BMJ Best Practice

# Pressure ulcer

The right clinical information, right where it's needed

Last updated: Nov 18, 2017

# Table of Contents


Summary 3

Basics 4

- Definition 4
- Epidemiology 4
- Aetiology 4
- Pathophysiology 5

Prevention 6

- Primary prevention 6
- Secondary prevention 7

Diagnosis 8

- Case history 8
- Step-by-step diagnostic approach 8
- Risk factors 11
- History & examination factors 12
- Diagnostic tests 13
- Differential diagnosis 14
- Diagnostic criteria 15

Treatment 18

- Step-by-step treatment approach 18
- Treatment details overview 20
- Treatment options 21
- Emerging 27

Follow up 28

- Recommendations 28
- Complications 28
- Prognosis 29

Guidelines 30

- Diagnostic guidelines 30
- Treatment guidelines 30

Online resources 33

Evidence scores 34

References 36

Disclaimer 43

# Summary

- Pressure ulcers are commonly encountered in patients admitted to hospital and those in long-term care facilities.
- Older people, and all patients with limited mobility or impaired sensation, are at particular risk.
- Pressure damage most commonly occurs over bony prominences but can develop on any part of the body subjected to sustained localised pressure.
- Pressure damage varies from small superficial lesions to extensive wounds with bony involvement that contain a mass of necrotic tissue.
- Prevention is better than cure; all patients at risk of sustaining pressure damage should be assessed and provided with appropriate pressure-reducing strategies. Using support surfaces, repositioning the patient, optimising nutritional status, and moisturising sacral skin are appropriate strategies to prevent pressure ulcers.
- Management of pressure ulcers is determined by the location and condition or severity of the wound. Wounds should be managed in accordance with wound care practices or policies.

# Definition

Pressure ulcers have been defined by the National Pressure Ulcer Advisory Panel and the European Pressure Ulcer Advisory Panel as localised injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure or of pressure in combination with shear.[1]

Pressure ulcers most commonly occur over bony prominences but can develop on any part of the body, including mucosal surfaces. They may be small, superficial wounds or blisters involving only epidermal elements or larger (sometimes massive) wounds, often covered or filled with necrotic tissue and involving deeper tissues, including fascia, muscle, or bone.

# Epidemiology

Accurate assessments of pressure ulcer incidence and prevalence are difficult to obtain. Studies differ considerably in their methodology. Lower rates are generally seen in those studies using large databases to examine thousands of patients, raising questions as to the ascertainment of ulcer status and the validity of the results. Studies involving the direct examination of patients generally have higher rates but the small sample sizes raise questions as to generalisability.

A comparative study of patients with pressure ulcer in the UK, US, and Canada demonstrated prevalence in hospitalised patients of 4.7% to 32.1%. In patients in community care the prevalence ranged from 4.4% to 33.0% and was 4.6% to 20.7% in patients in nursing homes.[2] A European study involving nearly 6000 patients in 5 countries reported that about 18% of patients admitted to hospital had a pressure ulcer.[1] Among elderly patients being seen by a general practitioner in the UK, an overall incidence rate of 0.58 ulcers per 100 person-years was noted; rates were considerably higher in people over age 85.[3] The US National Pressure Ulcer Advisory Panel has reviewed data from nearly 300 studies published between 2000 and 2011 on the incidence and prevalence of pressure ulcers across care settings in specific populations, such as people with spinal cord injuries, older people, infants, and children. Rates varied widely among the studies. Pressure ulcer incidence rates (over various time frames, such as hospital stay) in general acute care ranged from 2.8% to 9.0%. In long-term care, the incidence rates (again over various time frames) ranged from 3.6% to over 50%, and in home care from 4.5% to 6.3%. Prevalence rates ranged from 11.9% to 15.8% in general acute care, 4.3% to 32% in long-term care, and 2.9% to 19.1% in home care. Most pressure ulcers, regardless of setting, were located on the sacrum or coccyx and heels.[4] A 6-year sequential pressure ulcer prevalence survey conducted in the US, published in 2004 and involving up to 240 participating healthcare facilities, reported that prevalence ranged from a low of 14% (2001 and 2002) to a high of 17% (1999). Incidence ranged from a low of 7% (2001, 2003, 2004) to a high of 9% (2000).[5] The prevalence of pressure ulcers in children admitted to hospital varies from 0.47% to 13.1%.[6] Prevalence rates in patients with spinal injury are 20% to 30%, 1 to 5 years after injury. One survey reported that 23% of spinal injury patients in the community had pressure damage.[7]

# Aetiology

Pressure ulcers have traditionally been thought to develop through the interplay of 4 main factors: pressure, shear, friction, and moisture. Pressure is clearly the most important factor and both the duration and intensity of pressure are important. Irreversible tissue damage may occur as a result of relatively short exposures to high levels of pressure or longer exposure to lower levels of pressure. While it is generally recommended that patients be repositioned every 2 hours, it is likely that in some situations, considerably less time is required

for tissue damage to occur. Longer time intervals between repositionings might be appropriate if pressure is sufficiently relieved with an appropriate support surface.[8] Pressure forces are distributed throughout the soft tissue, the extent depending on the mechanical properties of both the soft tissues and any external devices.[9] Shear forces, typically generated when patients slide down in bed, may cause stretching and tearing of small blood vessels and contribute to pressure-induced damage.

The role of friction and moisture in the development of pressure ulcers, and in particular deep tissue injury, is less certain.[10] Both can clearly cause superficial injuries including skin tears and moisture-associated dermatitis. These superficial injuries could facilitate the transmission of pressure to the deeper tissues.

The role of circulation and tissue perfusion in the development of pressure ulcers is being increasingly recognised.[11] Inadequate blood flow to the skin and soft tissues, as occurs in the setting of sepsis, hypotension, heart failure, or peripheral vascular disease, almost certainly contributes to tissue ischaemia and pressure ulceration. Physiological changes occurring at life's end, including decrease perfusion, have also been used to describe the concept of 'skin failure' and the high incidence of pressure ulcers with terminal disease.[12]

Pressure ulcers occur most commonly over bony prominences and are caused principally by unrelieved interfacial pressure that exceeds capillary pressure.[13] The risk factors for the development of pressure damage include the following: increased age,[14] reduced mobility, neurological impairment resulting in loss of sensation or paralysis, and other conditions that prevent normal self-repositioning. Relatively short exposure to high levels of pressure can cause serious damage.[15] Pressures generated between the body and a hard surface may be transmitted through underlying tissue to a point well away from the original area of contact. Shearing and frictional forces produced when a patient slides down or is pulled up in a bed can also contribute to tissue damage. These forces stretch, distort, and ultimately damage or occlude small blood and lymph vessels contributing to localised ischaemia, reperfusion injury, and tissue death.[15]

Hundreds of patient characteristics have been suggested as being risk factors for pressure ulcer development. In most cases, these risk factors are associated with aetiological factors such as pressure , moisture, or impaired circulation. However, for other proposed risk factors, the causal pathway may not be clear and further research is indicated. How these different risk factors impact on individual susceptibility is currently being researched.[16] A few of these factors are described in the 'risk factors' section.

# Pathophysiology

The mechanism by which pressure induces tissue death is not fully understood. Ischaemia caused by capillary occlusion may be the dominant factor but other explanatory factors exist. The blockage of lymphatic flow may lead to the accumulation of toxic waste products. Reperfusion injury with the accompanying free radical development may damage cells. Finally, it is increasingly recognised that prolonged pressure may directly damage cells, leading to cell death.

Highest pressures are seen in the deep tissues, especially along bony prominences. These deep tissues also appear to be most susceptible to pressure damage. Consequently, pressure often causes extensive deep tissue injury with little apparent damage initially in the more superficial tissues.These deep tissue injuries may then initially present as an area of dark purple discoloration with intact overlying skin. This lesion may then evolve over the course of several days or weeks to a large necrotic ulcer with extensive undermining of the wound edges.

# Primary prevention

Many pressure ulcers are avoidable through the use of appropriate preventive measures. Implementation of preventive measures not only will result in better patient outcomes but will also be cost saving.[23] Successful preventive programmes will have at least two primary components: identification of at-risk individuals and addressing of risk factors through the use of appropriate interventions. The evidence base for pressure ulcer preventive measures has been reviewed in the NPUAP and EPUAP guidelines,[1] and in a comparative effectiveness review for the US Agency for Healthcare Research and Quality.[24] A toolkit for preventing pressure ulcers in hospital patients has also been developed. [AHRQ: preventing pressure ulcers in hospitals: a toolkit for improving quality of care]

Identification of at-risk individuals

- All patients should be assessed for their risk of pressure ulcer development on admission and periodically thereafter.[25]
- Risk assessment tools to assist in evaluating a patient's need for pressure ulcer prevention are available but there is no evidence to suggest that they reduce the incidence of pressure ulcers.[26] [27] [28]

  Five of the scales in use are the Norton,[29] Waterlow,[30] Waterlow Scale for Stratification of Pressure Sore Risk
  Stratifies risk of pressure sores based on multiple criteria.

  Braden,[31] [Prevention Plus: Braden scale for predicting pressure sore risk] Norton Scale to Stratify Risk of Pressure Sores
  Assessment scale for risk of pressure ulcers.

  the inter RAI Pressure Ulcer Risk Scale,[32] and the Ramstadius[33] scale. By looking at the total score, these scales have been shown to identify patients at higher risk of pressure ulcer development. Additionally, sub-scale scores can be reviewed to identify specific domains requiring interventions. The predictive value of these scales, though, was evaluated in a prospective cohort study involving 1229 patients.[34] The authors reported that, despite their widespread popularity, using these assessment scales to decide on preventive measures leads to ineffective and inefficient treatment for most patients.1[B]Evidence

  Conflicting advice followed a systematic review of risk assessment scales,[35] from which the authors concluded that, although there was no decrease in pressure ulcer incidence, which might be attributed to use of an assessment scale, both the Braden and Norton scales were more accurate than nurses' clinical judgement in predicting pressure ulcer risk.
- Comprehensive risk assessment involves more than the use of one of these prediction tools. The scales only examine a few domains and many important factors that place a patient at increased risk of pressure ulcers are not considered. All such factors should be identified.

Interventions to prevent pressure ulcers

- Interventions to prevent pressure ulcers should be tailored to the unique needs of each individual patient and should address areas identified using the comprehensive risk assessment. Evidence in support of various interventions has been reviewed in guidelines and systematic reviews.[1] [27]
- Pressure relief is critical. This is achieved through repositioning patients and use of an appropriate support surface. The optimal frequency with which patients should be repositioned is not known, and may vary depending on the support surface.[8] A 2011 RCT demonstrated that turning patients every 3 hours prevented the development of pressure ulcers.[36] More recently, a study conducted among nursing home residents (all of whom used high-density foam mattresses) found no difference in pressure ulcer incidence between those repositioned at 2-, 3-, or 4-hour intervals.[37] The standard of care is that at-risk individuals should be repositioned every 2 hours. This may be difficult to achieve in a busy clinical setting and use of prompting systems to encourage scheduled repositioning should be encouraged. [38] A systematic review concluded that, while there is a strong theoretical rationale for repositioning, there is only limited evidence from existing clinical trials.[39]

- Specialised support surfaces are better than standard hospital mattresses at preventing pressure ulcers.[1] [27] [39] However, evidence for the superiority of one specialised support surface over another is more limited. For example, in one randomised clinical trial of 1972 hospitalised patients with limited mobility, no differences in pressure ulcer incidence were seen with an alternating pressure mattress versus an alternating pressure overlay.[40] However, among nursing home residents using a wheelchair, a skin protection cushion with a fitted wheelchair was superior to a segmented foam cushion in preventing sacral and ischial tuberosity ulcers.[41] In patients with hip fracture, a heel elevation device, in addition to a support surface to redistribute pressure, reduces the incidence of heel ulcers.[42]
- Monitoring devices that provide continuous feedback on pressures may help with repositioning and the prevention of pressure ulcers.[43]
- Other interventions for the prevention of pressure ulcers include nutritional supports and use of skin moisturisers, particularly over the sacrum. While some studies have shown a benefit, results have not been consistent. A systematic review found no evidence of benefit for nutritional interventions to prevent pressure ulcers.[44]
- While dressings over bony prominences might help prevent pressure ulcers, a systematic review highlighted that evidence from existing trials was limited.[28]
- Given the many factors contributing to pressure ulcers, the prevention of pressure ulcers requires a team effort. Successful efforts at implementing a preventive programme generally require a multidisciplinary team to identify a bundle of best practices and test how best to implement this bundle within their organisation.[45] [AHRQ: preventing pressure ulcers in hospitals: a toolkit for improving quality of care] Key components of this bundle include standardisation of interventions and documentation, designation of 'skin champions', use of audit and feedback, and staff education.

# Secondary prevention

Preventing pressure damage depends on regular skin inspection of all patients who are judged to be potentially at risk and the timely use of pressure-reducing aids when early signs of damage are present or suspected. Risk assessment tools to evaluate patients' need for pressure ulcer prevention are available, but there is no evidence to suggest that they reduce the incidence of pressure ulcers.[26] [27] [28] Relatively minor changes in a patient's physical or mental health can have major implications for their predisposition to pressure damage, so regular inspection is essential if this occurs. Patients who have experienced pressure injuries previously are at particular risk. Attention should be paid to the positioning of tubes and catheters, particularly in infants, as these can cause localised tissue damage in areas not generally at risk of developing pressure injuries.

# Case history

## Case history #1

A 65-year-old man presents with fever and respiratory distress. He rapidly develops progressive hypoxia and hypotension requiring ventilator support and pressors. Efforts to reposition the patient result in marked desaturations. One week later, following stabilisation of his medical condition, he is noted to have an extensive area of tissue damage over his sacral region. The damage around the margin of the wound appears relatively superficial, with some signs of partial skin loss. Towards the centre, directly over the sacrum, is an area of necrotic tissue, which is starting to separate spontaneously, exposing a cavity containing viscous yellow slough. A purulent discharge is draining from this cavity.

## Case history #2

An 80-year-old woman in a residential care facility, who has recently suffered a stroke with a resulting right hemiplegia, presents with a painful area on right heel. On examination the heel is found to be covered with a layer of hard, black, necrotic tissue with a leathery appearance.

## Other presentations

Pressure ulcers, while typically thought of as a condition of frail elderly patients, may occur among patients of any age and in any setting. When not located over a bony prominence, pressure ulcers are usually the result of an external device. Examples include oxygen tubing causing a pressure ulcer on the ear or a lower leg cast causing an ulcer on the leg. External devices may also cause mucosal pressure injury.



# Step-by-step diagnostic approach

## Physical appearance

All patients should have a comprehensive skin examination at the time of admission and then at regular intervals thereafter. A diagnosis of pressure ulcer is typically obvious when an individual with risk factors develops evidence of skin damage over a bony prominence. Thus, damage is supported by the presence of one of the following:

- An area of non-blanchable erythema
- Marked localised skin changes
- A wound of varying severity on an anatomical site that is known (or suspected) to have previously been exposed to significant unrelieved pressure.

Multiple wounds may occur, often in symmetrical patterns (both heels and both buttocks, for example). Sometimes there is evidence of earlier healed wounds in areas at particular risk.

Pressure ulcer appearance is typically characterised by the depth of the wound. Since the early 1980s, many pressure ulcer classification schemes have been described in the literature. A review published in 1999 identified 16 different systems, which described 4, 5, or 6 grades of pressure damage.[46] All of

these systems are based on the physical appearance of the wound and the type of tissues affected, but this is often impossible to determine until the wound has been debrided sufficiently to reveal the full extent of the damage. Guidelines published jointly by the National Pressure Ulcer Advisory Panel (NPUAP) and the European Pressure Ulcer Advisory Panel (EPUAP) proposed the following scheme.[47] [48]

Category/grade/stage I: non-blanchable erythema

- Intact skin with non-blanchable redness of a localised area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding area.
- The area may be painful, firm, soft, or warmer or cooler compared with adjacent tissue. Category I may be difficult to detect in individuals with dark skin tones. May indicate 'at-risk' persons.

Category/grade/stage II: partial thickness

- Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled or serosanguinous-filled blister.
- Presents as a shiny or dry shallow ulcer without slough or bruising. Bruising indicates deep tissue injury. This category should not be used to describe skin tears, tape burns, incontinence-associated dermatitis, maceration, or excoriation.

Category/grade/stage III: full-thickness skin loss

- Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon, or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunnelling.
- The depth of a category/stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have (adipose) subcutaneous tissue and category/stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep category/stage III pressure ulcers. Bone/tendon is not visible or directly palpable.

Category/grade/stage IV: full-thickness tissue loss

- Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present. Often includes undermining and tunnelling.
- The depth of a category/stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have (adipose) subcutaneous tissue and these ulcers can be shallow. Category/stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, or joint capsule) making osteomyelitis or osteitis likely to occur. Exposed bone/ muscle is visible or directly palpable.

In the US, the NPUAP recognises four additional stages, described below.[48]

Unstageable/unclassified: full-thickness skin or tissue loss; depth unknown

- Full-thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, grey, green, or brown) and/or eschar (tan, brown, or black) in the wound bed. Until enough slough and/or eschar are removed to expose the base of the wound, the true depth cannot be determined, but it will be either a category/stage III or IV.
- Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as 'the body's natural (biological) cover'; sharp debridement of the eschar should often be avoided.



 

Suspected deep tissue injury; depth unknown

- Purple or maroon localised area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, or warmer or cooler compared with adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid, exposing additional layers of tissue even with optimal treatment.

Medical device-related pressure injury

- Medical device-related pressure injuries result from the use of devices designed and applied for diagnostic or therapeutic purposes. The resultant pressure injury generally conforms to the pattern or shape of the device. The injury should be staged using the staging system.

Mucosal membrane pressure injury

- Mucosal membrane pressure injury is found on mucous membranes with a history of a medical device in use at the location of the injury. Due to the anatomy of the tissue, these ulcers cannot be staged.

As part of the guideline development process, the NPUAP and EPUAP have discussed the use of terminology for an international pressure ulcer definition and classification system. It is proposed that whatever word, stage, grade or category, is most clear and understood should be used in describing pressure ulcers, although the actual definitions of pressure ulcers and the levels of skin-tissue injury are the same.

For the purpose of consistency, stage will be used for description in this monograph.



## History and risk factors

The pressure ulcer history should elucidate details about the ulcer including duration, presence of pain, and symptoms of infection (fever, exudates, odour), as well as information on predisposing risk factors that contributed to the development of the pressure ulcer. Risk factors include the following: immobility, sensory impairment, older age, surgery, intensive care stay, malnourishment, history of previous pressure ulcers, faecal or urinary incontinence, diabetes, and peripheral vascular disease.

## Environmental factors

The likelihood of developing pressure damage is greatly influenced by the nature of the surface on which the patient has been sitting or lying. Conventional mattresses, operating tables, trolleys, and wheelchair support surfaces do not provide adequate protection against pressure damage for patients who are unable to reposition themselves regularly for whatever reason. An immobile patient who has been placed on an inappropriate mattress is greatly at risk of developing pressure damage, and therefore consideration of these factors can add support to a diagnosis of pressure-induced injury.

## Investigations

Diagnostic tests are not used to make the diagnosis of pressure ulcers but may help in diagnosing complications of pressure ulcers including wound infection and osteomyelitis.

The diagnosis of a pressure ulcer is typically based on clinical grounds and there are no specific tests to confirm the diagnosis. Standard microbiological tests may be required to exclude the possibility of

infection or osteomyelitis. These tests may include FBC, inflammatory markers, or MRI. In cases of suspected wound infection, wound swab or deep tissue biopsy may rarely be required for confirmation, although wound infection is usually a clinical diagnosis. It should be noted that, while commonly used, wound swabs have not been shown to be useful in the management of potential infected pressure ulcers. Wound swab culture results often reflect colonisation and not actual infection. Consequently, guidelines do not recommend routine use of wound swabs. Particular care should be taken to differentiate pressure ulcers from moisture-associated dermatitis.

Where a diagnosis of pressure damage is uncertain, as in the case of wounds on the lower legs, tests may be appropriate to exclude alternative causes such as diabetes (blood glucose), venous insufficiency, or arterial impairment (vascular assessment).

A dermatological referral may be appropriate for wounds where there is no clear evidence that the damage was sustained from unrelieved pressure, to exclude the possibility of, for example, pyoderma gangrenosum.

# Risk factors

## Strong

### immobility

- All patients who have impaired mobility are at risk of developing pressure ulcers. Immobility may be permanent, such as from a cerebrovascular accident, or transitory, such as from sedative or restraint use.

### sensory impairment

- Neurological impairment resulting in loss of sensation may prevent normal self-repositioning in response to noxious stimuli.

### older age

- There is a strong correlation between increased age and pressure ulcer development. Up to two-thirds of ulcers occur on patients >70 years of age.[14] It is unclear whether this reflects age-related skin changes or the fact that conditions causing immobility are more common in older people.

### surgery

- Surgery, with its accompanying immobility and impaired sensation, places patients at considerable risk of pressure ulcer development. The risk may be particularly great with orthopaedic surgery following a hip fracture.[17]

### intensive care stay

- Patients in ICUs are at high risk of developing pressure ulcers due to immobility, sedation, and cardiovascular instability. Risk increased as a function of time such that the cumulative risk was 50% at 20 days.[18]

### malnourishment

- Malnutrition is very common among pressure ulcer patients. A mini-nutritional assessment conducted in 484 multimorbid older patients revealed that 39.5% of patients with ulcers were malnourished



 

and 2.5% were well nourished. Of patients without pressure ulcers, 16.6% were malnourished and 23.6% were well nourished.[19] How malnutrition interacts with pressure to cause skin damage is uncertain but may include alterations in skin resistance to pressure damage or impaired healing of early pressure-induced damage.

- Malnutrition also relates to body weight. Lower body-mass index is clearly associated with increased risk of pressure ulcer development; the risk at a very high body-mass is less certain.[20]

### Hx of previous pressure ulcers

- Stage 3 and 4 pressure ulcers heal through a process involving wound contraction and scar tissue formation. The resulting tissue is not normal skin and is particularly prone to break down again.

### environmental factors

- The likelihood of developing pressure damage is greatly influenced by the nature of the surface on which the patient has been sitting or lying. Conventional mattresses, operating tables, trolleys, and wheelchair support surfaces do not provide adequate protection against pressure damage for patients who are unable to reposition themselves regularly for whatever reason. An immobile patient who has been nursed on an inappropriate mattress is greatly at risk of developing pressure damage, and therefore consideration of these factors can add support to a diagnosis of pressure-induced injury.

## Weak

### faecal or urinary incontinence

- Skin wetness is frequently cited as a contributory factor to skin damage and moisture-associated dermatitis. How moisture may lead to deep tissue damage is less certain and studies suggest that urinary incontinence is only an indicator for other risk factors or a measure of the need for care without any causal relation to pressure sores.[21] Faecal incontinence may be a more significant risk factor, perhaps as a result of toxic substances present in faeces.[22]

### diabetes

- People with diabetes appear to be at higher risk of pressure ulcer development.[20] The mechanism is likely to be multifactorial and include impaired sensation, poor circulation, and impaired healing of early pressure-induced skin damage.

### peripheral vascular disease

- People with PVD have poor blood flow to the legs and are at particular risk for pressure ulcers of the heel.

# History & examination factors

## Key diagnostic factors

### presence of risk factors (common)

- Key risk factors include immobility, age >70 years, recent surgery or intensive care stay, and malnutrition.

### use of non-pressure-relieving support surface (common)

- A patient of any age with a wound or area of discolored skin who has been lying totally immobile for an extended period on a surface not specifically designed to reduce the possibility of pressure damage.

### localised skin changes on areas subjected to pressure (common)

- Non-blanching erythema or purple or maroon localised area of discolored intact skin, which may be painful, firm, mushy, boggy, or warmer or cooler than adjacent tissue.
- This indicates early stage of tissue damage or probable wound formation.

### shallow open wound or tissue loss on areas subjected to pressure (common)

- A blister or a shiny or dry shallow ulcer involving partial loss of dermis without slough indicates a grade 2 pressure ulcer.

### full-thickness wound on areas subjected to pressure with or without undermining (tunnelling) (common)

- Full-thickness wound possibly containing some slough with no bone tendon or muscle involvement/ exposure indicates a grade 3 pressure ulcer.

### full-thickness wound with involvement of major tissues on areas subjected to pressure with or without undermining (tunnelling) (common)

- Full-thickness tissue loss with exposed bone, tendon, or muscle possibly containing slough or eschar on some parts of the wound bed indicates a grade 4 pressure ulcer.
- When there is exposed bone, osteomyelitis should be considered.[49]

### localised tenderness and warmth around area of wound (common)

- Suggests infection.

### increased exudate and/or foul odour (common)

- Indicators of infection include the following: development of odour and excess exudate from a previously clean wound, change in the appearance of the wound bed, or a sudden deterioration in the condition of the wound or the patient.

## Diagnostic tests

### 1st test to order

| Test | Result |
|---|---|
| **clinical diagnosis**<br>• Diagnostic tests are not used to make the diagnosis of pressure ulcers but may help in diagnosing complications of pressure ulcers including wound infection and osteomyelitis. | **features of pressure ulcer** |



 

## Other tests to consider

| Test | Result |
|---|---|
| **wound swab**<br>• Often done when signs of infection evident. However, while commonly used, wound swabs have not been shown to be useful in the management of potential infected pressure ulcers.<br>• Culture results often reflect colonisation and not actual infection. Consequently guidelines do not recommend routine use of wound swabs. | **positive culture in infection** |
| **ESR**<br>• Suggests osteomyelitis; not confirmatory of pressure ulcer. | **>100 mm/hour in osteomyelitis** |
| **WBC**<br>• Suggests osteomyelitis; not confirmatory for pressure ulcers. | **>15.0 x 10^9/L (>15,000/microL) in osteomyelitis** |
| **serum glucose**<br>• To exclude diabetes. | **elevated in diabetes** |
| **deep tissue biopsy**<br>• The definitive method for diagnosing infection. Not always practical in a clinical setting. Diagnosing infection is a clinical skill rather than a microbiological technique.[50] | **evidence of infection** |
| **MRI**<br>• Appropriate whenever bony involvement is possible; not confirmatory for pressure ulcers. | **evidence of bony involvement** |

# Differential diagnosis

| Condition | Differentiating signs / symptoms | Differentiating tests |
|---|---|---|
| **Moisture-associated dermatitis** | • Erythematous, macerated skin in an area that is exposed to chronic moisture. | • There are no differentiating tests; diagnosis is clinical. |
| **Venous ulcers** | • Commonly occur on lower legs near ankles; frequently accompanied by skin staining.<br>• May be painful.<br>• May be associated with lipodermatosclerosis, in which the lower part of the leg is hardened. | • Duplex ultrasound demonstrates retrograde or reversed flow; valve closure time >0.5 seconds indicative of venous insufficiency. |

| Condition | Differentiating signs / symptoms | Differentiating tests |
|---|---|---|
| **Arterial ulcers** | • Found on feet, heels, or toes.<br>• Surrounding skin often white and shiny.<br>• Typically most painful in bed; pain sometimes relieved by having the legs dependent.<br>• Ulcers often have a pale base and a punched-out appearance. | • Vascular assessment and an ankle brachial index ≤0.90. |
| **Diabetic neuropathy** | • Neuropathic diabetic ulcers are caused by pressure resulting often from ill-fitting shoes or failure to reposition limb due to impaired perception of pain. Ulcers commonly occur on the toes, the heels, or other parts of the foot. | • HbA1c may support poor glucose control. |
| **Pyoderma gangrenosum** | • Most often affects people in their 40s or 50s.<br>• In approximately 50% of patients, it is associated with other conditions, including inflammatory bowel disease.<br>• Characteristically, the edge of an ulcer is purple and undermined (tunnelled) as it enlarges.<br>• Ulcers are painful and most commonly occur on the legs. Unlike pressure ulcers, pyoderma gangrenosum ulcers start as a small discrete area and enlarge rapidly. | • There are no differentiating tests; diagnosis is clinical. |
| **Osteomyelitis** | • Typical symptoms are bone pain, tenderness, and swelling. May be a previous history of osteomyelitis, recent surgery, or penetrating injury. If bone is exposed in a suspected pressure ulcer, osteomyelitis should be excluded.[49] | • MRI is diagnostic.<br>• Inflammatory markers are elevated. |



# Diagnostic criteria

Since the early 1980s, many pressure ulcer classification schemes have been described in the literature. A review published in 1999 identified 16 different systems, which described 4, 5, or 6 grades of pressure

 

damage.[46] All of these systems are based on the physical appearance of the wound and the type of tissues affected, but this is often impossible to determine until the wound has been debrided sufficiently to reveal the full extent of the damage. Guidelines published jointly by the National Pressure Ulcer Advisory Panel (NPUAP) and the European Pressure Ulcer Advisory Panel (EPUAP) proposed the following scheme.[47] [48]

Category/grade/stage I: non-blanchable erythema

- Intact skin with non-blanchable redness of a localised area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding area.
- The area may be painful, firm, soft, or warmer or cooler compared with adjacent tissue. Category I may be difficult to detect in individuals with dark skin tones. May indicate 'at-risk' persons.

Category/grade/stage II: partial thickness

- Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled or serosanguinous-filled blister.
- Presents as a shiny or dry shallow ulcer without slough or bruising. Bruising indicates deep tissue injury. This category should not be used to describe skin tears, tape burns, incontinence-associated dermatitis, maceration, or excoriation.

Category/grade/stage III: full-thickness skin loss

- Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon, or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunnelling.
- The depth of a category/stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have (adipose) subcutaneous tissue and category/stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep category/stage III pressure ulcers. Bone/tendon is not visible or directly palpable.

Category/grade/stage IV: full-thickness tissue loss

- Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present. Often includes undermining and tunnelling.
- The depth of a category/stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have (adipose) subcutaneous tissue and these ulcers can be shallow. Category/stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, or joint capsule) making osteomyelitis or osteitis likely to occur. Exposed bone/muscle is visible or directly palpable.

In the US, the NPUAP recognises four additional stages, described below.[48]

Unstageable/unclassified: full-thickness skin or tissue loss; depth unknown

- Full-thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, grey, green, or brown) and/or eschar (tan, brown, or black) in the wound bed. Until enough slough and/or eschar are removed to expose the base of the wound, the true depth cannot be determined, but it will be either a category/stage III or IV.
- Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as 'the body's natural (biological) cover'; sharp debridement of the eschar should often be avoided.

Suspected deep tissue injury; depth unknown

- Purple or maroon localised area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, or warmer or cooler compared with adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid, exposing additional layers of tissue even with optimal treatment.

Medical device-related pressure injury

- Medical device-related pressure injuries result from the use of devices designed and applied for diagnostic or therapeutic purposes. The resultant pressure injury generally conforms to the pattern or shape of the device. The injury should be staged using the staging system.

Mucosal membrane pressure injury

- Mucosal membrane pressure injury is found on mucous membranes with a history of a medical device in use at the location of the injury. Due to the anatomy of the tissue, these ulcers cannot be staged.

As part of the guideline development process, the NPUAP and EPUAP have discussed the use of terminology for an international pressure ulcer definition and classification system. It is proposed that whatever word, stage, grade or category, is most clear and understood should be used in describing pressure ulcers, although the actual definitions of pressure ulcers and the levels of skin-tissue injury are the same.

# Step-by-step treatment approach

Guidelines and systematic reviews on the treatment of pressure ulcers have been published.[1] [51] [52] As with prevention, pressure relief is critical to pressure ulcer treatment and patients should not be positioned on their wound. Pressure relief is achieved through repositioning and use of an appropriate support surface. No good trials have determined the optimal turning frequency. General consensus states that patients should be repositioned every 2 hours, but there is no evidence to suggest that this improves the healing rate of ulcers.[53] [54]

Appropriate pressure-reducing aids, including mattresses and wheelchair or seat cushions, should be immediately provided. Little evidence supports the use of a specific support surface over other alternatives.[39] [51]

Nutritional support is also recommended for patients with pressure ulcers, particularly if there is evidence of malnutrition. Goals should be to provide at least 30 to 35 kcal/kg body weight and 1.2 to 1.5 g protein/kg body weight. Additional protein or amino acid supplementation can enhance healing; however, there is limited evidence to support this.[44] [55] A small trial found that a nutritional formula enriched with arginine, zinc, and antioxidants led to improved healing.[56] Anabolic steroids have been used to promote weight gain and healing in patients with chronic wounds. However, a randomised clinical trial of oxandrolone in spinal cord injury patients with pressure ulcers failed to show any benefit compared with placebo.[57]

Another critical aspect of pressure ulcer treatment is the assessment and management of pain.[58] Assessment is aimed at identifying the type and extent of pain present so that appropriate therapy may be provided. Intermittent pain such as occurs at the time of wound debridement should be managed with an oral analgesic given 30 to 60 minutes before the procedure. Additionally, topical lidocaine applied to the wound may be of benefit. Management of cyclic pain occurring at the time of a dressing change depends on the extent of the pain. Mild pain can usually be managed with paracetamol or a non-steroidal anti-inflammatory drug (NSAID). Moderate to severe pain is usually managed with an opioid. Codeine or oxycodone, often given in combination with paracetamol, can be used for moderate pain, while severe pain may require oral or intravenous morphine. These same medications may be used for persistent pressure ulcer pain. Attention to the dressing used may also help alleviate cyclic pain. Ibuprofen-releasing foam dressings[59] or topical morphine[60] applied to the ulcer pain may also be considered; however, these therapies are generally not available as proprietary products in most countries, and may need to be compounded by a pharmacist.

Additional treatments for pressure ulcers should be tailored to their unique characteristics.

Stage I ulcers are treated, as for all patients, with pressure relief, good hygiene practice, and skin care, particularly in the sacral region. Additional treatments should be applied to stage 2 to 4 pressure ulcers.

## Superficial ulcers (stage 2 or shallow stage 3 ulcers)

These pressure ulcers should be managed conservatively with good hygiene and skin care, and the application of appropriate dressings. A hydrocolloid dressing typically will provide a suitable environment to promote healing.

## Deep ulcers (deep stage 3 or stage 4)

Wound-bed preparation is key to healing of these deep wounds.[61] If necrotic tissue is present, the wound may require initial debridement. This may be achieved in several ways: sharp debridement;[62] autolytic debridement (using products such as hydrogels to facilitate autolysis); or the application of

enzymatic agents,[63] maggots,[64] [65] or high-pressure water jet.[66] There is no clear evidence on the most effective form of debridement.2[C]Evidence However, if there is concern of a serious deep tissue infection, immediate sharp debridement is indicated. Once free of necrosis, the wound should be dressed with suitable products designed to keep the wound bed moist and promote granulation and epithelialisation. Although honey-impregnated dressings are being popularised, there is no evidence of efficacy in improving wound-healing rates.[68] There is little evidence to suggest a preference for any specific type of wound-cleansing solution or technique, although a single, small clinical trial has suggested that a saline spray containing aloe vera, silver chloride, and decyl glucoside, may be better than saline alone.[69]

In the absence of clear evidence from RCTs on the relative merits of different types of dressings available, topical therapy should be chosen using a structured approach.[70] [71] 3[C]Evidence This includes assessing the condition of the wound using clearly identified criteria and treatment objectives, such as ulcer stage, condition of wound bed, presence of infection, level of exudate, degree of pain, condition of surrounding skin, position, and patient's preference.[72]

Surgery may be considered in patients whose ulcers are not healing with conservative therapy or when rapid closure is desirable. Options may include ulcer excision, skin grafting, and flap formation. Recurrence rates can be high even when patients are carefully selected. A diverting colostomy may be considered in the setting of faecal soilage of large sacral wounds but the benefits have not been well defined.

## Management of infection

A wound containing quantities of necrotic tissue or slough will inevitably contain bacteria, typically *Staphylococcus aureus* , *Proteus mirabilis* , *Pseudomonas aeruginosa* , and certain *Bacteroides* species. A swab taken from such a wound will usually detect large numbers of organisms, a finding that may initiate unnecessary treatment with antibiotics. It has therefore been recommended that wounds should be cultured after wound infection has been clinically diagnosed, and not swabbed routinely.[1]

When there are clinical signs of infection that do not respond to treatment, radiological examination should be undertaken to exclude osteomyelitis and joint infection. Appropriate systemic antibiotic therapy should be initiated for patients with sepsis, advancing cellulitis, or osteomyelitis, all of which are complications. Systemic antibiotics are not required for pressure ulcers that exhibit only clinical signs of local infection.[1] In these situations, topical antimicrobial therapy may be indicated. One systematic review emphasised that the effects of topical and systemic antimicrobial treatments remain unclear.[73] The routine use of medicated dressings in other circumstances is probably not indicated.[74]

## Adjunctive therapies

There is no evidence that adjunctive therapies improve pressure ulcer healing.[51]

There is insufficient evidence to demonstrate clinical benefit of vacuum therapy (or topical negative pressure therapy), although it is widely used.[75] [76] [77] [78] [79] [80] 4[C]Evidence

While hyperbaric oxygen therapy may provide short-term benefit for lower extremity diabetic ulcers, there is little evidence for its use in patients with pressure ulcers.[81] [82] 5[C]Evidence

Other systematic reviews found insufficient evidence to reach conclusions regarding the contributions of laser therapy, therapeutic ultrasound, ultraviolet (UV) therapy, and electromagnetic therapy to chronic wound healing. Overall, there are limited data to support routine use of these expensive adjunctive



 

therapies in managing pressure ulcers.[83] [84] However, there is some evidence to support the use of electrical stimulation therapy to enhance wound healing.[55]

# Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. ( see Disclaimer )

## Acute ( summary )

| Patient group | Tx line | Treatment |
|---|---|---|
| all patients | 1st | pressure-reducing aids + repositioning |
| | plus | hygiene and cleansing + dressings |
| | plus | analgesia |
| | plus | dietary optimisation |
| | adjunct | antimicrobial therapy |
| ▪ deep ulcers (deep stage 3 or stage 4): patient unsuitable for surgery | plus | debridement of necrotic tissue |
| ▪ deep ulcers (deep stage 3 or stage 4): patient suitable for surgery | plus | surgical debridement and reconstruction with flap formation |

# Treatment options

## Acute

| Patient group | Tx line | Treatment |
|---|---|---|
| **all patients** | **1st** | **pressure-reducing aids + repositioning** |

» Treatment for all patients should include pressure relief, good hygiene practice, and skin care, particularly in the sacral region.

» Guidelines and systematic reviews on the treatment of pressure ulcers have been published.[1] [51] As with prevention, pressure relief is critical to pressure ulcer treatment and patients should not be positioned on their wound. Pressure relief is achieved through repositioning and use of an appropriate support surface. No good trials have determined the optimal turning frequency. General consensus states that patients should be repositioned every 2 hours, but there is no evidence to suggest that this improves the healing rate of ulcers.[53] [54]

» Appropriate pressure-reducing aids, including mattresses and wheelchair or seat cushions, should be immediately provided. Little evidence supports the use of a specific support surface over other alternatives.[39] [51]

» Compared with standard mattresses, using special foam mattresses reduces the incidence of pressure ulcers in high-risk patients, but there is no clear evidence relating to which mattresses are preferred.[39] 6[C]Evidence 7[C]Evidence The presence of excess moisture may exacerbate damage caused by frictional or shear force, so it is important to ensure that the skin of incontinent patients is regularly cleansed and dried and protective creams applied as appropriate. A full nutritional assessment should also be performed.[85]

| | | |
|---|---|---|
| | **plus** | **hygiene and cleansing + dressings** |

» Treatment for all patients should include pressure relief, good hygiene practice, and skin care, particularly in the sacral region.

» Superficial pressure ulcers (stage 2 or shallow stage 3) should be managed conservatively with good hygiene and skin care, and the application of appropriate dressings. A hydrocolloid dressing typically will provide a suitable environment to promote healing.

## Acute

| Patient group | Tx line | Treatment |
|---|---|---|
| | | » Topical treatment of all these wounds depends on location, condition, and severity. The aim of topical treatment is to achieve a clean wound bed, an essential precursor to healing.[63] [86] |
| | | » Once the wound has been cleansed the choice of dressing is influenced by several factors including the depth and size of the wound and the amount of exudate produced.[72] There is no evidence to suggest a preference for any specific type of wound-cleansing solution or technique.[87] |
| | | » In the absence of clear evidence from RCTs on the relative merits of different types of dressings available, topical therapy should be chosen using a structured approach.[70] [71] 3[C]Evidence This includes assessing the condition of the wound using clearly identified criteria and treatment objectives, such as ulcer stage, condition of wound bed, presence of infection, level of exudate, degree of pain, condition of surrounding skin, position, and patient's preference.[72] |
| | | » In the absence of hard evidence for the optimal treatment for different types of wounds, this is generally determined by local policies and formularies. |
| | **plus** | **analgesia** |
| | | » Another critical aspect of pressure ulcer treatment is the assessment and management of pain.[58]Assessment is aimed at identifying the type and extent of pain present so that appropriate therapy may be provided. Intermittent pain such as occurs at the time of wound debridement should be managed with an oral analgesic given 30 to 60 minutes before the procedure. Additionally, topical lidocaine applied to the wound may be of benefit. Management of cyclic pain occurring at the time of a dressing change depends on the extent of the pain. Mild pain can usually be managed with paracetamol or a non-steroidal anti-inflammatory drug (NSAID) such as ibuprofen. Moderate to severe pain is usually managed with an opioid. Codeine or oxycodone, often given in combination with paracetamol, can be used for moderate pain, while severe pain may require oral or intravenous morphine. These same medications may be used for persistent pressure ulcer pain. |

## Acute

| Patient group | Tx line | Treatment |
|---|---|---|
| | | » Attention to the dressing used may also help alleviate cyclic pain. Ibuprofen-releasing foam dressings[59] or topical morphine[60] applied to the ulcer may also be considered; however, these therapies are generally not available as proprietary products in most countries, and may need to be compounded by a pharmacist.<br><br>» The therapies listed below may be used as monotherapy, or some drugs may be combined as described above.<br><br>**Primary options**<br><br>» paracetamol: 500-1000 mg orally every 4-6 hours when required, maximum 4000 mg/day<br><br>**OR**<br><br>**Primary options**<br><br>» lidocaine topical: (5% ointment) apply to the affected area(s) two to three times daily when required<br><br>**OR**<br><br>**Primary options**<br><br>» ibuprofen: 200-400 mg orally every 4-6 hours when required, maximum 2400 mg/day<br><br>**OR**<br><br>**Primary options**<br><br>» codeine phosphate: 30-60 mg orally every 4 hours when required, maximum 240 mg/day<br><br>**OR**<br><br>**Primary options**<br><br>» oxycodone: 5-15 mg orally (immediate-release) every 4-6 hours when required<br><br>**OR**<br><br>**Primary options**<br><br>» morphine: 10 mg orally (immediate-release) every 4 hours when required; 2.5 to 5 mg intravenously every 3-4 hours when required |
| | **plus** | **dietary optimisation**<br><br>» All patients at risk of pressure ulcers or who have pressure ulcers should be referred to a |



 

## Acute

| Patient group | Tx line | Treatment |
|---|---|---|
| | | dietitian to ensure adequate total calorie and total protein intake.[88]<br><br>» An abbreviated nutritional assessment, as defined by the Nutrition Screening Initiative,[89] should be performed at least every 3 months for patients at risk for malnutrition. These include patients who are unable to take food by mouth or who experience an involuntary change in weight.<br><br>» If normal feeding mechanisms and dietary supplements are insufficient to meet the nutritional needs of the patient, nutritional support (usually by tube feeding) should be used to place the patient into positive nitrogen balance (approximately 30-35 kcal/kg/day and 1.25-1.50 g of protein/kg/day). As much as 2 g of protein/kg/day may be needed in some instances, together with vitamin and mineral supplements, where deficiencies are suspected or confirmed.<br><br>» There is no evidence that routine over-supplementation of nutrients improves healing. Additional protein or amino acid supplementation can enhance healing; however, there is limited evidence to support this.[55] A small trial found that a nutritional formula enriched with arginine, zinc, and antioxidants led to improved healing.[56] |
| | **adjunct** | **antimicrobial therapy**<br><br>» Unless clinical signs of infection are present, wounds should not be swabbed routinely.[1]<br><br>» Systemic antibiotic therapy is required for patients with bacteraemia, sepsis, advancing cellulitis, or osteomyelitis. Systemic antibiotics are not required for pressure ulcers that exhibit only signs of local infection.[1] In these situations topical antimicrobial therapy may be indicated in accordance with local policies and procedures. This may involve the use of dressings containing iodine, silver, or other antibacterial agents.<br><br>» The routine use of medicated dressings in other circumstances is probably not indicated.[74]<br><br>» In patients due for surgery, it is necessary to review the microbiological status of the wound and provide systemic antimicrobial cover where appropriate. |

## Acute

| Patient group | Tx line | Treatment |
|---|---|---|
| **deep ulcers (deep stage 3 or stage 4): patient unsuitable for surgery** | **plus** | **debridement of necrotic tissue**<br><br>» Wound-bed preparation is key to healing of these wounds.[61]<br><br>» Some wounds may be covered with necrotic tissue or a thick layer of slough and therefore require debridement using an appropriate technique such as sharp debridement;[62] autolytic debridement (using products such as hydrogels to facilitate autolysis); or the application of enzymatic agents,[63] maggots,[64] [65] or high-pressure water jet.[66] There is no clear evidence on the most effective form of debridement.2[C]Evidence However, if there is concern of a serious deep tissue infection, immediate sharp debridement is indicated. Once free of necrosis, the wound should be dressed with suitable products designed to keep the wound bed moist and promote granulation and epithelialisation. Although honey-impregnated dressings are being popularised, there is no evidence of efficacy in improving wound-healing rates.[68] There is little evidence to suggest a preference for any specific type of wound-cleansing solution or technique, although a single, small clinical trial has suggested that a saline spray containing aloe vera, silver chloride, and decyl glucoside, may be better than saline alone.[69] |
| **deep ulcers (deep stage 3 or stage 4): patient suitable for surgery** | **plus** | **surgical debridement and reconstruction with flap formation**<br><br>» Wound-bed preparation is key to healing of deep wounds.[61]<br><br>» Surgery may be considered in patients whose ulcers are not healing with conservative therapy or when rapid closure is desirable. Options may include ulcer excision, skin grafting, and flap formation. Recurrence rates can be high even when patients are carefully selected. A diverting colostomy may be considered in the setting of faecal soilage of large sacral wounds but the benefits have not been well defined.<br><br>» In patients who are candidates for surgical reconstruction, wounds are first subjected to extensive debridement followed by a suitable surgical procedure, typically a flap rotation, the precise nature of which is determined by the location of the wound. |



 

## Acute

| Patient group | Tx line | Treatment |
| --- | --- | --- |
| | | » Appropriate pressure relief, nutritional support, and skin care should be provided after surgery to prevent recurrence.<br><br>» General consensus states that patients should be repositioned, such as turning the patient every 2 hours, for both preventing and treating pressure ulcers, but there is no evidence to suggest that this improves the incidence or healing rates of ulcers.[53] [54] |

# Emerging

## Topical recombinant human granulocyte-macrophage colony-stimulating factor

Topical application of recombinant human granulocyte-macrophage colony-stimulating factor (rhGM-CSF) may have a beneficial effect on pressure ulcers but there is as yet insufficient evidence to recommend its use.[90]

## Topical phenytoin

Topical application of phenytoin (as a powder via an impregnated dressing, or as a cream) may have a beneficial effect on pressure ulcers. However, from the evidence available, it is uncertain whether it improves ulcer healing for grade I and II pressure ulcers. Further adequately powered RCTs are needed.[91]

FOLLOW UP

# Recommendations

## Monitoring

Patients at risk of pressure damage, or those who have had pressure damage in the past, should be monitored daily to check for early signs of recurrence. This should involve assessing the skin at areas of potential risk and ensuring that appropriate pressure-relieving aids are available and being used appropriately. Patients should also be regularly repositioned where this is possible.

## Patient instructions

Patients who are susceptible to pressure damage but are otherwise healthy and capable of taking some responsibility for their own care, such as spinal injury patients, should be fully instructed in the importance of regular repositioning, the need to have their skin checked regularly by a third party, and the need for timely intervention if early signs of damage are detected. Patients should also be reminded of the importance of diet and skin hygiene.

# Complications

| Complications | Timeframe | Likelihood |
|---|---|---|
| **sepsis** | **variable** | **medium** |
| Organisms present in wound invade local tissue and enter the blood stream. Treatment is systemic antibiotics, which should be determined by sensitivity testing. | | |
| **cellulitis** | **variable** | **medium** |
| If the pressure ulcer becomes infected, the infection may spread to surrounding tissues, resulting in cellulitis. This is treated with systemic antibiotics. | | |
| **osteomyelitis** | **variable** | **medium** |
| High-grade pressure ulcers may expose the bone and become infected. Osteomyelitis is treated with systemic antibiotics and surgical debridement. | | |
| **mortality** | **variable** | **low** |
| Study of in-hospital mortality has shown that mortality is higher for pressure ulcer-related hospitalisations, especially with a diagnosis of secondary pressure ulcers. Mortality in-hospital occurred in 11.6% of stays with secondary pressures ulcers, compared with 4.2% of stays for pressure ulcers as the primary diagnosis.[92] | | |

# Prognosis

- Spinal injury patients: assuming a patient is otherwise in good health, prognosis is good, as long as appropriate treatment is provided promptly; however, these patients are at high risk of recurrence.
- Seriously ill patients with extensive ulcers: for patients who have other serious medical problems, the prognosis is poor, as they may be unable to tolerate or accept the intensive treatment required to close their wound surgically.
- Infirm patients with superficial damage: prognosis is reasonable as long as appropriate local wound care is provided.

# Diagnostic guidelines

## Europe

### Pressure ulcers: prevention and management

**Published by:** National Institute for Health and Care Excellence **Last published:** 2014

**Summary:** Evidence-based advice on the prevention of pressure ulcers. Recommendations include methods for identification and risk assessment.

## North America

### Pressure injury staging illustrations

**Published by:** National Pressure Ulcer Advisory Panel **Last published:** 2016

### Risk assessment and prevention of pressure ulcers

**Published by:** Registered Nurses' Association of Ontario **Last published:** 2011

## Oceania

### Pan Pacific clinical practice guideline for the prevention and management of pressure injury

**Published by:** Australian Wound Management Association **Last published:** 2012

**Summary:** This evidence-based guideline includes recommendations on pressure injury risk assessment, prevention assessment, and monitoring.

# Treatment guidelines

## Europe

### Pressure ulcers: prevention and management

**Published by:** National Institute for Health and Care Excellence **Last published:** 2014

**Summary:** Evidence-based advice on the management of pressure ulcers. Includes recommendations on wound care, adjunctive therapies and support surfaces.

### Management of chronic venous leg ulcers: a national clinical guideline

**Published by:** Scottish Intercollegiate Guidelines Network **Last published:** 2010

### Best practice statement: prevention and management of pressure ulcers

**Published by:** Healthcare Improvement Scotland **Last published:** 2009

**Summary:** Guideline provides information on the prevention and management of pressure ulcers in which there is a lack of robust evidence. Therefore includes professional consensus.

## Europe

### Pressure relieving support surfaces: a randomised evaluation

**Published by:** Health Technology Assessment NHS R&D HTA Programme **Last published:** 2006

## International

### Prevention and treatment of pressure ulcers: quick reference guide

**Published by:** European Pressure Ulcer Advisory Panel; American National Pressure Ulcer Advisory Panel **Last published:** 2014

**Summary:** Summarises evidence-based guidelines on the prevention and treatment of pressure ulcers.

## North America

### Pressure ulcers and other wounds CPG

**Published by:** American Medical Directors Association **Last published:** 2017

### Risk assessment and prevention of pressure ulcers: a clinical practice guideline from the American College of Physicians

**Published by:** American College of Physicians **Last published:** 2015

**Summary:** This guideline provides clinical recommendations based on the comparative effectiveness of risk assessment scales and preventive interventions for pressure ulcers.

### Treatment of pressure ulcers: a clinical practice guideline from the American College of Physicians

**Published by:** American College of Physicians **Last published:** 2015

### Wound Healing Society 2015 update on guidelines for pressure ulcers

**Published by:** Wound Healing Society **Last published:** 2015

**Summary:** Provide recommendations on the prevention of pressure ulcers and include discussion of the following: Pressure Ulcer Risk Screening (PURS); Pressure Ulcer Risk Assessment (PURA); Pressure Ulcer Prevention Care Plan (PUPCP); selection of prevention interventions; consideration of positioning and support surfaces; consideration of friction and shear prevention interventions; management of moisture or incontinence; management of nutrition; healthcare provider, patient, and/or caregiver education; and the interdisciplinary approach.

### Risk assessment and prevention of pressure ulcers

**Published by:** Registered Nurses' Association of Ontario **Last published:** 2011

## Oceania

### Pan Pacific clinical practice guideline for the prevention and management of pressure injury

**Published by:** Wounds Australia (Australian Wound Management Association) **Last published:** 2012

**Summary:** This evidence-based guideline includes recommendations on pressure injury pain management and interventions for treatment.

# Online resources

1. AHRQ: preventing pressure ulcers in hospitals: a toolkit for improving quality of care *(external link)*

2. Prevention Plus: Braden scale for predicting pressure sore risk *(external link)*

# Evidence scores

1. Risk-assessment scales: there is medium-quality evidence that using assessment scales to decide on preventive measures led to ineffective and inefficient treatment for most patients. The predictive values of 4 pressure ulcer assessment scales have been evaluated: Norton, Waterlow, Braden,[34] and Ramstadius[33].
**Evidence level B:** Randomized controlled trials (RCTs) of <200 participants, methodologically flawed RCTs of >200 participants, methodologically flawed systematic reviews (SRs) or good quality observational (cohort) studies.

2. Debridement techniques: a systematic review of debridement techniques reported insufficient evidence on which to compare different methods.[67]
**Evidence level C:** Poor quality observational (cohort) studies or methodologically flawed randomized controlled trials (RCTs) of <200 participants.

3. Wound-healing rates: it is not known whether hydrocolloid dressings are more effective at improving ulcer healing than gauze dressings soaked in hypochlorite or povidone iodine.
**Evidence level C:** Poor quality observational (cohort) studies or methodologically flawed randomized controlled trials (RCTs) of <200 participants.

4. Clinical benefit of negative pressure therapy on pressure ulcers: systematic reviews of vacuum or topical negative pressure therapy concluded that there was insufficient evidence to demonstrate clinical benefit, and the large number of prematurely terminated and unpublished trials of vacuum therapy is of concern.[76] [77] [78]
**Evidence level C:** Poor quality observational (cohort) studies or methodologically flawed randomized controlled trials (RCTs) of <200 participants.

5. Clinical benefit of hyperbaric oxygen therapy on pressure ulcers: systematic reviews could not conclude whether hyperbaric oxygen therapy had any benefit on pressure ulcers.[81] [82]
**Evidence level C:** Poor quality observational (cohort) studies or methodologically flawed randomized controlled trials (RCTs) of <200 participants.

6. Foam versus standard mattresses: there is poor-quality evidence that foam alternatives to the standard hospital mattress reduced the incidence of pressure ulcers in people at high risk.
**Evidence level C:** Poor quality observational (cohort) studies or methodologically flawed randomized controlled trials (RCTs) of <200 participants.

7. Wound-healing rates: there is poor-quality evidence that, compared with standard care (regular changes of position, alternating pressure mattresses, sheepskin, gel pads, or limb protector), air-fluidised supports increased healing rates at 15 days in pressure ulcers.

**Evidence level C:** Poor quality observational (cohort) studies or methodologically flawed randomized controlled trials (RCTs) of <200 participants.

REFERENCES

## Key articles


- European Pressure Ulcer Advisory Panel; National Pressure Ulcer Advisory Panel. Prevention and treatment of pressure ulcers. Washington DC: National Pressure Ulcer Advisory Panel; 2014. http://www.npuap.org/ (last accessed 31 March 2017). Full text
- Qaseem A, Humphrey LL, Forciea MA, et al; Clinical Guidelines Committee of the American College of Physicians. Treatment of pressure ulcers: a clinical practice guideline from the American College of Physicians. Ann Intern Med. 2015;162:370-379. Full text Abstract


## References


1. European Pressure Ulcer Advisory Panel; National Pressure Ulcer Advisory Panel. Prevention and treatment of pressure ulcers. Washington DC: National Pressure Ulcer Advisory Panel; 2014. http://www.npuap.org/ (last accessed 31 March 2017). Full text
2. Kaltenhaler E, Whitfield MD, Walters SJ, et al. UK, USA, and Canada: how do their pressure ulcer prevalence and incidence data compare? J Wound Care 2001;10:530–535. Abstract
3. Margolis DJ, Bilker W, Knauss J, et al. The incidence and prevalence of pressure ulcers among elderly patients in general medical practice. Ann Epidemiol. 2002;12:321-325. Abstract
4. Pieper, M, ed. Pressure ulcers: prevalence, incidence, and implications for the future. Washington, DC: National Pressure Ulcer Advisory Panel; 2013.
5. Whittington KT, Briones R. National prevalence and incidence study: 6-year sequential acute care data. Adv Skin Wound Care. 2004;17:490-494. Abstract
6. Willock J, Baharestani MM, Anthony D. The development of the Glamorgan paediatric pressure ulcer risk assessment scale. J Child Young Peoples Nurs. 2007;1:211-218.
7. Raghavan P, Raza WA, Ahmed YS, et al. Prevalence of pressure sores in a community sample of spinal injury patients. Clin Rehabil. 2003;17:879-884. Abstract
8. Defloor T, De Bacquer D, Grypdonck MH. The effect of various combinations of turning and pressure reducing devices on the incidence of pressure ulcers. Int J Nurs Stud. 2005;42:37-46. Abstract
9. Berlowitz DR, Brienza DM. Are all pressure ulcers the result of deep tissue injury? A review of the literature. Ostomy Wound Manage. 2007;53:34-38. Abstract
10. Cooney LM Jr. Pressure sores and urinary incontinence. J Am Geriatr Soc. 1997;45:1278-1279. Abstract
11. Bliss M, Simini B. When are the seeds of postoperative pressure sores sown? Often during surgery. BMJ. 1999;319:863-864. Full text Abstract

12. Sibbald RG, Krasner DL, Lutz J. SCALE: skin changes at life's end: final consensus statement: October 1, 2009. Adv Skin Wound Care. 2010;23:225-236. Abstract

13. Scales JT. Pathogenesis of pressure sores. In: Bader DL, ed. Pressure sores: clinical practice and scientific approach. London: The Macmillan Press; 1990:15-25.

14. Allman RM. Pressure ulcers among the elderly. N Engl J Med. 1989;320:850-853. Abstract

15. Reddy NP. Effects of mechanical stresses on lymph and interstitial fluid flows. In: Bader DL, ed. Pressure sores: clinical practice and scientific approach. London: The Macmillan Press; 1990:1203-1220.

16. Coleman S, Nixon J, Keen J, et al. A new pressure ulcer conceptual framework. J Adv Nurs. 2014;70:2222-2234. Full text Abstract

17. Houwing R, Rozendaal M, Wouters-Wesseling W, et al. Pressure ulcer risk in hip fracture patients. Acta Orthop Scand. 2004;75:390-393. Abstract

18. Boyle M, Green M. Pressure sores in intensive care: defining their incidence and associated factors and assessing the utility of two pressure sore risk assessment tools. Aust Crit Care. 2001;14:24-30. Abstract

19. Hengstermann S, Fischer A, Steinhagen-Thiessen E, et al. Nutrition status and pressure ulcer: what we need for nutrition screening. JPEN J Parenter Enteral Nutr. 2007;31:288-294. Abstract

20. Berlowitz DR, Brandeis GH, Morris JN, et al. Deriving a risk-adjustment model for pressure ulcer development using the Minimum Data Set. J Am Geriatr Soc. 2001;49:866-871. Abstract

21. Krause T, Anders J, von Renteln-Kruse W. Urinary incontinence as a risk factor for pressure sores does not withstand a critical examination. Pflege. 2005;18:299-303. Abstract

22. Allman RM, Laprade CA, Noel LB, et al. Pressure sores among hospitalized patients. Ann Intern Med. 1986;105:337-342. Abstract

23. Padula WV, Mishra MK, Makic MB, et al. Improving the quality of pressure ulcer care with prevention: a cost-effectiveness analysis. Med Care. 2011;49:385-392. Abstract

24. Chou R, Dana T, Bougatsos C, et al. Pressure ulcer risk assessment and prevention: a systematic comparative effectiveness review. Ann Intern Med. 2013;159:28-38. Full text Abstract

25. Gould L, Stuntz M, Giovannelli M, et al.Wound Healing Society 2015 update on guidelines for pressure ulcers. Wound Repair Regen. 2016;24:145-162. Full text Abstract

26. Anthony D, Parboteeah S, Saleh M, et al. Norton, Waterlow and Braden scores: a review of the literature and a comparison between the scores and clinical judgement. J Clin Nurs. 2008;17:646-653. Abstract

27. Reddy M, Gill SS, Rochon PA. Preventing pressure ulcers: a systematic review. JAMA. 2006;296:974-984. Abstract

REFERENCES


28. Moore ZE, Cowman S. Risk assessment tools for the prevention of pressure ulcers. Cochrane Database Syst Rev. 2014;(2):CD006471. Full text Abstract

29. Norton D, McLaren R, Exton-Smith AN. Pressure sores: an investigation of geriatric nursing problems in hospital. New York, NY: Churchill Livingstone; 1975.

30. Waterlow J. Pressure sores: a risk assessment card. Nurs Times. 1985;81:49-55. Abstract

31. Braden B, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. Rehabil Nurs. 1987;12:8-12. Abstract

32. Xie H, Peel NM, Hirdes JP, et al. Validation of the interRAI Pressure Ulcer Risk Scale in acute care hospitals. J Am Geriatr Soc. 2016;64:1324-1328. Abstract

33. Webster J, Coleman K, Mudge A, et al. Pressure ulcers: effectiveness of risk-assessment tools. A randomised controlled trial (the ULCER trial). BMJ Qual Saf. 2011;20:297-306. Abstract

34. Schoonhoven L, Haalboom JR, Bousema MT, et al. Prospective cohort study of routine use of risk assessment scales for prediction of pressure ulcers. BMJ. 2002;325:797. Full text Abstract

35. Pancorbo-Hidalgo PL, Garcia-Fernandez FP, Lopez-Medina IM, et al. Risk assessment scales for pressure ulcer prevention: a systematic review. J Adv Nurs. 2006;54:94-110. Abstract

36. Moore Z, Cowman S, Conroy RM. A randomised controlled clinical trial of repositioning, using the 30° tilt, for the prevention of pressure ulcers. J Clin Nurs. 2011;20:2633-2644. Full text Abstract

37. Bergstrom N, Horn SD, Rapp MP, et al. Turning for Ulcer ReductioN: a multisite randomized clinical trial in nursing homes. J Am Geriatr Soc. 2013;61:1705-1713. Full text Abstract

38. Yap TL, Kennerly SM, Simmons MR, et al. Multidimensional team-based intervention using musical cues to reduce odds of facility-acquired pressure ulcers in long-term care: a paired randomized intervention study. J Am Geriatr Soc. 2013;61:1552-1559. Abstract

39. McInnes E, Jammali-Blasi A, Bell-Syer SE, et al. Support surfaces for pressure ulcer prevention. Cochrane Database Syst Rev. 2015;(9):CD001735. Full text Abstract

40. Nixon J, Cranny G, Iglesias C, et al. Randomised, controlled trial of alternating pressure mattresses compared with alternating pressure overlays for the prevention of pressure ulcers: PRESSURE (pressure relieving support surfaces) trial. BMJ. 2006;332:1413. Full text Abstract

41. Brienza D, Kelsey S, Karg P, et al. A randomized clinical trial on preventing pressure ulcers with wheelchair seat cushions. J Am Geriatr Soc. 2010;58:2308-2314. Abstract

42. Donnelly J, Winder J, Kernohan WG, et al. An RCT to determine the effect of a heel elevation device in pressure ulcer prevention post-hip fracture. J Wound Care. 2011;20:309-312,314-318. Abstract

43. Walia GS, Wong AL, Lo AY, et al. Efficacy of monitoring devices in support of prevention of pressure injuries: systematic review and meta-analysis. Adv Skin Wound Care. 2016;29:567-574. Full text Abstract

44. Langer G, Fink A. Nutritional interventions for preventing and treating pressure ulcers. Cochrane Database Syst Rev. 2014;(6):CD003216. Full text Abstract

45. Sullivan N, Schoelles KM. Preventing in-facility pressure ulcers as a patient safety strategy: a systematic review. Ann Intern Med. 2013;158:410-416. Full text Abstract

46. Edwards L, Banks V. Pressure sore classification grading systems. J Community Nurs. 1999;13:28-35.

47. European Pressure Ulcer Advisory Panel (EPUAP). Guide to pressure ulcer grading. 2001. http://www.epuap.org/ (last accessed 31 March 2017). Full text

48. National Pressure Ulcer Advisory Panel. NPUAP pressure injury stages. April 2016. http://www.npuap.org/ (last accessed 30 March 2017). Full text

49. Grayson ML, Gibbons GW, Balogh K, et al. Probing to bone in infected pedal ulcers. A clinical sign of underlying osteomyelitis in diabetic patients. JAMA. 1995;273:721-723. Abstract

50. Dow G. Bacterial swabs and the chronic wound: when, how, and what do they mean? Ostomy Wound Manage. 2003;49:8-13. Abstract

51. Reddy M, Gill SS, Kalkar SR, et al. Treatment of pressure ulcers: a systematic review. JAMA. 2008;300:2647-2662. Full text Abstract

52. Smith ME, Totten A, Hickam DH, et al. Pressure ulcer treatment strategies: a systematic comparative effectiveness review. Ann Intern Med. 2013;159:39-50. Full text Abstract

53. Vanderwee K, Grypdonck MH, De Bacquer D, et al. Effectiveness of turning with unequal time intervals on the incidence of pressure ulcer lesions. J Adv Nurs. 2007;57:59-68. Abstract

54. Moore ZE, Cowman S. Repositioning for treating pressure ulcers. Cochrane Database Syst Rev. 2015;(1):CD006898. Full text Abstract

55. Qaseem A, Humphrey LL, Forciea MA, et al; Clinical Guidelines Committee of the American College of Physicians. Treatment of pressure ulcers: a clinical practice guideline from the American College of Physicians. Ann Intern Med. 2015;162:370-379. Full text Abstract

56. Cereda E, Klersy C, Serioli M, et al; OligoElement Sore Trial Study Group. A nutritional formula enriched with arginine, zinc, and antioxidants for the healing of pressure ulcers: a randomized trial. Ann Intern Med. 2015;162:167-174. Abstract

57. Bauman WA, Spungen AM, Collins JF, et al. The effect of oxandrolone on the healing of chronic pressure ulcers in persons with spinal cord injury: a randomized trial. Ann Intern Med. 2013;158:718-726. Abstract

58. Pieper B, Langemo D, Cuddigan J. Pressure ulcer pain: a systematic literature review and national pressure ulcer advisory panel white paper. Ostomy Wound Manage. 2009;55:16-31. Abstract

59. Arapaglou V, Katsenis K, Syrigos KN, et al. Analgesic efficacy of an ibuprofen-releasing foam dressing compared with local best practices for painful exuding wounds. J Wound Care. 2011;20:319-325. Abstract

60. Zeppetella G, Paul J, Ribeiro MD. Analgesic efficacy of morphine applied topically to painful ulcers. J Pain Symptom Manage. 2003;25:555-558. Abstract

61. Sibbald RG, Orsted HL, Coutts PM, et al. Best practice recommendations for preparing the wound bed: update 2006. Adv Skin Wound Care. 2007;20:390-405. Abstract

62. Leaper D. Sharp technique for wound debridement. December 2002. http://www.worldwidewounds.com/ (last accessed 31 March 2017). Full text

63. Vowden K, Vowden P. Wound bed preparation. March 2002. http://www.worldwidewounds.com/ (last accessed 31 March 2017). Full text

64. Sherman RA, Wyle F, Vulpe M. Maggot therapy for treating pressure ulcers in spinal cord injury patients. J Spinal Cord Med. 1995;18:71-74. Abstract

65. Gray M. Is larval (maggot) debridement effective for removal of necrotic tissue from chronic wounds? J Wound Ostomy Continence Nurs. 2008;35:378-384. Abstract

66. Gurunluoglu R. Experiences with waterjet hydrosurgery system in wound debridement. World J Emerg Surg. 2007;2:10. Full text Abstract

67. Bradley M, Cullum N, Sheldon T. The debridement of chronic wounds: a systematic review. Health Technol Assess. 1999;3:iii-iv,1-78. Abstract

68. Jull AB, Cullum N, Dumville JC, et al. Honey as a topical treatment for wounds. Cochrane Database Syst Rev. 2015;(3):CD005083. Full text Abstract

69. Moore ZE, Cowman S. Wound cleansing for pressure ulcers. Cochrane Database Syst Rev. 2013;(3):CD004983. Full text Abstract

70. Bradley M, Cullum N, Nelson EA, et al. Systematic reviews of wound care management: (2). Dressings and topical agents used in the healing of chronic wounds. Health Technol Assess. 1999;3:1-35. Abstract

71. Bouza C, Saz Z, Munoz A, et al. Efficacy of advanced dressings in the treatment of pressure ulcers: a systematic review. J Wound Care. 2005;14:193-199. Abstract

72. Thomas S. A structured approach to the selection of dressings. July 1997. http://www.worldwidewounds.com/ (last accessed 31 March 2017). Full text

73. Norman G, Dumville JC, Moore ZE, et al. Antibiotics and antiseptics for pressure ulcers. Cochrane Database Syst Rev. 2016;(4):CD011586. Full text Abstract

74. Ebright JR. Microbiology of chronic leg and pressure ulcers: clinical significance and implications for treatment. Nurs Clin North Am. 2005;40:207-216. Abstract

75. Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. Ann Plast Surg. 1997;38:563-576. Abstract

76. Gregor S, Maegele M, Sauerland S, et al. Negative pressure wound therapy: a vacuum of evidence? Arch Surg. 2008;143:189-196. Full text Abstract

77. Ubbink DT, Westerbos SJ, Nelson EA, et al. A systematic review of topical negative pressure therapy for acute and chronic wounds. Br J Surg. 2008;95:685-692. Abstract

78. Van Den Boogaard M, De Laat E, Spauwen P, et al. The effectiveness of topical negative pressure in the treatment of pressure ulcers: a literature review. Eur J Plast Surg. 2008;31:1-7.

79. Desai KK, Hahn E, Pulikkottil B, et al. Negative pressure wound therapy: an algorithm. Clin Plast Surg. 2012;39:311-324. Abstract

80. Dumville JC, Webster J, Evans D, et al. Negative pressure wound therapy for treating pressure ulcers. Cochrane Database Syst Rev. 2015;(5):CD011334. Full text Abstract

81. Kranke P, Bennett M, Martyn-St James M, et al. Hyperbaric oxygen therapy for chronic wounds. Cochrane Database Syst Rev. 2015;(6):CD004123. Full text Abstract

82. Roeckl-Wiedmann I, Bennett M, Kranke P. Systematic review of hyperbaric oxygen in the management of chronic wounds. Br J Surg. 2005;92:24-32. Abstract

83. Cullum N, Nelson EA, Flemming K, et al. Systematic reviews of wound care management: (5) beds; (6) compression; (7) laser therapy, therapeutic ultrasound, electrotherapy and electromagnetic therapy. Health Technol Assess. 2001;5:1-221. Abstract

84. Aziz Z, Bell-Syer SE. Electromagnetic therapy for treating pressure ulcers. Cochrane Database Syst Rev. 2015;(9):CD002930. Full text Abstract

85. National Institute for Health and Care Excellence. Pressure ulcers: prevention and management of pressure ulcers. April 2014. http://www.nice.org.uk/ (last accessed 31 March 2017). Full text

86. Falanga V. Classifications for wound bed preparation and stimulation of chronic wounds. Wound Repair Regen. 2000;8:347-352. Abstract

87. Moore Z, Cowman S. A systematic review of wound cleansing for pressure ulcers. J Clin Nurs. 2008;17:1963-1972. Abstract

88. Stratton RJ, Ek AC, Engfer M, et al. Enteral nutritional support in prevention and treatment of pressure ulcers: a systematic review and meta-analysis. Ageing Res Rev. 2005;4:422-450. Abstract

89. Posner BM, Jette AM, Smith KW, et al. Nutrition and health risks in the elderly: the nutrition screening initiative. Am J Public Health. 1993;83:972-978. Full text Abstract

90. Hu X, Sun H, Han C, et al. Topically applied rhGM-CSF for the wound healing: a systematic review. Burns. 2011;37:729-741. Abstract


 

REFERENCES

91. Hao XY, Li HL, Su H, et al. Topical phenytoin for treating pressure ulcers. Cochrane Database Syst Rev. 2017;(2):CD008251. Full text Abstract

92. Russo CA, Steiner C, Spector W. Hospitalizations related to pressure ulcers among adults 18 years and older, 2006: Statistical Brief #64. Healthcare Cost and Utilization Project (HCUP) Statistical Briefs [Internet]. Rockville (MD): Agency for Health Care Policy and Research (US); 2006-2008. Full text Abstract

# Disclaimer

This content is meant for medical professionals situated outside of the United States and Canada. The BMJ Publishing Group Ltd ("BMJ Group") tries to ensure that the information provided is accurate and up-to-date, but we do not warrant that it is nor do our licensors who supply certain content linked to or otherwise accessible from our content. The BMJ Group does not advocate or endorse the use of any drug or therapy contained within nor does it diagnose patients. Medical professionals should use their own professional judgement in using this information and caring for their patients and the information herein should not be considered a substitute for that.

This information is not intended to cover all possible diagnosis methods, treatments, follow up, drugs and any contraindications or side effects. In addition such standards and practices in medicine change as new data become available, and you should consult a variety of sources. We strongly recommend that users independently verify specified diagnosis, treatments and follow up and ensure it is appropriate for your patient within your region. In addition, with respect to prescription medication, you are advised to check the product information sheet accompanying each drug to verify conditions of use and identify any changes in dosage schedule or contraindications, particularly if the agent to be administered is new, infrequently used, or has a narrow therapeutic range. You must always check that drugs referenced are licensed for the specified use and at the specified doses in your region. This information is provided on an "as is" basis and to the fullest extent permitted by law the BMJ Group and its licensors assume no responsibility for any aspect of healthcare administered with the aid of this information or any other use of this information.

View our full Website Terms and Conditions.



 

# Contributors:

## // Authors:

**Dan R. Berlowitz, MD**

Professor of Health Policy and Management
Boston University School of Public Health, Director of the Center for Health Quality, Outcomes, and Economic Research, Boston, MA
DISCLOSURES: DRB declares that he has no competing interests.

## // Acknowledgements:

Dr Dan R. Berlowitz would like to gratefully acknowledge Dr Madhuri Reddy and Dr Stephen Thomas, the previous contributors to this monograph. MR is the author of a number of references cited in this monograph. ST declares that he has no competing interests.

## // Peer Reviewers:

**Jane Deng, MD**

Assistant Professor of Medicine
David Geffen School of Medicine at UCLA, Los Angeles, CA
DISCLOSURES: JD declares that she has no competing interests.

**Keith Harding, FRCGP, FRCP, FRCS**

Sub Dean of Innovation & Engagement
Head of Section of Wound Healing, School of Medicine, Cardiff University, Cardiff, UK
DISCLOSURES: KH has worked in the area of wound healing for many years and has helped establish and sustain a section of wound healing in a university medical school. The school is entirely self-funded and receives funding from a wide range of commercial concerns in addition to the NHS and grant-giving bodies. The funding is provided to the University rather than to KH personally, and this funding is used to provide sustainability for a wide range of individuals employed within this section.