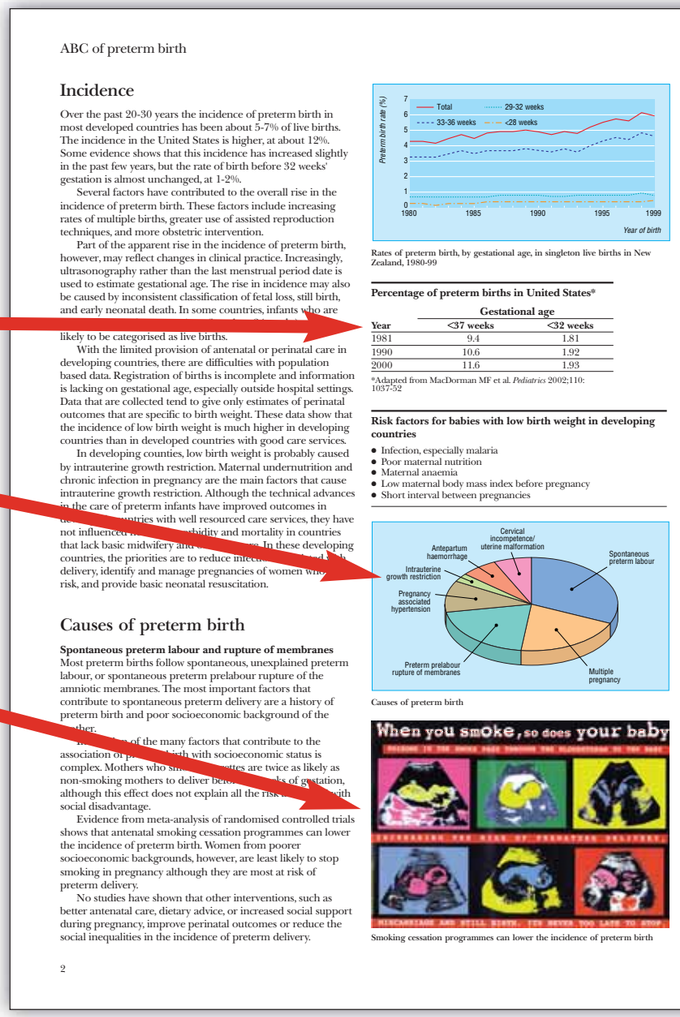
ABC SERIES

Features of the ABC Series

- The ABC titles are serialised and peer reviewed in the **BMJ** before being published in this great series of books
- The pages are always laid out in two columns with the highly illustrated ‘slide show’ of relevant visual aids alongside the text, pulling out key points from the text
- Each book is easy to read and contains a consistent style and the following key features which help to show the important aspects of the text



ABC of preterm birth

Incidence

Over the past 20-30 years the incidence of preterm birth in most developed countries has been about 5-7% of live births. The incidence in the United States is higher, at about 12%. Some evidence shows that this incidence has increased slightly in the past few years, but the rate of births before 32 weeks' gestation is almost unchanged, at 1-2%.

Several factors have contributed to the overall rise in the incidence of preterm birth. These factors include increasing rates of multiple births, greater use of assisted reproduction techniques, and more obstetric intervention.

Part of the apparent rise in the incidence of preterm birth, however, may reflect changes in clinical practice. Increasingly, ultrasonography rather than the last menstrual period date is used to estimate gestational age. The rise in incidence may also be caused by inconsistent classification of fetal loss, still birth, and early neonatal death. In some countries, infants who are likely to be categorised as live births.

With the limited provision of antenatal or perinatal care in developing countries, there are difficulties with population based data. Registration of births is incomplete and information is lacking on gestational age, especially outside hospital settings. Data that are collected tend to give only estimates of perinatal outcomes that are specific to birth weight. These data show that the incidence of low birth weight is much higher in developing countries than in developed countries with good care services.

In developing counties, low birth weight is probably caused by intrauterine growth restriction. Maternal undernutrition and chronic infection in pregnancy are the main factors that cause intrauterine growth restriction. Although the technical advances in the care of preterm infants have improved outcomes in [illegible] countries with well resourced care services, they have not influenced [illegible] bility and mortality in countries that lack basic midwifery and [illegible]. In these developing countries, the priorities are to reduce infec[illegible] delivery, identify and manage pregnancies of women who risk, and provide basic neonatal resuscitation.

Rates of preterm birth, by gestational age, in singleton live births in New Zealand, 1980-99

Percentage of preterm births in United States*

	Gestational age	
Year	<37 weeks	<32 weeks
1981	9.4	1.81
1990	10.6	1.92
2000	11.6	1.93

*Adapted from MacDorman MF et al. *Pediatrics* 2002;110: 1037-52

Risk factors for babies with low birth weight in developing countries

- Infection, especially malaria
- Poor maternal nutrition
- Maternal anaemia
- Low maternal body mass index before pregnancy
- Short interval between pregnancies

Causes of preterm birth

Smoking cessation programmes can lower the incidence of preterm birth

Causes of preterm birth

Spontaneous preterm labour and rupture of membranes
Most preterm births follow spontaneous, unexplained preterm labour, or spontaneous preterm prelabour rupture of the amniotic membranes. The most important factors that contribute to spontaneous preterm delivery are a history of preterm birth and poor socioeconomic background of the [illegible]ber.

[illegible] of the many factors that contribute to the association of p[illegible]th with socioeconomic status is complex. Mothers who sm[illegible]ttes are twice as likely as non-smoking mothers to deliver be[illegible]ks of gestation, although this effect does not explain all the risk [illegible]with social disadvantage.

Evidence from meta-analysis of randomised controlled trials shows that antenatal smoking cessation programmes can lower the incidence of preterm birth. Women from poorer socioeconomic backgrounds, however, are least likely to stop smoking in pregnancy although they are most at risk of preterm delivery.

No studies have shown that other interventions, such as better antenatal care, dietary advice, or increased social support during pregnancy, improve perinatal outcomes or reduce the social inequalities in the incidence of preterm delivery.

The rate of preterm birth varies between ethnic groups. In the United Kingdom, and even more markedly in the United States, the incidence of preterm birth in black women is higher than that in white women of similar age. The reason for this variation is unclear because differences remain after taking into account socioeconomic risk factors.

Preterm births by ethnic group in United States 2000*

- Black—17.3%
- Hispanic—11.2%
- Non-Hispanic white—10.4%

*Adapted from MacDorman MF et al. *Pediatrics* 2002;110:1037-52

Multiple pregnancy and assisted reproduction
Multifetal pregnancy increases the risk of preterm delivery. About one quarter of preterm births occur in multiple pregnancies. Half of all twins and most triplets are born preterm. Multiple pregnancy is more likely than singleton pregnancy to be associated with spontaneous preterm labour and with preterm obstetric interventions, such as induction of labour or delivery by caesarean section.

The incidence of multiple pregnancies in developed countries has increased over the past 20-30 years. This rise is mainly because of the increased use of assisted reproduction techniques, such as drugs that induce ovulation and in vitro fertilisation. For example, the birth rate of twins in the United States has increased by 55% since 1980. The rate of higher order multiple births increased fourfold between 1980 and 1998, although this rate has decreased slightly over the past five years. In some countries two embryos only are allowed to be placed in the uterus after in vitro fertilisation to limit the incidence of higher order pregnancy.

Singleton pregnancies that follow assisted reproduction are at a considerable increased risk of preterm delivery, probably because of factors such as cervical trauma, the higher incidence of uterine problems, and possibly because of the increased risk of infection.

Twin pregnancy increases the risk of preterm birth

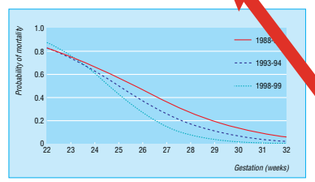
Maternal and fetal complications
About 15% to 25% of preterm infants are delivered because of maternal or fetal complications of pregnancy. The principal causes are hypertensive disorders of pregnancy and severe intrauterine growth restriction, which is often associated with hypertensive disorders. The decision to deliver these infants is informed by balancing the risks of preterm birth for the infant against the consequence of continued pregnancy for the mother and fetus. Over the past two decades improved antenatal and perinatal care has increased the rate of iatrogenic preterm delivery. During that time the incidence of still birth in the third trimester has fallen.

Outcomes after preterm birth

Broadly, outcomes improve with increasing gestational age, although for any given length of gestation survival varies with birth weight. Other factors, including ethnicity and gender also influence survival and the risk of neurological impairment.

The outcomes for preterm infants born at or after 32 weeks of gestation are similar to those for term infants. Most serious problems associated with preterm birth occur in the 1% to 2% of infants who are born before 32 completed weeks' gestation, and particularly the 0.4% of infants born before 28 weeks' gestation. Modern perinatal care and specific interventions, such as prophylactic antenatal steroids and exogenous surfactants, have contributed to some improved outcomes for very preterm infants. The overall prognosis remains poor, however, particularly for infants who are born before 26 weeks' gestation.

The outcome for preterm infants of multiple pregnancies can be better than that of singleton pregnancies of the same gestation. In term infants the situation is reversed. The

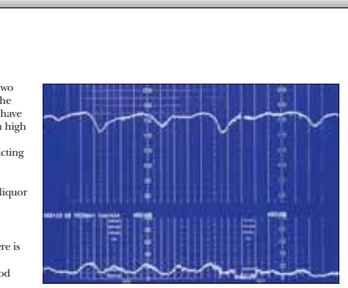


Mortality in UK neonatal intensive care cohorts of infants born before 32 weeks' gestation. Adapted from Parry G, et al. *Lancet* 2003;361:1789-91

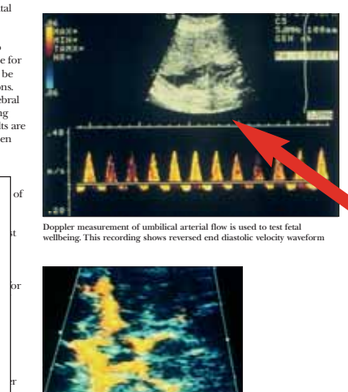
Outcomes for infants live born before 26 weeks' gestation in British Isles*

Gestation (weeks)	Survival to discharge (%)	Survival without handicap at 30 months (%)
22	1	0.7
23	11	5
24	26	12
25	44	23

*Adapted from Wood NS et al. *New Engl J Med* 2000;343:378-84



Monitoring the fetal heart rate can help determine the physiological wellbeing of the fetus. This cardiotocogram shows fetal tachycardia with reduced variability and decelerations



Doppler measurement of umbilical arterial flow is used to test fetal wellbeing. This recording shows reversed end diastolic velocity waveform

Doppler measurement of middle cerebral arterial flow. Abnormal waveforms can show cardiovascular adaptations to placental insufficiency

Induction of labour is most likely to be successful in a woman with a favourable cervix (as assessed by the Bishop score) who has had no caesarean sections and has a history of vaginal delivery

Please scroll down to see a sample chapter

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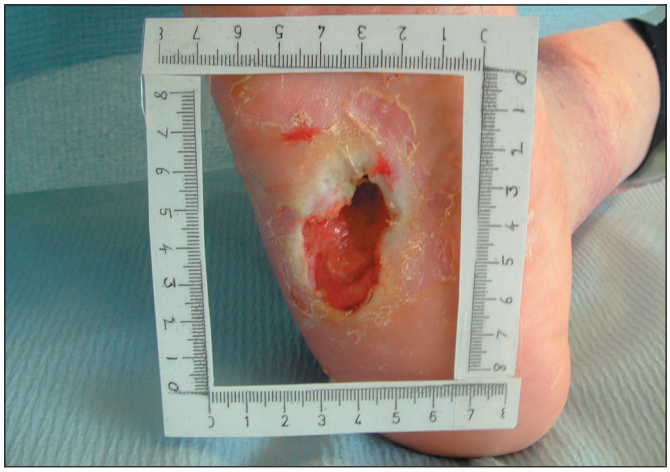
1 Wound assessment

Joseph E Grey, Stuart Enoch, Keith G Harding

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.



Wounds are not just skin deep, and accurate assessment is an essential part of treatment

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)

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.

Local and systemic factors that impede wound healing

Local factors

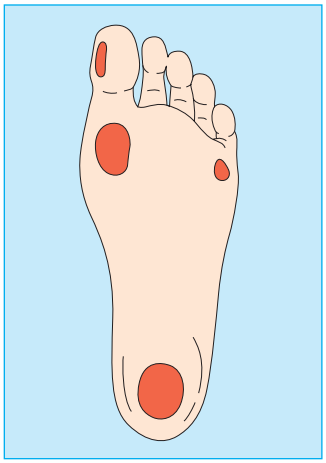
- Inadequate blood supply
- Increased skin tension
- Poor surgical apposition
- Wound dehiscence
- Poor venous drainage
- Presence of foreign body and foreign body reactions
- Continued presence of micro-organisms
- Infection
- Excess local mobility, such as over a joint

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

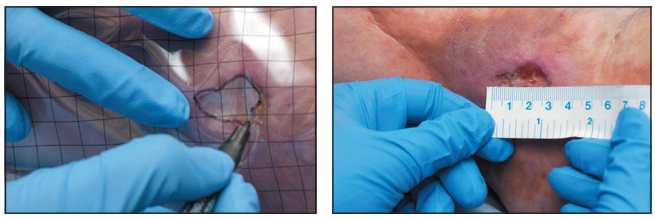


Areas of abnormal pressure distribution in the diabetic foot. Plantar ulcers are most commonly seen under the hallux, on the first and fifth metatarsal heads, and under the heel

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 on to 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.



Tracing a wound for measurement and measuring a wound

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.

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

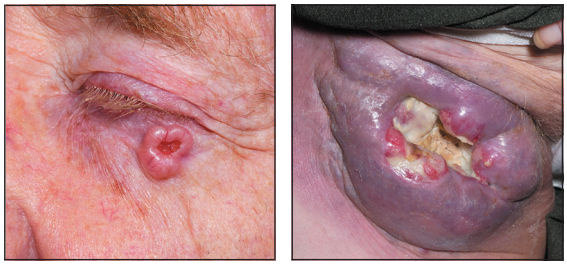
The site of the wound may aid diagnosis; diabetic foot ulcers often arise in areas of abnormal pressure distribution arising from disordered foot architecture. Venous ulceration occurs mostly in the gaiter area of the leg (see next article in this series). Non-healing ulcers, sometimes in unusual sites, 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

Laboratory investigations before treating a wound

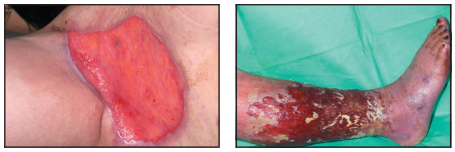
Investigation	Rationale
Haemoglobin	Anaemia may delay healing
White cell count	Infection
Platelet count	Thrombocytopenia
Erythrocyte sedimentation rate; C reactive protein	Non-specific markers of infection and inflammation; useful in diagnosis and monitoring treatment of infectious or inflammatory ulceration
Urea and creatinine	High urea impairs wound healing. Renal function important when using antibiotics
Albumin	Protein loss delays healing
Glucose, haemoglobin A_{1C}	Diabetes mellitus
Markers of autoimmune disease (such as rheumatoid factor, antinuclear antibodies, anticardiolipin antibodies, lupus anticoagulant)	Indicative of rheumatoid disease, systemic lupus erythematosus, 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



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

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



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

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.

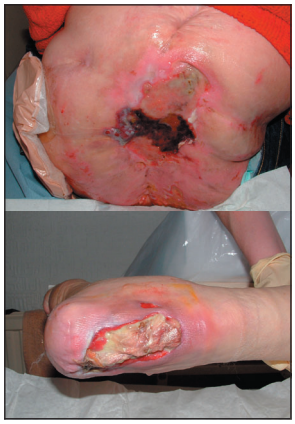
The type of tissue at the base of the wound will provide useful information relating to expectation of total healing time and the risk of complications—for example, bone at the base may suggest osteomyelitis and delayed or non-healing.

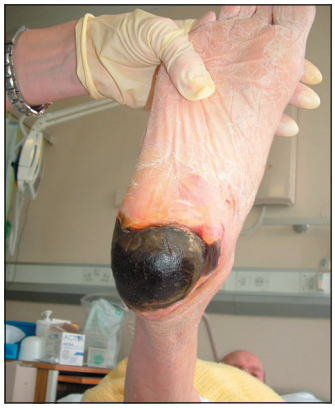
Bone at the base of a wound may suggest a protracted healing time and the possibility of underlying osteomyelitis

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.

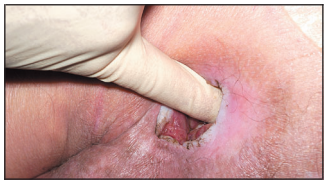


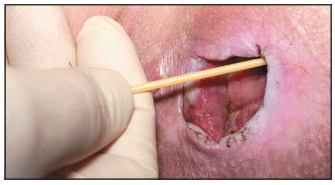


Top: Necrotic tissue (black areas) in a pressure ulcer. Bottom: Slough at the base of a pressure ulcer. Right: Eschar covering a heel pressure ulcer

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)

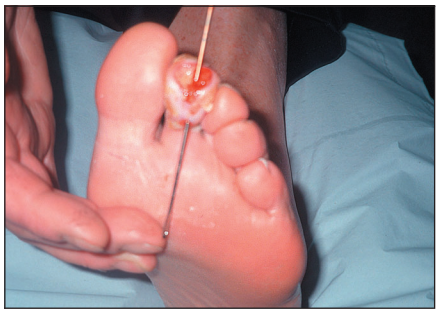




Left: Digital examination of a wound. Right: Examining a wound with a probe

Depth

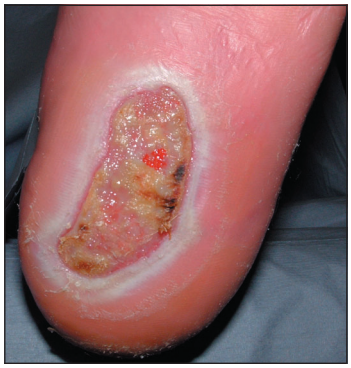
Accurate methods for measuring wound depth are not practical or available in routine clinical practice. However, approximate measurements of greatest depth should be taken to assess wound progress. Undermining of the edge of the wound must be identified by digital examination or use of a probe. The depth 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.



Fistula in a diabetic foot ulcer

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 sometimes 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.



Maceration of the skin surrounding a diabetic foot ulcer

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, 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 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.

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

The causes of malodorous wounds include infection and the presence of necrotic tissue. Infection should be treated with antibiotics. Odour associated with necrotic tissue may be reduced by removal of the necrotic tissue or use of agents impregnated with antiseptics or charcoal. Treatment with topical metronidazole and use of odour absorbing dressings may help to reduce odour from fungating malignant wounds. Larval therapy may also be helpful in the debridement of malodorous tissue

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.

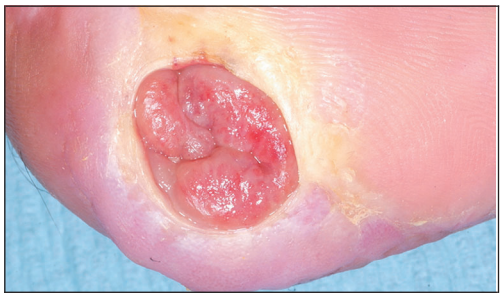
Clinical features of non-healing wounds

- 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
- Clinical or subclinical infection

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.

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.



Overgranulation may be a sign of infection or non-healing

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.
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- Falanga V, Phillips TJ, Harding KG, Moy RL, Peerson LJ, eds. *Text atlas of wound management*. London: Martin Dunitz, 2000.

Competing interests: KGH's unit receives income from many commercial companies for research and education, and for advice. It does not support one company's products over another.

The drawing on page 1 is adapted from one provided by Wendy Tyrrell, School of Health and Social Sciences, University of Wales Institute, Cardiff.