (often reflection of systemic disorder)
- Haematological disease (red blood cell disorders (for example, sickle cell disease); white blood cell disorders (for example, leukaemia); platelet disorders (for example, thrombocytosis)
- Dysproteinaemias (for example, cryoglobulinaemia, amyloidosis)
- Immunodeficiency (for example, HIV, immunosuppressive therapy)
- Neoplastic (for example, basal cell carcinoma, squamous cell carcinoma, metastatic disease)
- Infectious (bacterial, fungal, viral)
- Panniculitis (for example, necrobiosis lipoidica)
- Traumatic (for example, pressure ulcer, radiation damage)
- Iatrogenic (for example, drugs)
- Factitious (self harm, "dermatitis artefacta")
- Others (for example, sarcoidosis)

Local and systemic factors that impede wound healing

Local factors

- Inadequate blood supply
- Increased skin tension
- Poor surgical apposition
- Wound dehiscence
- Poor venous drainage
- Presence of foreign body and foreign body reactions
- Continued presence of micro-organisms
- Infection
- Excess local mobility, such as over a joint

Systemic factors

- Advancing age and general immobility
- Obesity
- Smoking
- Malnutrition
- Deficiency of vitamins and trace elements
- Systemic malignancy and terminal illness
- Shock of any cause
- Chemotherapy and radiotherapy
- Immunosuppressant drugs, corticosteroids, anticoagulants
- Inherited neutrophil disorders, such as leucocyte adhesion deficiency
- Impaired macrophage activity (malacoplakia)

Some complications of chronic wounds

- Sinus formation
- Fistula
- Unrecognised malignancy
- Malignant transformation in the ulcer bed (Marjolin's ulcer)
- Osteomyelitis
- Contractures and deformity in surrounding joints
- Systemic amyloidosis
- Heterotopic calcification
- Colonisation by multiple drug resistant pathogens, leading to antibiotic resistance
- Anaemia
- Septicaemia

Most wounds, of whatever aetiology, heal without difficulty. Some wounds, however, are subject to factors that impede healing, although these do not prevent healing if the wounds are managed appropriately. A minority of wounds will become chronic and non-healing. In these cases the ultimate goal is to control the symptoms and prevent complications, rather than healing the wound.

It is important that the normal processes of developing a diagnostic hypothesis are followed before trying to treat the wound.

A detailed clinical history should include information on the duration of ulcer, previous ulceration, history of trauma, family history of ulceration, ulcer characteristics (site, pain, odour, and exudate or discharge), limb temperature, underlying medical conditions (for example, diabetes mellitus, peripheral vascular disease, ischaemic heart disease, cerebrovascular accident, neuropathy, connective tissue diseases (such as rheumatoid arthritis), varicose veins, deep venous thrombosis), previous venous or arterial surgery, smoking, medications, and allergies to drugs and dressings. Appropriate investigations should be carried out.

Assessing wounds

Size of wound

The size of the wound should be assessed at first presentation and regularly thereafter. The outline of the wound margin should be traced onto transparent acetate sheets and the surface area estimated: in wounds that are approximately circular, multiply the longest diameter in one plane by the longest diameter in the plane at right angles; in irregularly shaped wounds, add up the number of squares contained within the margin of the outline of the wound from an acetate grid tracing.

These methods are the simplest, but it should be recognised that they are not precise. However, they do provide a means by which progress over time to wound closure can be identified. Patient positioning, body curvature, or tapering of the limbs will affect the accuracy of these techniques.

Edge of wound

Although not diagnostic, examination of the edge of the wound may help to identify its aetiology in the context of the history of the wound. For example, venous leg ulcers generally have gently sloping edges, arterial ulcers often appear well demarcated and "punched out," and rolled or everted edges should raise the suspicion of malignancy. A biopsy should be taken of any suspicious wound.

Site of wound

The site of the wound may aid diagnosis; diabetic foot ulcers often arise in areas of abnormal pressure distribution arising from disordered foot architecture. Venous ulceration occurs mostly in the gaiter area



of the leg (see next article in this series). Non-healing ulcers, sometimes in unusual sites, should prompt consideration of malignancy.

Wound bed

Healthy granulation tissue is pink in colour and is an indicator of healing. Unhealthy granulation is dark red in colour, often bleeds on contact, and may indicate the presence of wound infection. Such wounds should be cultured and treated in the light of microbiological results. Excess granulation or overgranulation may also be associated with infection or non-healing wounds. These often respond to simple cautery with silver nitrate or with topically applied steroid preparations. Chronic wounds may be covered by white or yellow shiny fibrinous tissue (see next article in this series). This tissue is avascular, and healing will proceed only when it is removed. This can be done with a scalpel at the bedside.

The type of tissue at the base of the wound will provide useful information relating to expectation of total healing time and the risk of complications—for example, bone at the base may suggest osteomyelitis and delayed or non-healing.

Necrotic tissue, slough, and eschar

The wound bed may be covered with necrotic tissue (non-viable tissue due to reduced blood supply), slough (dead tissue, usually cream or yellow in colour), or eschar (dry, black hard necrotic tissue). Such tissue impedes healing. Necrotic tissue and slough may be quantified as excessive (+++), moderate (++) , minimal (+) or absent (-).

Since necrotic tissue can also harbour pathogenic organisms, removal of such tissue helps to prevent wound infection. Necrotic tissue and slough should be debrided with a scalpel so that the wound bed can be accurately assessed and facilitate healing. Eschar may be adherent to the wound bed, making debridement with a scalpel difficult. Further debridement, as part of wound management, may be required using other techniques.

Accurate methods for measuring wound depth are not practical or available in routine clinical practice. However, approximate measurements of greatest depth should be taken to assess wound progress.

Undermining of the edge of the wound must be identified by digital examination or use of a probe. The depth and extent of sinuses and fistulas should be identified. Undermining areas and sinuses should be packed with an appropriate dressing to facilitate healing.

Undermining wounds and sinuses with narrow necks that are difficult to dress may be amenable to be laid open at the bedside to facilitate drainage and dressing. Wounds associated with multiple sinuses or fistulas should be referred for specialist surgical intervention.

Surrounding skin

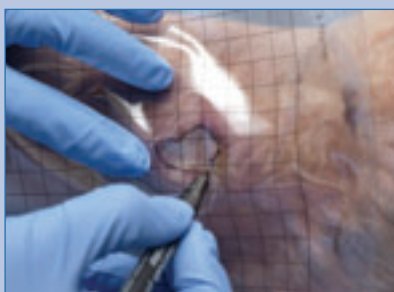
Cellulitis associated with wounds should be treated with systemic antibiotics. Eczematous changes may need treatment with potent topical steroid preparations. Maceration of the surrounding skin is often a sign of inability of the dressing to control the wound exudate, which may respond to more frequent dressing changes or change in dressing type. Callus surrounding and sometime covering neuropathic foot ulcers (for example, in diabetic patients) must be debrided to (a) visualise the wound, (b) eliminate potential source of infection, and (c) remove areas close to the wound subject to abnormal pressure that would otherwise cause enlargement of the wound. This can be done at the bedside.

Infection

All open wounds are colonised. Bacteriological culture is indicated only if clinical signs of infection are present or if infection control issues (such as methicillin resistant staphylococcus aureus (MRSA)) need to be considered. The classic signs of infection are heat, ➤

Laboratory investigations before treating a wound

Investigation	Rationale
Haemoglobin	Anaemia may delay healing
White cell count	Infection
Platelet count	Thrombocytopenia
Erythrocyte sedimentation rate; C reactive protein	Non-specific markers of infection and inflammation; useful in diagnosis and monitoring treatment of infectious or inflammatory ulceration
Urea and creatinine	High urea impairs wound healing. Renal function important when using antibiotics
Albumin	Protein loss delays healing
Glucose, haemoglobin A1C	Diabetes mellitus
Markers of autoimmune disease (such as rheumatoid factor, antinuclear antibodies, anticardiolipin antibodies, lupus anticoagulant)	Indicative of rheumatoid disease, SLE and other connective tissue disorders
Cryoglobulins, cryofibrinogens, prothrombin time, partial thromboplastin time	Haematological disease
Deficiency or defect of antithrombin III, protein C, protein S, factor V Leiden	Vascular thrombosis
Haemoglobinopathy screen	Sickle cell anaemia, thalassaemia
HIV status	Kaposi's sarcoma
Serum protein electrophoresis; Bence-Jones proteins	Myeloma
Urine analysis	Useful in connective tissue disease
Wound swab	Not routine; all ulcers colonised (not the same as infection); swab only when clinical signs of infection



Above: Tracing a wound for measurement and measuring a wound



Above: Overgranulation may be a sign of infection or non-healing

Wound edge characteristics

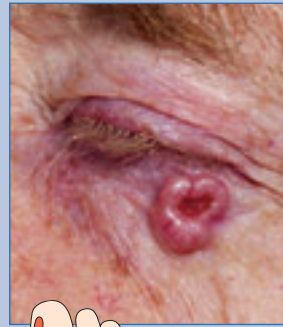
Edges	Type of ulcer
Sloping	Venous ulcer
Punched out	Arterial or vasculitic ulcer
Rolled	Basal cell carcinoma
Everted	Squamous cell carcinoma
Undermining	Tuberculosis, syphilis
Purple	Vasculitic (such as pyoderma gangrenosum)

Site of wound and type of ulcer

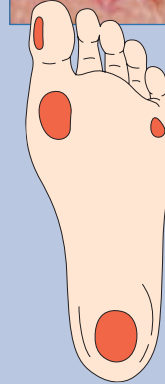
Site	Type of ulcer
Gaiter area of the leg	Venous ulcer
Sacrum, greater trochanter, heel	Pressure ulcer
Dorsum of the foot	Arterial or vasculitic ulcer
Shin	Necrobiosis lipoidica
Lateral malleolus	Venous, arterial, or pressure ulcer or hydroxyurea induced ulceration
Plantar and lateral aspect of foot and toes	Diabetic ulcer
Sun exposed areas	Basal cell carcinoma; squamous cell carcinoma

Types of debridement

- Sharp*—At the bedside (using scalpel or curette)
- Surgical*—In the operating theatre
- Autolytic*—Facilitation of the body's own mechanism of debridement with appropriate dressings
- Biological*—Larval (maggot) therapy
- Enzymatic*—Not widely used; pawpaw (papaya) or banana skin used in developing countries
- Mechanical*—Wet-to-dry dressings (not widely used in the UK)



Left: Basal cell carcinoma with rolled edges. Below: Lymphoma presenting as groin ulceration



Areas of abnormal pressure distribution in the diabetic foot.



Wound exudate

Wound exudate may be serous, serosanguinous, or sanguinous. The quantity of exudate is usually classified as heavy (+++ (dressing soaked)) medium (++ (dressing wet)), or minimal (+ (dressing dry)). Excessive exudate may be due to wound infection or gross oedema in the wound area and may complicate wound healing. The exudate should be controlled with the use of dressings appropriate for the level of exudate and any infection treated. Barrier films applied to the surrounding skin help to prevent further maceration (see the ninth article in the series). The oedematous leg should be raised when the patient is seated.

redness, swelling, and pain. Additional signs of wound infection include increased exudate, delayed healing, contact bleeding, odour, and abnormal granulation tissue. Treatment with antimicrobials should be guided by microbiological results and local resistance patterns.

Pain

Pain is a characteristic feature of many healing and non-healing wounds. Pain can be caused by both nociceptive and neuropathic stimuli. Intermittent pain is often related to dressing removal or recent application of new dressings and may necessitate the use of analgesia before the dressing is changed. Constant pain may arise as a result of the underlying condition, such as ischaemia, neuropathy, tissue oedema, chronic



Left: Healthy granulation tissue in a hidradenitis suppurativa excision wound. Below: Unhealthy granulation tissue in a venous leg ulcer



tissue damage (for example, lipodermatosclerosis), infection, or scarring (for example, atrophie blanche). The nature and type of pain should be identified and treated appropriately. Pain assessment tools can help to assess the nature and severity of pain. With recalcitrant pain or pain that is difficult to control, consider referral to a local pain team.


Non-healing wounds

Non-healing wounds have traditionally been defined as those that fail to progress through an orderly sequence of repair in a timely fashion. Such wounds are sometimes thought of as being caused by neglect, incompetence, misdiagnosis, or inappropriate treatment strategies. However, some wounds are resistant to all efforts of treatment aimed at healing, and alternative end points should be considered; measures aimed at improving the quality of life will be paramount in these instances.

Quality of life

Several studies have shown that patients with non-healing wounds have a decreased quality of life. Reasons for this include the frequency and regularity of dressing changes, which affect daily routine; a feeling of continued fatigue due to lack of sleep; restricted mobility; pain; odour; wound infection; and the physical and psychological effects of polypharmacy.

The loss of independence associated with functional decline can lead to changes, sometimes subtle, in overall health and wellbeing. These changes include altered eating habits, depression, social isolation, and a gradual reduction in activity levels.

Many patients with non-healing wounds complain of difficulties with emotions, finances, physical health, daily activities, friendships, and leisure pursuits. 

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Quality of life is not always related to healing of the wound. It may be clear from the outset that wounds in some patients will be unlikely to heal. In such patients control of symptoms and signs outlined above—particularly odour, exudate, and pain—may improve the individual's quality of life.

Additionally, optimal chronic wound management will lead to a reduction in the frequency of dressing changes, further enhancing quality of life. In a minority of instances, seemingly drastic measures—such as amputation in a person with chronic leg ulceration—may need to be considered when the quality of life is severely affected by the non-healing wound and its complications.

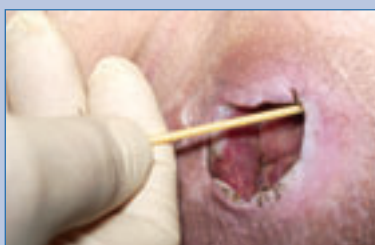
The drawing is adapted from one provided by Wendy Tyrrell, School of Health and Social Sciences, University of Wales Institute, Cardiff.



Top: Necrotic tissue (black areas) in a pressure ulcer.
Top right: Slough at the base of a pressure ulcer. Right: Eschar covering a heel pressure ulcer



Left: Digital examination of a wound. Left below: Examining a wound with a probe



Below left: Fistula in a diabetic foot ulcer
Below: Maceration of the skin surrounding a diabetic foot ulcer



Clinical features of non-healing wounds

- Absence of healthy granulation tissue
- Presence of necrotic and unhealthy tissue in the wound bed
- Excess exudate and slough
- Lack of adequate blood supply
- Failure of re-epithelialisation
- Cyclical or persistent pain
- Recurrent breakdown of wound bed
- Clinical or subclinical infection

Further reading

Lazarus GS, Cooper DM, Knighton DR, Margolis DJ, Pecoraro RE, Rodeheaver G, et al. Definitions and guidelines for assessment of wounds and evaluation of healing. *Arch Dermatol* 1994;130:489-93.
Izadi K, Ganchi P. Chronic wounds. *Clin Plast Surg* 2005;32:209-22.
Falanga V, Phillips TJ, Harding KG, Moy RL, Peerson LJ, eds. *Text atlas of wound management*. London: Martin Dunitz, 2000.