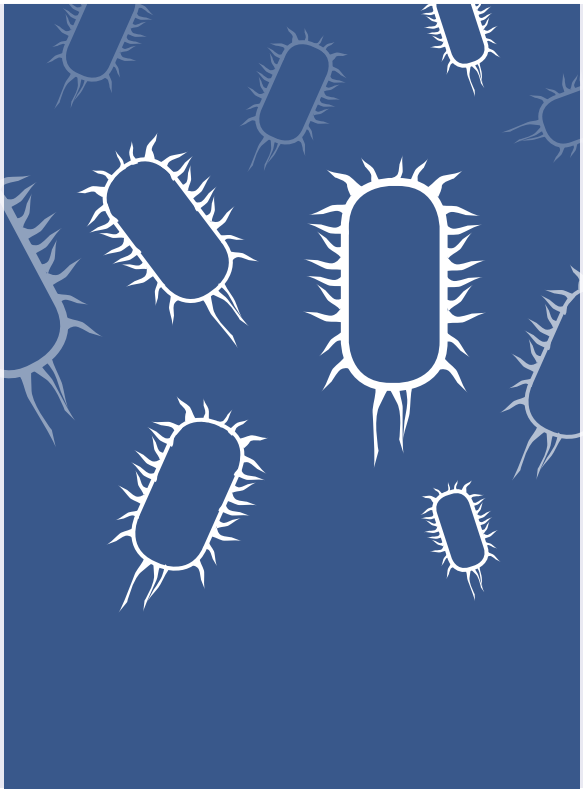
WUK BPS

Best Practice Statement

Making day-to-day management of biofilm simple

2017



Wounds UK

BEST PRACTICE STATEMENT: MAKING DAY-TO-DAY MANAGEMENT OF BIOFILM SIMPLE

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Wounds UK
A division of Omniamed,
1.01 Cargo Works
1–2 Hatfields, London SE1 9PG, UK
Tel: +44 (0)20 7627 1510
Web: www.wounds-uk.com

Wounds UK

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EXPERT WORKING GROUP:
Chair: **Jacqui Fletcher**, Independent Consultant

Leanne Atkin, Lecturer practitioner/Vascular Nurse Specialist, School of Human and Health Sciences, University of Huddersfield and Mid Yorkshire NHS Trust

Simon Barrett, Lead Tissue Viability Nurse Specialist, Humber NHS Foundation Trust

Paul Chadwick, Consultant Podiatrist, Salford Royal NHS Foundation Trust

Brenda King, Nurse Consultant Tissue Viability, Sheffield Teaching Hospitals NHS Foundation Trust

Richard Shorney, Managing Director, Real Healthcare Solutions Ltd, Cheshire

Kathryn Vowden, Lecturer, University of Bradford; Honorary Nurse Consultant, Bradford Teaching Hospitals NHS Foundation Trust

REVIEW PANEL:
Valerie Edwards-Jones, Emeritus Professor of Medical Microbiology, School of Healthcare Science, Manchester Metropolitan University

Martin Kiernan, Richard Wells Research Centre, University of West London

Andrew Kingsley, Formerly Lead Nurse for Healthcare Associated Infections, Northern, Eastern and Western Devon Clinical Commissioning Group

Making day-to-day management of biofilm simple

CONTENTS

GUIDE TO USING THIS DOCUMENT

This document will aid healthcare practitioners' day-to-day practice by providing an easy-to-follow guide to the principles of treating biofilm within the context of holistic wound care. The content is based on the discussions and conclusions of a group of wound care experts who met in January 2017. The document was finalised after extensive review by the initial expert group and by a panel of additional reviewers.

- The flowchart in Section 1 of the document provides an overview of how biofilm treatment fits in to the management of wounds. The flowchart is linked to a series of Best Practice Statements and Knowledge and Skills Self-assessments that are found in Sections 2 and 3
- Each Best Practice Statement (BPS) in Section 2 is supported by a boxed overview of the reasoning behind the statement, which is followed by a more detailed explanation of the rationale and evidence. Some practitioners may find that for some Best Practice Statements the overview is sufficient for their needs, only referring to the more detailed information when more in-depth knowledge is required
- The Knowledge and Skills Self-assessments in Section 3 help clinicians to identify whether they have the appropriate knowledge and skills to undertake wound assessment and management and to recognise when escalating assessment or management is appropriate. The self-assessments will also encourage individuals to identify areas for further development and to seek appropriate training.
- The structure and life cycle of biofilms is described in Appendix 1 (page 30)

RECOMMENDED RESOURCES

- Biofilms: the Myths and Realities. (Mahoney K (2015) Wound Essentials 10(2): 38–42*
- Biofilms Made Easy. Phillips PL, Wolcott RD, Fletcher J, Schultz GS (2010) Wounds International 1(3)**
- Management of biofilm. World Union of Wound Healing Societies. Wounds International, 2016**
- Effective Debridement in a Changing NHS: A UK Consensus (2013)*
- TIMES model of wound bed preparation Quick Guide (2017) Wounds UK 13(1)*

*Available from: www.wounds-uk.com;
**Available from: www.woundsinternational.com

Making day-to-day management of biofilm simple

Biofilms are communities of microbes that attach to and grow on surfaces. A well known example is tooth plaque. However, biofilms also grow in chronic wounds and, in some cases, may be the cause of delayed healing.

This document is for everyone involved in the treatment and management of wounds that are slow to heal (chronic wounds) who would like to:

- Help these wounds to heal more quickly, and so:
  - Improve patients' lives
  - Reduce wound care costs by reducing overall treatment time, and reducing the number of dressing changes
- Meet national standards for wound care.

Wounds that are slow to heal are a large and growing problem in the UK. These include venous leg ulcers, pressure ulcers, diabetic foot ulcers, post-operative wounds, arterial ulcers and burns (Box 1). An ageing population and increased incidence of diabetes and obesity are compounding the issue.

Problems caused by delayed wound healing

Wounds that are slow to heal are costly – in financial and human terms. Contact time with clinicians, including appointments for dressing changes, makes up a large part of the financial cost. Interestingly, the dressings themselves form a relatively small proportion of the total cost of treating wounds (Box 1).

The impact on patients can be severe: wounds that are slow to heal can be painful and impair physical, mental and social wellbeing. Treatment is time consuming and may be uncomfortable.

Causes of delayed wound healing

The wide range of factors that can delay the healing of a wound can be put into three groups:

- **Underlying cause** – has not been treated or removed, e.g. a patient with a venous leg ulcer is not being treated with appropriate compression therapy
- **New factor** – is causing healing problems and delaying healing, e.g. the wound has become infected or the patient is receiving a treatment that is interfering with healing (such as treatment for cancer)
- **Treatment issue** – the treatment applied to the wound is delaying or stalling healing, e.g. the dressing is not absorbent enough and the excess wound fluid is causing damage to the wound bed and deterioration of the adjacent skin.

Box 1: Estimated UK incidence and NHS costs of wounds (Guest et al, 2016; Guest et al, 2015))

- Annual occurrence of wounds: 2.2 million wounds*
- Annual total cost of managing these wounds: £4.5-5.1bn**
- Annual cost of wound care products: £742.7m (about 14.5% of the total cost of wound care)**
- About 61% of wounds healed within the year of the study and 39% (0.9 million) remained unhealed
- Unhealed wounds required 20% more practice nurse visits, 104% more community nurse visits, 13% more GP visits, 18% more hospital outpatient visits and 40% more drug prescriptions than healed wounds
- The mean cost of an unhealed wound was about 2.5 times more than that of a healed wound

*Excludes patients with a surgical wound healed within four weeks and patients with a dermatological tumour. Numbers of wounds were derived from a model that used patient data spanning May 1, 2012 to April 30, 2013 and were applied to an estimated UK population of 63.7 million people.

**Costs were based on figures for 2013-2014.

Biofilm delays healing

Evidence is accumulating of the role biofilm plays in hard-to-heal wounds. Biofilm is formed by microbes (mainly bacteria) that are firmly embedded in the wound and encapsulated in a matrix, which contains host material, making both dispersal and treatment problematic (Appendix 1, page 30).

Biofilm in chronic wounds is usually a mixture of different microbial species (some may be pathogenic (capable of causing infection) and some may be non-pathogenic (not capable of causing infection). In a biofilm the microbes can work together and they are capable of forming a stable environment that can continue *ad infinitum* unless disrupted in some way.

The microbes in a biofilm are protected from the patient's immune system and antimicrobial agents, such as antiseptics and antibiotics, in two ways:

- By the barrier formed by the protective coating
- By becoming inactive or 'going to sleep' (bacteria need to be active and 'awake' to be susceptible to antimicrobials).

Biofilm is thought to delay wound healing by upsetting healing processes, causing additional wound damage and acting as a source of infection.

Treatment of biofilm

Currently, there are no easy tests to detect biofilm in a wound, and no tests to show when a wound biofilm is causing a problem. But, we do know it is likely that all wounds that are slow to heal contain biofilm (see BPS 6, pages 16–19).

So, if the patient has received appropriate management for a chronic wound, its cause and any contributory factors, but the wound is slow to heal, it is logical to suspect that biofilm is causing healing problems.

Reducing the amount of biofilm in a chronic wound may tip the balance in favour of healing. If biofilm is suspected of delaying healing of a chronic wound, it should be treated proactively by:

- Repeatedly breaking up and removing the biofilm — through vigorous/active cleansing and/or debridement
- Reducing biofilm reformation — by decreasing the number of bacteria left in the wound through the use of an antimicrobial dressing or topical antiseptic preparation left in place between each session of biofilm removal (Figure 1); (Box 6, page 12, describes these types of treatment).

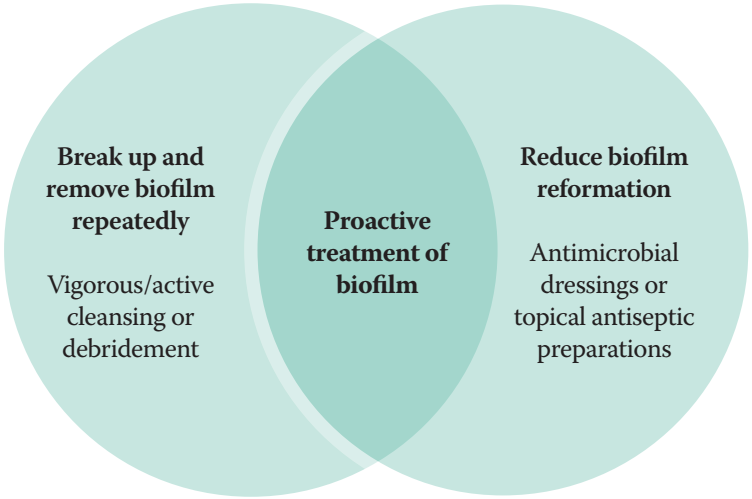
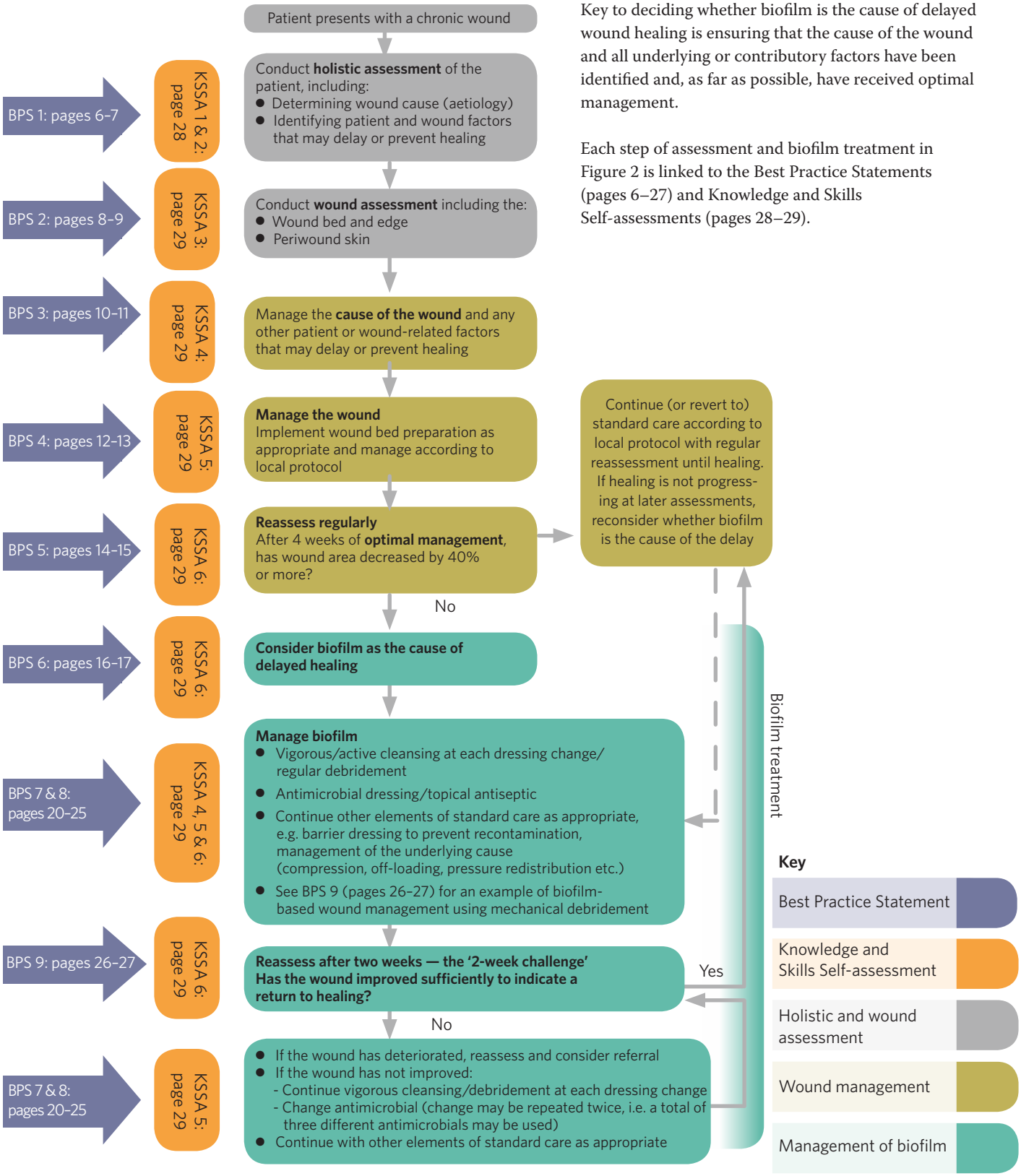


Figure 1: Proactive treatment of a chronic wound in which delayed healing is possibly due to biofilm

SECTION 1: TREATMENT OF BIOFILM IN THE MANAGEMENT OF WOUNDS

Key to deciding whether biofilm is the cause of delayed wound healing is ensuring that the cause of the wound and all underlying or contributory factors have been identified and, as far as possible, have received optimal management.

Each step of assessment and biofilm treatment in Figure 2 is linked to the Best Practice Statements (pages 6–27) and Knowledge and Skills Self-assessments (pages 28–29).



SECTION 2: BEST PRACTICE STATEMENTS — MANAGEMENT OF BIOFILM

A recent study of a large UK patient database revealed that 41.8% of wounds being treated within the NHS lacked a differential diagnosis (Guest et al, 2016), suggesting that for at least some of these wounds the underlying cause was not being managed.

In addition, contrary to guidelines, only 16% of all patients with a leg or foot ulcer had an ankle brachial pressure index (ABPI) to assess peripheral arterial circulation (Guest et al, 2016).

The Best Practice Statements produced by the Expert Working Group on the management of biofilm (Table 1) highlight the importance of assessment and determining the underlying cause of the wound (diagnosis) and any other factors that may be delaying healing.

The Best Practice Statements also provide clear criteria for when the presence of biofilm should be suspected as causing delayed healing before explaining how to treat biofilm and to assess whether treatment is successful.

The assessment and treatment of all wounds should be carried out by a registered healthcare practitioner (see Knowledge and Skills Self-assessments 1 and 2, pages 24–25).

All of the Best Practice Statements in this document refer to activities that should only be carried out by healthcare professionals.

All activities carried out by healthcare professionals should be documented and reported in line with local protocol.

Table 1. Best Practice Statements on the management of wounds and biofilm

	Best Practice Statement	Pages	Corresponding Knowledge and Skills Self-assessment(s)	Page
1	On first contact with a registered healthcare professional, all patients with a wound should have a documented holistic assessment that includes identifying all factors that may be causing or eventually result in delayed healing	6–7	1. Assess the whole patient (holistic assessment) 2. Identify the underlying cause of the wound	28
2	On first contact with a registered healthcare professional and following holistic assessment, all patients with a wound should have a documented wound assessment	8–9	3. Assess the wound	28
3	Management of the underlying cause of the wound and all factors that may be contributing to delayed or failed wound healing should be managed or corrected where possible	10–11	4. Plan and implement management of the underlying cause of the wound	29
4	The wound should be cared for using the principles of wound bed preparation as appropriate and according to local protocol	12–13	5. Plan and implement treatment of the wound	29
5	All wounds should be assessed regularly for signs of progress (or deterioration)	14–15	6. Evaluate the response of the wound and the underlying cause	29
6	In the absence of overt wound infection, biofilm should be considered as the possible cause of delayed healing in all wounds that are failing to progress adequately after four weeks of optimal management	16–19	6. Evaluate the response of the wound and the underlying cause	29
7	Once biofilm is suspected to be the cause of delayed healing, proactive treatment should include strategies to physically disrupt and remove the existing biofilm, i.e. vigorous/active cleansing or debridement	20–23	4. Plan and implement management of the underlying cause of the wound 5. Plan and implement treatment of the wound	29
8	Following disruption of biofilm, treatment should include strategies to reduce microbial load by using an antimicrobial dressing or a topical antiseptic preparation for a two-week trial	24–25	6. Evaluate the response of the wound and the underlying cause	29
9	Wounds being treated for biofilm should be re-evaluated after two weeks of biofilm-based wound management. If the wound has not responded and biofilm is still suspected as the cause of delayed healing, consider a second or third round of treatment with a different antimicrobial dressing or topical antiseptic	26–27	6. Evaluate the response of the wound and the underlying cause	29

BEST PRACTICE STATEMENT 1

BPS 1. ALL PATIENTS WITH A WOUND SHOULD HAVE A DOCUMENTED, HOLISTIC ASSESSMENT THAT INCLUDES IDENTIFYING ALL FACTORS THAT MIGHT CAUSE OR RESULT IN DELAYED HEALING

Holistic assessment

Holistic assessment of a patient with a wound 'involves identifying, gathering and interpreting information about the patient and the wound to ensure accurate diagnosis, appropriate treatment, ongoing monitoring and prevention of complications' (Benbow, 2016).

It should involve the patient, family and caregivers (Dowsett et al, 2015). Strictly speaking, wound assessment is part of holistic assessment. In this document, however, wound assessment is covered separately (pages 11–13) to emphasise the importance of assessing other aspects of the patient.

The assessment should take a structured approach that includes ascertaining general health information such as:

- Comorbidities – including any factors affecting systemic and local blood supply, susceptibility to infection and skin integrity
- Current medication – including any medication that may affect wound healing
- General skin condition
- Nutritional status
- Previous investigations
- Previous surgery
- Allergies and sensitivities
- Psychosocial status – including the impact of the wound on the patient and concordance.

The information gathered may reveal the cause of the wound and/or factors that may contribute to delayed healing (Ousey & Cook, 2011).

Diagnosis and underlying cause

The medical history of the patient and location of the wound may indicate the likely cause of the wound (Grey et al, 2009). Further investigations may be needed to confirm the diagnosis and/or indicate the suitability of possible treatment options or need for referral. For example, a patient with a suspected venous leg ulcer may need a venous duplex scan to confirm the presence of chronic venous insufficiency.

As compression therapy is an important element of venous leg ulcer management, arterial vascular assessment, e.g. determining ABPI, is important to detect severe peripheral arterial disease which would contraindicate compression (Wounds UK, 2016).

Investigations, such as blood tests, may also be needed to identify or monitor comorbidities, e.g. full blood count to detect anaemia, or blood glucose and HbA1c to evaluate control of diabetes.

Factors that may contribute to delayed healing

In addition to identifying conditions and medication that may be delaying healing (Table 2), it is important to assess issues such as psychological state, concordance with treatment and living conditions.

Pain assessment

Assessment of pain, whether related to the wound or not, is often neglected even though pain and psychological stress can adversely affect wound healing (Flanagan, 2007; Woo, 2012; Gouin & Kiecolt-Glaser, 2011). Therefore, it is important to assess pain so that the cause of the pain can be determined and an appropriate management strategy can be put in place.

For further reading, see page 33.

Table 2. Factors that may contribute to delayed healing (Best Practice Statement, 2008; Guo & Dipietro, 2010; Ousey & Cook, 2012; Wounds UK, 2013; Benbow, 2016)

Factor	Examples	
Medical conditions or previous treatment	■ Diabetes ■ Venous disease, e.g. chronic venous insufficiency, deep venous thrombosis ■ Arterial disease, e.g. atherosclerosis, peripheral arterial disease ■ Immobility, paralysis or loss of sensation ■ Malnutrition ■ Advancing age ■ Immune disorders, e.g. rheumatoid arthritis ■ Cardiac failure	■ Liver disease ■ Kidney disease ■ Obesity ■ Surgery ■ Radiotherapy ■ Skin conditions ■ Anaemia ■ Systemic malignancy (cancer)
Current medication	■ Corticosteroids ■ Immunosuppressants ■ Non-steroidal anti-inflammatories ■ Anti-platelet medication and anti-coagulants	
Pain	■ Wound and non-wound related pain	
Psychosocial factors	■ Psychological status — stress, anxiety, depression ■ Poor living conditions and/or diet ■ Suboptimal concordance with treatment ■ Self-induced skin or wound damage	
Other factors	■ Advanced age ■ Smoking ■ Alcoholism ■ Debris or foreign body in the wound, e.g. hair	

KEY POINTS

1. The healing outcomes of wounds are improved by identifying and managing the cause of the wound and any factors that might delaying healing as early as possible
2. Holistic assessment should involve gathering information about the patient as a whole, as well as about the wound, which is then used to ensure the appropriate treatment is implemented
3. Assessment should include:
   - The patients' medical history including: previous and current conditions such as diabetes, current medication, general skin condition, nutritional status, previous investigations and surgery, allergies and sensitivities, and psychosocial status
   - The location of the wound – it may indicate the aetiology of the wound
   - Pain – both wound and non-wound related – including site, duration, nature and triggers and soothers of pain
4. Assessment may indicate the need for further tests or investigations to confirm the cause of the wound, identify or check the status of comorbidities (e.g. diabetes), or check suitability of treatments such as compression therapy (e.g. ABPI)
5. All assessments and subsequent updates, including confirmatory text results, should be recorded in line with local policy. If the aetiology of the wound cannot be confirmed at the time of documentation, a provisional (working) diagnosis should be recorded along with any plans for confirmatory investigations
6. For knowledge and skills needed to carry out an holistic assessment see Knowledge and Skills Self-assessments 1&2, page 28.

BEST PRACTICE STATEMENT 2

BPS 2. FOLLOWING HOLISTIC ASSESSMENT, THE WOUND SHOULD BE ASSESSED AND THE AETIOLOGY IDENTIFIED

Purposes of wound assessment

A thorough and systematic wound assessment will:

- Assess the condition of the wound and the skin around the wound
- Identify any local factors, such as infection, that are hindering healing
- The findings of the assessment will:
    1. Indicate whether further investigations are needed — e.g. X-rays if bone is visible or can be probed in the base of the wound or biopsy if malignancy is suspected
    2. Inform the treatment plan — e.g. indicate whether infection is present and appropriate dressing use
    3. Provide a baseline from which to monitor progress or detect deterioration.

The findings may also provide further evidence for the aetiology of the wound.

Frameworks that may aid systematic wound assessment are listed in Box 2. Table 3 (page 8) summarises aspects of the wound that should be assessed.

Photographs are a useful way of monitoring a wound (Sperring & Baker, 2014) and should be obtained and stored according to local policy and after obtaining patient consent.

Exudate levels

In general, the amount of exudate a wound produces decreases as healing progresses. A sudden increase in exudate may indicate infection (WUWHS, 2007).

There is no straightforward way of measuring how much exudate a wound is producing. The current dressing may provide guidance (WUWHS, 2007).

Wound measurement

Wound measurement provides an objective means of assessing healing progress. A wide variety of methods of measurement is available (Khoo & Jansen, 2016). The method used should be in line with local policy. It is important that the same method is used for successive wound measurements – irregularities in the results could lead to variations being attributed to changes in the wound. Measurement should take place regularly.

Signs of infection

In acute wounds the symptoms and signs of infection may be clear: pain, redness, warmth, swelling and purulent discharge may be present (WUWHS, 2007).

In chronic wounds, the signs of infection may be less clear (Box 3). There is overlap between the signs and symptoms of infection in chronic wounds and those thought to indicate that biofilm is delaying healing (Table 6, page 18–19). Pages 14–19 explain in more detail how to determine whether biofilm is the cause of delayed healing.

Infection can be confirmed by sampling the wound for microbiological analysis. This should be carried out in line with local protocols (Patten, 2010).

Wound swabbing is the most widely used sampling technique, but may provide misleading results as it may only sample superficial microbes and not collect deeper microbes that may be the cause of the problem. Other sampling techniques include needle aspiration and biopsy and may be used following consultation with the local laboratory.

Routine microbiology tests are not usually justified (Cooper, 2010) because of the delays to care and cost considerations. It is recommended that clinicians look for signs of infection (see Box 3) and act accordingly.

Box 2. Frameworks to aid systematic wound assessment

- **TIMES**: **T**issue, **I**nfection/ inflammation, **M**oisture imbalance, **E**dge of the wound, **S**urrounding skin (Wounds UK, 2016; Quick Guide, 2017)
- **Triangle of Wound Assessment**: wound bed, wound edge, periwound skin (Dowsett et al, 2015)
- **TIME**: **T**issue, **I**nfection/ inflammation, **M**oisture imbalance, **E**dge of the wound (Schultz et al, 2004).

Table 3. Wound assessment (Grey et al, 2006; WUWHS, 2007; Hess, 2011; Ousey & Cook, 2012; Nix, 2012; Dowsett et al, 2015; Benbow, 2016; Wounds UK, 2016)

Aspect to be assessed	Notes
Location	■ **May indicate aetiology** ■ May affect dressing choice ■ If over a joint, may be prone to movement that hinders healing
Size	■ Length, width, depth, area, volume ■ Check for undermining, tunnelling or fistulae
Wound bed	■ **May indicate stage of healing** ■ Examine for proportion of epithelial tissue, granulation tissue, necrotic tissue/eschar, slough ■ Necrotic tissue, eschar and slough can act as media for microbial growth
Exudate level	■ Amount — informs dressing selection and dressing change frequency: - high exudate levels may cause maceration/excoriation - low exudate levels may prevent cell migration across the wound bed ■ Colour: - Cloudy or green – possible bacterial infection - Pink or red – presence of blood - Yellow or brown – presence of wound slough - Grey or blue – may be related to the use of silver-containing dressings ■ Viscosity: - High (thick) – may be due to infection, inflammation, necrotic material, dressing residue - Low (thin) – may accompany a venous leg ulcer, congestive cardiac disease or malnutrition
Edges	■ Sloping — may indicate a venous leg ulcer ■ Punched out — may indicate an arterial wound ■ Raised, rolled or everted — may indicate chronicity or malignancy ■ Purple — may indicate a vasculitic wound (e.g. *pyoderma gangrenosum*)
Periwound skin	■ **May indicate aetiology and other pathology:** - Oedema, brawny (brown) discolouration, hyperkeratosis — venous leg ulcer - Pale, cool, hairless — arterial ulcer - Red, hot, swollen — infection ■ Maceration and excoriation — may indicate high exudate levels ■ General skin condition ■ Hygiene issues
Odour	■ Unpleasant odour may be due to bacterial growth or necrotic tissue ■ Some dressings produce a distinctive odour
Pain	■ Assess site, duration, type, severity (e.g. through numerical rating scales or visual analogue scales), factors that reduce or trigger the pain ■ New or suddenly worsening wound pain may indicate infection

Box 3. Signs of possible infection in chronic wounds (WUWHS, 2008)

- New, increased or altered pain
- Malodour or change in odour
- Increased or altered/purulent exudate
- Delayed healing
- Periwound oedema
- Bleeding or easily damaged (friable) granulation tissue
- Wound bed discolouration
- Induration (hardening of the skin and subcutaneous tissues, a sign of inflammation)
- Pocketing – smooth, non-granulating areas in the wound base surrounded by granulation tissue
- Bridging – incomplete epithelialisation resulting in strands or patches of tissue across the wound.

KEY POINTS

1. Assessment should include the location, size and depth, wound bed, wound edge, exudate level, periwound skin, odour and pain
2. The same method of measurement of wound size should be used to monitor changes
3. The most common signs of infection in a chronic wound are new or worsening pain, a sudden increase in exudate level or the production of purulent exudate, and malodour
4. Sampling for microbiological analysis, e.g. swabbing, should be carried out in line with local policy; routine swabbing should be avoided
5. For knowledge and skills needed to carry out a wound assessment, see Knowledge and Skills Self-assessment 3, page 29.

BEST PRACTICE STATEMENT 3

BPS 3. THE UNDERLYING CAUSE OF THE WOUND AND ALL FACTORS THAT MAY BE CONTRIBUTING TO DELAYED OR FAILED WOUND HEALING SHOULD BE MANAGED OR CORRECTED WHERE POSSIBLE

Management of wound cause and all other contributory factors

Holistic management of a patient with a wound should occur alongside wound management and often requires a multidisciplinary approach (Frykberg & Banks, 2015).

Further investigation and referral may be needed to assess the severity of the underlying cause and to indicate the most appropriate type(s) of treatment. For example, arterial imaging is needed to determine whether arterial insufficiency is of a type that is suitable for surgery or whether non-surgical treatment is the best option.

Management should aim to remove or ameliorate the:

- Underlying cause
- Any other patient- or wound-related factors that are hampering healing.

Management of the underlying cause

Correction or amelioration of the underlying cause may improve the chance of healing the wound and, importantly, will also reduce the risk of development of further wounds. Table 4 outlines approaches that may be used to manage the underlying cause of the main types of chronic wound.

Other patient-related factors

A wide range of other patient-related factors may delay healing, as listed in Table 2 (page 7). Not all of these factors are treatable, e.g. advanced age, but wherever treatment is possible, management should form part of the patient's overall treatment plan.

Where medication such as corticosteroids or immunosuppressants may be delaying healing, a careful assessment of the overall risks and benefits to the patient and the wound of adjusting or discontinuing treatment is needed.

The prescribing clinician/clinic/service should be involved before any changes to medication are made. When patients are on multiple medications,

Table 4. Treatment of the underlying cause of the main types of chronic wound (Grey et al, 2006; Agale, 2013)

Wound type	Main factor(s) to address	Treatment options
Venous leg ulcer	Venous insufficiency/hypertension	• Compression therapy • Surgery • Leg elevation
Arterial ulcer	Reduced arterial perfusion	• Revascularisation
Diabetic foot ulcer	Poor blood glucose control Pressure caused by callous or disturbed foot architecture Vascular insufficiency	• Review treatment for diabetes and improve blood glucose control • Reduce pressure through offloading • Maintenance debridement • Revascularisation
Pressure ulcer	Pressure, shear and friction	• Relieve pressure and reduce the risk of shear and friction

a review may be helpful in identifying potential drug interactions and medications that may be reduced or discontinued.

Where relevant, pain should be managed effectively, and mental health issues addressed to enhance psychological wellbeing. A creative approach may be needed if low concordance with any aspect of treatment is identified. Ascertaining as far as possible the reasons why the patient finds it difficult to concord may highlight ways to help.

"A creative approach may be needed if low concordance with any aspect of treatment is identified. Ascertaining as far as possible the reasons why the patient finds it difficult to concord may highlight ways to help."

KEY POINTS

1. Management of the underlying disease and any other factors that may be delaying healing requires a multidisciplinary approach, and possibly further investigations and referral
2. The aim is to remove or reduce the effects of the underlying cause of the wound or any other patient- or wound-related factors that are hampering healing
3. Treatment of the underlying cause should improve healing of the wound and help prevent further wounds
4. Other patient- and wound-related factors should be identified and included in the documented management plan
5. Decisions regarding management approach on issues identified, along with requested investigations and referrals, should be documented according to local policy
6. For knowledge and skills needed to identify and manage underlying cause, see Knowledge and Skills Self-assessment 4, page 29.

BPS 4. THE WOUND SHOULD BE CARED FOR USING THE PRINCIPLES OF WOUND BED PREPARATION AS APPROPRIATE AND IN LINE WITH LOCAL PROTOCOL

Wound bed preparation

The concept of wound bed preparation (WBP) was initially developed for non-healing chronic wounds, but has also been used successfully in burns wounds (Schultz & Dowsett, 2012). It has been defined as the management of a wound to:

- Accelerate healing
- Facilitate the effectiveness of other therapeutic measures (Dowsett & Newton, 2005).

Underpinning wound bed preparation is the systematic identification and management of any factors that may be delaying wound healing. As described in Box 2 (page 8), various frameworks have been devised to assist in the process of wound bed preparation.

Wound bed preparation may comprise:

- Debridement (Box 4) and cleansing (Box 5) – to remove non-viable tissue, slough or debris from the wound
- Management of infection and inflammation – to reduce wound bacterial load and level of inflammation
- Facilitation of moist wound healing – to aid cell migration and prevent problems caused by desiccation or excess moisture
- Consideration of the use of skin grafts or advanced biological agents — to aid re-epithelialisation (Schultz et al, 2003; Moore, 2012; Wolcott & Fletcher, 2014).

The elements of WBP included in the management plan for an individual should be selected according to the results of the assessment for that wound.

Debridement

The type of debridement used will depend on factors related to the wound, patient, practitioner and healthcare setting (Box 7, page 20), and should be carried out in line with local protocol. Depending on the method selected and the condition of the wound, debridement may need to be performed more than once (Dowsett & Newton, 2005).

The modes of action, advantages and disadvantages of the different types of debridement are reviewed in Table 7 (pages 22–23). There are several types of debridement, including:

- Autolytic: this method is slow to produce results, yet is the most commonly used approach that can be undertaken by HCPs without specialist skills (Atkin, 2014).
- Mechanical: this is a faster method that also does not require specialist skills. It is carried out using a monofilament fibre debridement pad (Debridement Consensus, 2013; NICE, 2014)
- Larval: this form of debridement is faster than autolytic debridement (Debridement Consensus, 2013) and involves the introduction of live, disinfected maggots onto the wound
- Sharp or surgical: this form of debridement requires specialist training and is particularly useful for hard eschar or for large areas (Debridement Consensus, 2013)
- Ultrasonic and hydrosurgical: these forms of debridement may be limited to wound clinics because of the need for specialist equipment and training (Debridement Consensus, 2013).

Cleansing

Cleansing (Box 5) should be carried out in line with local policy and with clear goals in mind. Ritualistic cleansing should be avoided as it may cause tissue damage (NATVNS Guidance; Atiyeh et al, 2009). Wounds that have a wound bed comprised of clean granulating or epithelialising tissue generally do not need to be cleansed (NATVNS Guidance).

Box 4. Definition of debridement (Debridement Consensus, 2013)

Debridement is the removal of dead, non-viable/devitalised tissue, and infected or foreign material from the wound bed and surrounding skin.

Box 5. Wound cleansing (Carr, 2006; NATVNS Guidance)

Wound cleansing is used to remove loose debris, which may include slough, necrotic tissue, excess exudate and wound dressing remnants, from the wound bed and periwound skin.

Box 6. Differentiating antimicrobial dressings, topical antiseptic preparations and irrigation/cleansing solutions

- Antimicrobial dressings: dressings that are impregnated with an antiseptic agent, e.g. silver, iodine, PHMB or octenidine
- Topical antiseptic preparation: gels containing an antiseptic agent, e.g. PHMB or octenidine that are left in the wound between dressing changes; usually requires the use of a suitable secondary dressing
- Irrigation/cleansing solutions: saline, potable tap water or solutions containing antiseptic agents, e.g. PHMB or octenidine; these are used at dressing change to flush out the wound before a suitable antimicrobial dressing or topical antiseptic preparation is applied.

Irrigation is generally considered the most appropriate method of wound cleansing. Unless the wound is infected or it is suspected that biofilm is delaying healing (see pages 16–25), tap water or sterile saline can be used. In wounds that are infected or being treated for biofilm, irrigation with a solution that has antimicrobial properties may be appropriate (Wolcott & Fletcher, 2014).

Cleansing may be necessary immediately before debridement to remove any loose material from the wound, and then again afterwards to wash out any remaining debris (Wolcott & Fletcher, 2014).

Management of infection and inflammation

Treatment of wound infection (see Box 3, page 9, for signs of infection in chronic wounds) should involve:

- Optimising the patient's immune response – e.g. by treating underlying conditions such as diabetes, optimising nutrition and hydration
- Reducing wound microbial load – through debridement, cleansing, and the use of antimicrobial dressings or topical antiseptic preparations (with an appropriate dressing)
- Reducing the risk of reinfection or further infections – e.g. through use of aseptic technique as appropriate and patient/caregiver education (IWII, 2016).

Debridement and cleansing play an important role in the treatment of infected wounds. Further reduction of microbial load can be achieved by using antiseptic wound cleansing agents, e.g. polyhexamethylene biguanide (PHMB) and octenidine dihydrochloride, and the application of antimicrobial dressings or topical antiseptic preparations (with an appropriate dressing) (IWII, 2016). Antiseptic agents commonly used in the treatment of wound infection include silver and iodine (Swanson et al, 2014).

The 2-week challenge

Expert opinion recommends that the effect of an antimicrobial dressing or topical antiseptic preparation (Box 6) is reviewed after 2 weeks (the '2-week challenge') (IWII, 2016). If after 2 weeks, the wound has improved but continues to show signs of infection, use of the current antimicrobial dressing or topical antiseptic preparation may be justifiable. If the wound has not improved, it should be reassessed and an antimicrobial dressing or topical antiseptic preparation containing a different antiseptic agent considered. If the wound has improved and there are no longer any signs of infection, the antimicrobial dressing or topical antiseptic preparation should be discontinued (International Consensus, 2012b).

Systemic antibiotics

Systemic antibiotics should not be used to treat localised wound infection — because of potential to induce antibiotic resistance — unless there are signs of spreading local infection, systemic infection (i.e. the patient is unwell) or associated osteomyelitis (IWII, 2016).

For management of a wound that has delayed or stalled healing that is suspected to be due to biofilm, see pages 16–19.

Facilitation of moist wound healing

A moist wound environment will aid healing of most open wounds. The moisture level needs to be controlled carefully: insufficient or excessive moisture levels in the wound bed can hinder healing by damaging the wound bed and surrounding skin (Sibbald et al, 2015). However, some wounds in specific circumstances should be kept dry to reduce the risk of systemic infection, e.g. dry necrotic wounds overlying poorly vascularised areas. The aim of this approach may be to allow demarcation of the wound so that tissue can be conserved during future surgery, or to enable auto-amputation of a necrotic toe.

The selection of an appropriate dressing and dressing change frequency are key to achieving optimal wound moisture levels (WUWHS, 2007; Sibbald et al, 2015).

KEY POINTS

1. WBP may comprise debridement, cleansing, treatment of infection, moist wound healing and consideration of skin grafts or advanced biological treatments
2. Debridement is central to WBP. It removes dead tissue, wound debris and biofilm (see pages 16–25) to help the wound to heal
3. There are numerous methods of debridement; the type used will depend on the needs of the wound and the skills of the HCP
4. Not all wounds need cleansing. If required, cleansing should be carried out with potable tap water or sterile saline
5. If a wound is infected, cleansers containing antiseptic agents and antimicrobial dressings or topical antiseptic agents may be used.
6. Unless wound infection is spreading or the patient is showing signs of being unwell, oral or intravenous antibiotics should not be used
7. For most wounds, the aim is to keep the wound bed moist, but not wet, by using dressings of appropriate absorption which are changed at suitable intervals
8. For knowledge and skills needed to carry out wound bed preparation, see Knowledge and Skills Self-assessment 5, page 29.

BEST PRACTICE STATEMENT 5

BPS 5. ALL WOUNDS SHOULD BE ASSESSED REGULARLY FOR SIGNS OF FAILURE TO PROGRESS

Reassessment

Wounds should be reassessed regularly to:

- Observe for signs of healing progress or signs of deterioration
- Assess the suitability of:
  - The current dressing type and dressing change frequency
  - Any other treatment modalities in use, e.g. offloading for diabetic foot ulcers, pressure redistribution for pressure ulcers, compression therapy for venous leg ulcers.

Frequency of reassessment

Informal observation of the wound is likely to take place at each dressing change. However, it is advisable to plan for formal reassessment at specified intervals. For diabetic foot ulcers, it has been suggested that reassessment is carried out weekly. (WUWHS, 2016b).

Reassessment should follow the same steps used when assessing a patient (see BPS 1, page 6 and Table 2, page 7). Clinicians should look for signs of improvement or deterioration (Table 5). Signs of deterioration should be investigated for cause. For example, increased exudate or pain levels may indicate infection, or in a venous leg ulcer may also indicate sub-therapeutic compression therapy. Fluctuations in the frequency of dressing changes required may also indicate alterations in the wound condition.

Change in wound area after four weeks

Change in wound area is commonly used as a relatively objective means of tracking wound healing progress. It is calculated as the percentage reduction in wound area from the initial assessment.

Research suggests that the percentage of wound reduction in a given timeframe may be used as an indicator of healing, for example:

- Venous leg ulcers and pressure ulcers – a percentage area reduction of ≥40% after 4 weeks of treatment is indicative of healing (Phillips et al, 2000; Kantor & Margolis, 2000; Flanagan, 2003; Günes, 2009)
- Diabetic foot ulcers – a percentage area reduction of ≥50% after 4 weeks of treatment is indicative of healing by 12 weeks (Sheehan et al, 2003; Coerper et al, 2009; Snyder et al, 2010; WUWHS, 2016b) (Figure 3).

As a result, percentage area reduction at 4 weeks can be used as a measure of chronicity.

Table 5. Local wound indicators of improvement/deterioration

Parameter	Change that may indicate:	
	Improvement	Deterioration
Wound bed	• Increase in granulation tissue • Decrease in slough/necrotic tissue • Reduction in wound area/volume (although a wound may increase in size as necrotic tissue and slough are removed)	• Increase in slough/necrotic tissue • Reduction in granulation tissue • Granulation tissue becomes friable • Increase in wound area/volume
Exudate level	• Levels are usually decreased as the wound heals • Changed from cloudy to clear	• Increased amount of exudate (may be indicated by dressing saturation or leakage) • Changed from clear to discoloured • Unpleasant odour • Wound bed too dry
Periwound skin	• Reduction, if present, in extent of: - Maceration/excoriation - Erythema and swelling	• Development of or increase in extent of: - Maceration/excoriation - Erythema and swelling
Odour	• Less noticeable if previously an issue	• Development of unpleasant odour
Pain	• Reduced level or frequency	• Change in nature of pain or increase in pain level

In practice, the 4-week period should start from when the appropriate treatment for wound type is implemented.

The healing of venous leg ulcers and pressure ulcers that have not reduced by ≥40% in area by the end of week 4 (or by ≥50% for diabetic foot ulcers) should be considered delayed. However, using these criteria requires accurate wound surface area calculation.

Multiplying length by width results in overestimates of wound area of between 10% and 44% (Chang et al, 2011). A range of techniques of more accurately calculating wound surface area are available, including the use of gridded acetate sheets and digital methods.

Use of the same method over the course of a patient's treatment is vital to ensuring comparability of consecutive results of measurement.

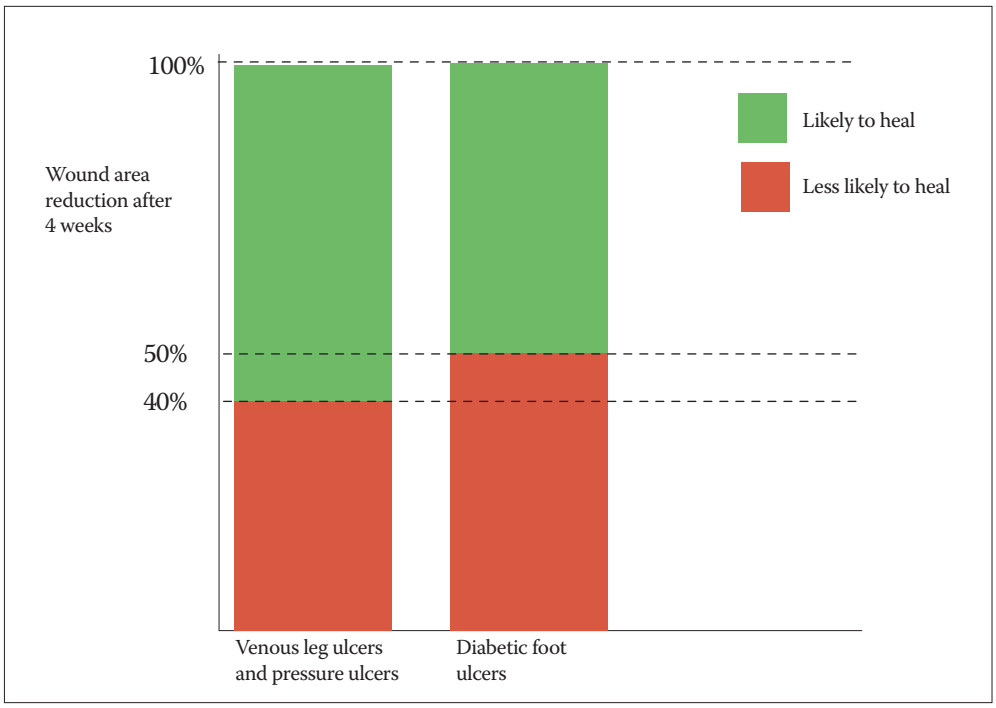


Figure 3: Wound area reduction as an indicator of likelihood of healing

KEY POINTS

1. Wounds should be reassessed regularly for signs of progress or deterioration, and to assess the suitability of the dressing and any other treatments in use
2. Signs of deterioration should be further assessed to determine cause and to adjust treatment as needed
3. Chronic wounds that have not reduced by ≥40% in area after 4 weeks of treatment (≥50% for diabetic foot ulcers) should be considered to have delayed healing
4. Each dressing change and reassessment, including wound area and wound area reduction from initial assessment at week 4, should be recorded
5. For the skills and knowledge needed to assess for signs of delayed healing see Knowledge and Skills Self-assessment 6, page 29.

BEST PRACTICE STATEMENT 6

BPS 6. IN THE ABSENCE OF OVERT INFECTION, BIOFILM SHOULD BE CONSIDERED AS THE POSSIBLE CAUSE OF DELAYED HEALING IN ALL WOUNDS THAT ARE FAILING TO PROGRESS AFTER 4 WEEKS OF OPTIMAL MANAGEMENT

High prevalence of biofilm in chronic wounds

A recent meta-analysis found the prevalence of biofilms in chronic wounds to be 78.2% (95% confidence interval 61.6-89.0, $p<0.002$) (Malone et al, 2017). The meta-analysis was based on nine studies that had biopsied wounds and used microscopy (and sometimes molecular methods) to identify the presence of biofilm.

The studies had 2–50 participants with a range of chronic wound types: diabetic foot ulcers, pressure ulcers, venous leg ulcers, non-healing surgical wounds, and chronic wounds of unspecified aetiology. Six of the nine studies found evidence of biofilm in 100% of the samples.

The authors suggested that the results indicate that biofilm is present in all chronic wounds and that the lower prevalence seen in some of the studies included may have been due to limitations in the methodology (Malone et al, 2017). For example, as distribution of biofilm within a wound is not uniform but 'patchy', a sample taken during biopsy may not include an area containing biofilm.

Biofilm and delayed wound healing

Exactly how biofilm disrupts healing is not clear and the presence of biofilm in a wound is not easy to determine. However, it is thought that biofilm causes a heightened inflammatory state (Schultz et al, 2016). This results in the release of factors such as enzymes (proteases) and reactive oxygen species (ROS) that further damage molecules important for healing, such as growth factors and components of the extracellular matrix.

Ultimately, these effects enable the biofilm to persist and delay wound healing by impairing granulation tissue formation and interfering with epithelialisation (Metcalf & Bowler, 2013; Bjarnsholt et al, 2016).

Although biofilm can be said to be present in all chronic wounds, clearly some wounds heal despite its presence (Percival et al, 2015a). The reasons for this are unclear, but may be related to the diverse and highly variable nature of biofilms and/or to the way that the biofilm and patient interact (Figure 4) (WUWHS, 2016a).

Figure 4 illustrates the paradox in chronic wounds. The force driving clockwise momentum is the virulence of the bacteria; the figure in the centre is driving counterclockwise movement, representing the healing capacity of the patient. The healthier the patient (local and systemically), the more virulent the bacteria need to be to delay or halt healing. This implies that patients in poorer health will suffer from even the most opportunistic infections.

Current treatment of chronic wounds aims at reducing local impairment using interventions such as compression, off-loading and moist wound dressings. In addition, the systemic impairments are managed by correcting

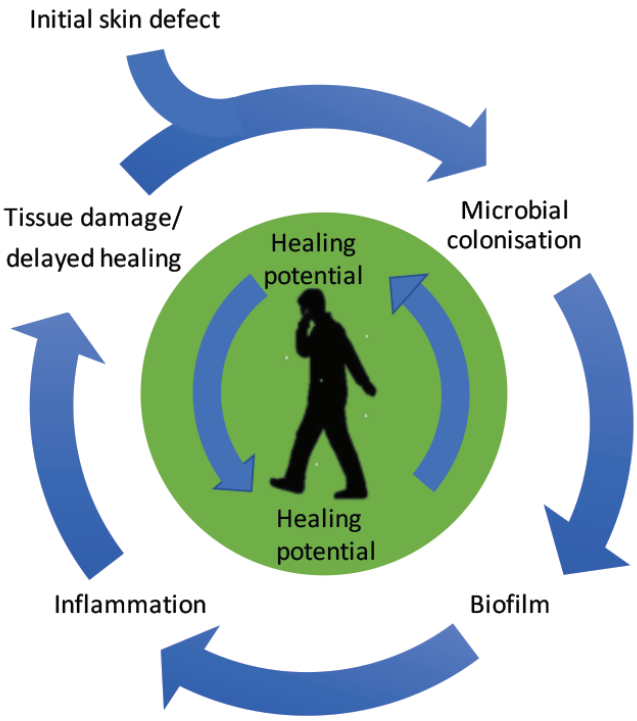


Figure 4: The wound treadmill (adapted from Bjarnsholt et al, 2016)

A simplified approach to identifying problematic biofilm

Identification of biofilm in a wound can be problematic because:

- Even though all chronic wounds probably contain biofilm, some heal in the absence of biofilm-based wound treatments
- There are considerable overlaps between the (sometimes subtle) signs of chronic wound infection and biofilm

Therefore, it is reasonable to suspect biofilm is causing a problem in chronic wounds that have not reduced in area by ≥40% (or ≥50% for diabetic foot ulcers) after 4 weeks of optimal standard care for the wound type that includes management of comorbidities or other relevant factors.

issues such as malnourishment or by adjusting glycosylated haemoglobin (HbA1c) levels.

Suspecting the presence of biofilm in wounds

Currently, only advanced microscopy or specialised culture techniques can categorically identify biofilm, although development of other methods is under investigation (Keast et al, 2014; Kalia et al, 2017; Nakagami et al, 2017). Various clinical criteria have been developed with the aim of aiding identification of biofilm presence in wounds.

Controversy exists over whether biofilm is sometimes visible in a wound bed (White & Cutting, 2012). Specialised microscopy of wound samples has found biofilm aggregates to be 0.0005mm to 0.2mm in diameter and to be patchily distributed across a wound bed (Høiby et al, 2014).

Biofilm is not visible at a macroscopic level – the one exception is oral plaque. Some clinicians have used rhetoric to promote what they believe to be visible clinical cues of biofilm presence; descriptions include a 'shiny', 'slimy' or 'translucent' layer on the non-healing wound surface (Lenselink and Andersen, 2011; Hurlow and Bowler, 2012). Although the presence of these clues is arguable, biofilm cannot in fact be seen with the naked eye (WUWHS, 2016).

Criteria

Criteria for the identification of biofilm in a chronic wound have been suggested based on clinical experience, but a definitive list is awaited. In addition, as it is now recognised that all chronic wounds contain biofilm, further research may suggest that these signs are indicative of when biofilm is having a negative effect on healing.

Table 6 (pages 18–19) summarises the criteria listed in four key papers as specific to the presence of biofilm in a chronic wound. The criteria identified by all papers were:

- Delayed healing despite optimal management of the wound and comorbidities
- Failure of response to antibiotic therapy
- Signs of local infection (IWII, 2016; Percival et al, 2015a; Keast et al, 2014; Metcalf et al, 2014).

Other criteria cited by three out of four papers include:

- Poor quality granulation tissue
- Increased exudate/moisture level (IWII, 2016; Percival et al, 2015a; Keast et al, 2014; Metcalf et al, 2014).

Other criteria mentioned, but not in all papers include:

- Failure to respond to antiseptic treatment
- Inconclusive or negative wound culture
- Surface material that is easy to remove from the wound bed but rebuilds quickly
- Slough and necrotic tissue in the wound
- Infection of >30 days' duration
- Response to anti-inflammatory agents (IWII, 2016; Percival et al, 2015a; Keast et al, 2014; Metcalf et al, 2014).

KEY POINTS

1. Studies suggest that biofilm is present in all chronic wounds
2. Currently, it is not possible to distinguish in which wounds biofilm will delay healing
3. Biofilm delays healing by keeping the wound in continuous inflammatory state which prevents normal wound healing
4. Tests for identifying biofilm in a wound are not routinely available
5. Biofilm should be suspected as being the cause of delayed healing in wounds that have not reduced in area by ≥40% (≥50% for diabetic foot ulcers) after 4 weeks of optimal treatment of the wound and underlying cause
6. For the skills and knowledge needed to assess for signs of delayed healing see Knowledge and Skills Self-assessment 6, page 29.

Table 6. Possible criteria and clinical signs for suspecting the presence of biofilm in a chronic wound derived from four key references (IWII, 2016; Percival et al, 2015a; Keast et al, 2014; Metcalf et al, 2014)

Criterion	Statements from references	Rationale
Delayed healing even when wound treatment is optimal and the underlying cause and any other contributory factors have been addressed	• Delayed healing despite optimal wound management and health support (IWII, 2016) • Failing to heal as expected (Percival et al, 2015a) • Non-healing in spite of optimal wound management and host support (Keast et al, 2014) • Wound remains recalcitrant even though all obvious underlying comorbidities have also been addressed (Metcalf et al, 2014)	• Biofilm is probably present in all chronic wounds and some heal despite it. If all other factors that may be delaying healing have been addressed, biofilm is a logical candidate for the cause of continued chronicity (Bjarnsholt et al, 2016)
Failure of response to antibiotic therapy	• Failure of appropriate antibiotic therapy (IWII, 2016) • Unresponsive to topical or systemic antibiotics (Percival et al, 2015a) • Antibiotic failure or recurring infection following antibiotic cessation (Keast et al, 2014) • History of antibiotic failure or persistent or recurring infection, despite choice of antimicrobial therapy (Metcalf et al, 2014)	• By nature the microbes in biofilms are protected from antibiotic therapy and are many times more resistant to antibiotics than the same bacterium when in planktonic (active) form (Melo, 2013; Bjarnsholt et al, 2016) • Antibiotic sensitivities reported in microbiology reports on wound samples are likely to be those for planktonic (active, free-floating) bacteria rather than for the bacteria embedded in a biofilm, meaning that an antibiotic used on the basis of the sensitivity report is not necessarily targeting biofilm bacteria (Bjarnsholt et al, 2017) • Recurrence of infection after antibiotic treatment may be explained by shedding of mobile (planktonic) microbes from biofilms (Zhao et al, 2013)
Signs of local infection	• Secondary signs of infection; low level erythema (IWII, 2016) • Signs of local infection and inflammation (Percival et al, 2015a) • Signs and symptoms of local infection (Keast et al, 2014) • Signs of local infection (redness, heat, swelling, pain, odour) (Metcalf et al, 2014)	• Biofilm can induce inflammatory changes in the wound as well as shedding microbes that themselves may cause infection and inflammation (Zhao et al, 2013) • Secondary signs of infection (increased exudate, stalled healing and cyclical waxing and waning of infection) may indicate a chronic infection and have been suggested to predominate in biofilm-affected wounds (Wolcott et al, 2008a; Wolcott et al, 2010b)
Poor quality granulation tissue	• Poor granulation/friable hypergranulation (IWII, 2016) • No evidence of granulation (Percival et al, 2015a) • Poor-quality granulation tissue (e.g. friable, hypergranulation) (Keast et al, 2014)	• Abnormal granulation tissue can be a sign of wound infection/inflammation (Grey et al, 2006) • Biofilm may shed bacteria and act as a source of infection (Zhao et al, 2013)
Increased exudate/moisture level	• Increased exudate/moisture (IWII, 2016) • Excessive moisture/exudate (Keast et al, 2014) • Excessive moisture (Metcalf et al, 2014)	• Increased exudation can be a sign of wound infection and is due to increased vascular permeability as a result of the inflammatory response (Grey et al, 2006). • Biofilm may cause chronic inflammation and may also shed bacteria and act as a source of infection (Zhao et al, 2013). • A paper reporting a case study series suggested that excessive exudate production may contribute to biofilm formation (Hurlow & Bowler, 2012)
Failure to respond to antiseptic treatment	• Recalcitrance to appropriate antimicrobial treatment (IWII, 2016) • Poor or slow response to dressings containing antiseptics (Metcalf et al, 2014)	• By nature, the microbes in biofilms are protected from the action of antimicrobials and antiseptics (Bjarnsholt et al, 2016)

Inconclusive or negative wound culture report	• Swab results are often inconclusive (Keast et al, 2014) • Culture-negative results despite signs of critical colonisation or a high suspicion of clinical infection (Metcalf et al, 2014)	• Wound culture based on samples acquired by swabbing will generally identify planktonic microbes only and is not suitable for sampling biofilm microbes (Wolcott & Ehrlich, 2008; Fonseca, 2011)
Surface material is easy to remove and rebuilds quickly	• Gelatinous material easily removed from the wound surface; surface substance reforms quickly (Keast et al, 2014) • Surface substance: - Reforms quickly (in 1 to 2 days) in the absence of frequent intervention, e.g. cleansing, debridement - Persists despite use of autolytic or enzymatic debridement - Detaches easily and atraumatically from the underlying wound bed using physical removal techniques such as swabs, pads or sharp debridement (Metcalf et al, 2014)	• Some researchers disagree that this is a sign of biofilm, in part at least because biofilm occurs in patches across a wound bed (Percival et al, 2015a; Percival et al, 2015b) • The 'slimy' layer may be mainly fibrinous slough produced in response to biofilm (Bourdillon, 2016) • A study of an *in vitro* model of biofilm formation and 'debridement' using sprayed saline found that biofilm reformed within 24–48 hours of debridement (Wolcott et al, 2010a)
Other criteria • Slough and necrotic tissue in the wounds (Percival et al, 2015a) may act as surfaces for biofilm attachment (Wolcott et al, 2010b)		

BEST PRACTICE STATEMENT 7

BPS 7. ONCE BIOFILM IS SUSPECTED PROACTIVE TREATMENT SHOULD INCLUDE STRATEGIES TO PHYSICALLY DISRUPT AND REMOVE THE EXISTING BIOFILM

Proactive biofilm treatment

The inherent resistance of biofilm to topical or systemic antimicrobial agents requires a proactive approach to treatment, known as biofilm-based wound care, that:

- Repeatedly physically disrupts and removes biofilm – through vigorous/active cleansing or debridement
- Reduces reformation of biofilm – through the use of antimicrobial dressings or topical antiseptic preparations (see BPS 8, pages 24–25) (Wolcott et al, 2009; Bianchi et al, 2016; Fletcher et al, 2016).

Other elements of standard care for the wound type should be continued during biofilm-based wound care, e.g. compression therapy should be continued for venous leg ulcers and pressure redistribution for pressure ulcers.

Physical disruption and removal of biofilm

Biofilms are highly resistant to cleansing by standard irrigation (Atiyeh et al, 2009). Consequently, disruption and removal of biofilm needs to be achieved by more thorough methods.

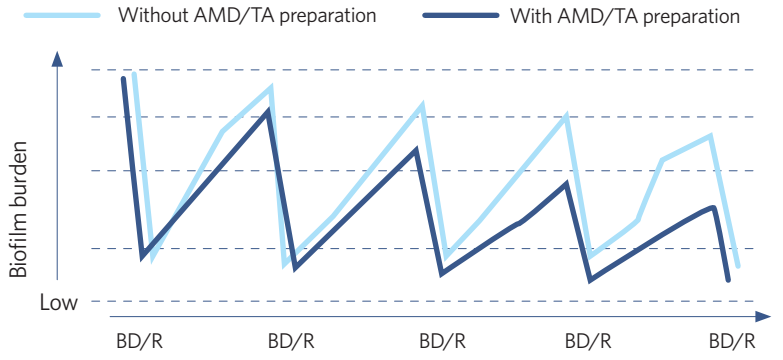
In addition to removing dead, non-viable tissue, slough and infected or foreign material from the wound bed, some forms of vigorous/active cleansing and debridement will also remove biofilm (Wolcott et al, 2010a; Debridement Consensus, 2013).

As covered in BPS 6, since biofilm is not uniformly distributed across a wound attempts to physically disrupt and remove it are unlikely to eradicate it, and ongoing, regular disruption/removal may be necessary (Fazli et al, 2009; Wolcott et al, 2009).

As well as reducing the amount of biofilm in a wound, disrupting and removing it may also increase the vulnerability of the biofilm to antimicrobial agents. As the biofilm tries to reform, the remaining microbes proliferate, becoming more metabolically active for a short period of time. This disruption provides a 'window' of increased susceptibility to antimicrobial agents (Wolcott et al, 2009).

A study using an *in vitro* model of biofilm showed that it is able to reform within a few days of being physically disrupted but that it is more susceptible to antimicrobial treatments for 24–48 hours post-disruption (Wolcott et al, 2010a). These findings support the rationale for the combined use of antimicrobial dressings or topical antiseptic preparations and frequent, repeated cycles of disruption/removal, in the form of vigorous/active cleansing and/or debridement/, to reduce overall biofilm load.

Figure 5 is a hypothetical illustration of effects on biofilm burden of physical disruption/removal of biofilm alone and disruption/removal combined with antimicrobial dressing or topical antiseptic preparation use.



BD/R= biofilm disruption/removal due to vigorous/active cleansing or debridement
AMD= antimicrobial dressing
TA= topical antiseptic

Box 7. Definition of surfactant

Surfactant – 'surface active agent' – is a term applied to a large group of molecules that lower the surface tension of a liquid and that have wide-ranging applications in industry and medicine. The most widely known surfactant is soap.

Box 8. Factors affecting choice of method for biofilm disruption/removal based on factors affecting choice of debridement method (Moore, 2012; Atkin, 2014)

- Patient – e.g. contraindications to a method of debridement, patient preference/consent
- Wound being treated – e.g. aetiology, anatomical location, amount of non-viable tissue that needs to be removed, anatomical location
- Knowledge and skills of the healthcare practitioner
- Environment – e.g. availability of equipment/resources needed, local policy and regulations.

Figure 5: Hypothetical effect of cycles of biofilm disruption/removal with and without an antimicrobial dressing or topical antiseptic preparation on biofilm burden in a wound

Methods of biofilm disruption and removal

Table 7 (pages 22–23) provides an overview of the main methods of vigorous/active cleansing/debridement that may be used for disruption or removal of biofilm, and indicates which require specialist training. The table also summarises the effects of the different methods on biofilm. Mechanical and/or sharp debridement are the techniques recommended for most chronic wounds (Bianchi et al, 2016) with some exceptions (see Box 8).

For further details on the different types of debridement, see BPS 4, page 12.

Although autolytic debridement is the most widely used form of debridement, it is slow to act and its effects on biofilm are unclear (Wolcott et al, 2009). Mechanical debridement with a monofilament fibre debridement pad is in effect a form of vigorous/active cleansing. It is easy to use and acts quickly to remove biofilm (Wilkinson et al, 2016). Sharp debridement, while quick and effective, requires additional or specialist training, as do larval, ultrasonic, hydrosurgical and surgical debridement.

Other methods of biofilm disruption and removal

Surfactants

Some surfactants (Box 7) can aid solubilisation of proteins and block cell adhesion to surfaces (Yang et al, 2016). In wound care therefore, surfactants are of interest as potential agents to aid cleansing, and to prevent and remove biofilm (Leaper et al, 2012).

Currently, products containing surfactants often contain an antimicrobial agent (Table 8, page 24), e.g. betaine (surfactant) combined with PHMB (antimicrobial), and octenidine dihydrochloride (antimicrobial) combined with ethylhexylglycerin (surfactant). Both combinations have been shown to remove biofilm *in vitro* and to reduce wound bioburden (Bradbury & Fletcher, 2011; Braun et al, 2013).

Interest is growing in the effects on biofilm of surfactants alone. A topical gel based on a poloxamer surfactant has been shown to reduce biofilm bacteria to undetectable levels over 3 days in a porcine wound model that incorporated daily wiping with gauze to simulate cleansing (Yang et al, 2016).

Dressings

An *in vitro* study of dialkylcarbamoyl chloride (DACC)-coated dressings found that *Pseudomonas aeruginosa* and methicillin-resistant *Staphylococcus aureus* (MRSA) biofilms became bound to the dressing (Cooper & Jenkins, 2016). The clinical significance of this effect on biofilm is not yet clear.

Others

Agents that interfere with biofilm attachment include lactoferrin, xylitol and honey (WUWHS, 2016a). Ethylenediaminetetraacetic acid (EDTA) also disrupts the extrapolymeric substance (EPS — the microbe-protecting matrix of a biofilm) (WUWHS, 2016a).

Implementing biofilm disruption and removal

A variety of factors will affect the biofilm disruption and removal strategy chosen (Box 8). The strategy chosen should be within the competency of the practitioner using it. Some wound types require specialist involvement when considering debridement (Box 9).

The ideal frequency of disruption/removal in the treatment of biofilm has not yet been determined. Laboratory data on biofilm reformation rates and development of antimicrobial resistance suggest every 48–72 hours (Wolcott et al, 2010a). However, the frequency will depend on factors such wound characteristics and method used. As examples, frequencies used in clinical studies of biofilm-based wound management include the 2 to 3 times weekly use of a monofilament fibre debridement pad in lower limb wounds (mainly venous leg ulcers and pressure ulcers), and once weekly with sharp debridement in patient with critical limb ischaemia (Wolcott et al, 2008b; Morris et al, 2016).

If a wound is not improving as expected using one type of biofilm disruption/removal, a more 'aggressive' form of vigorous/active cleansing/debridement may need to be considered, with specialist referral where appropriate (Phillips et al, 2010).

For further reading, see page 33.

KEY POINTS

1. Treatment of biofilm requires a proactive approach that:
   - Repeatedly physically disrupts and removes the biofilm, e.g. vigorous/active cleansing or debridement; and
   - Reduces reformation of the biofilm through the use of antimicrobial dressings or topical antiseptic preparations (see pages 24–25)
2. Other aspects of standard care specific to the wound type should be continued
3. Repeated cycles of disruption and removal with intervening antimicrobial therapy are needed
4. Biofilm disruption and removal are usually achieved by vigorous/active cleansing/debridement
5. The method of debridement used should be chosen with care and carried out by a specialist where specific skills are required
6. For the skills and knowledge needed to proactively treat biofilm see Knowledge and Skills Self-assessment 4, 5 and 6, page 29.

Box 9. Wound types that may need specialist involvement for debridement (Vowden & Vowden, 2011)

- Wounds on the hands, feet or face
- Lower limb wounds in patients with arterial disease
- Inflammatory wounds such as pyoderma gangrenosum
- Wounds in which malignancy is suspected or that are associated with a congenital abnormality
- Wounds with a prosthetic implant nearby

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Table 7. Types of vigorous/active cleansing or debridement that may be used for biofilm disruption and removal (Gray et al, 2011; Debridement Consensus, 2013; Strohal et al, 2013; Collins et al, 2014; Collier, 2014; Atkin, 2014; Korzendorfer & Hettrick, 2014; Percival & Suleman, 2015)

Type	Mode of action	Advantages	Disadvantages	Effect on biofilm
Autolytic May be performed by a generalist nurse or specialist practitioner	• Devitalised tissues are softened and liquefied through the action of enzymes occurring naturally in the wound • Aided by dressings that manage exudate or donate moisture to produce a moist wound environment	• Can be used before or between other methods of debridement • Does not cause pain • Selective and non-invasive	• Slow – may take weeks • May cause maceration • Ease of use may lead to overuse and delay in use of more appropriate forms of debridement	• Autolytic debridement encourages separation of devitalised tissue and slough, which may contain biofilm. However, it is slow to act and does not work specifically on biofilm (Wolcott et al, 2009) • Evidence of effect on biofilm is lacking
Mechanical May be performed by a generalist nurse or specialist practitioner	• Vigorous cleansing with a soft monofilament fibre debridement pad (e.g. Debrisoft®, Activa L&R) or cloth is used on the wound surface to detach and remove devitalised tissue *(N.B. Traditional wet-to-dry dressing debridement is not recommended in the UK)*	• Quick; easy to use • Can be used in conjunction with autolytic debridement • In addition, effective on hyperkeratosis • Patients can use for self-care under supervision	• Unsuitable for hard, dry eschar	• *In vitro*: in a glass plate biofilm model, a monofilament fibre debridement pad was found to remove and retain biofilm significantly better than a cloth with poloxamer and cotton gauze pads (Wiegand et al, 2014); monofilament devices have been shown to disrupt and remove biofilm in a porcine skin wound model study that used microscopy and culture techniques, and to reduce bacterial counts (Wilkinson et al, 2016) • Indirect clinical evidence: in an audit of 475 patients with chronic wounds treated for 2 weeks using a biofilm-based wound care pathway that included using a monofilament fibre debridement pad (Debrisoft, Activa L&R) at wound dressing changes and a topical antimicrobial, clinicians reported that: - 94% of wounds had a positive healing response - 95% had a reduction in the amount of slough and debris - 94% had an improvement in granulation tissue and skin condition - 43% of patients in a follow-up audit of 142 patients went on to heal within a 1 to 3-month period (Morris et al, 2016)

Sharp May be performed by a surgeon or specialist practitioner who has undergone further training	Devitalised tissue is removed using a scalpel, scissors and/or forceps	• Quick and selective • Useful on hard eschar	• Requires specialist training • Risk of damaging underlying or nearby structures such as nerves, blood vessels and tendons	• *In vivo:* a study using an animal model of biofilm found that the total amount of bacteria (used as a measure of both planktonic and biofilm bacteria) at day 2 after sharp debridement was significantly lower than with no debridement (Nusbaum et al, 2012) • Indirect clinical evidence: stated to be clinically effective in removing biofilm on the basis of clinical studies and case series that have shown improved healing when sharp debridement is used (sometimes in combination with antimicrobials) (Wolcott et al, 2009; Hurlow & Bowler, 2009; Hurlow & Bowler, 2012)
Surgical May be performed by a surgeon or specialist practitioner who has undergone further training	Excision/resection of non-viable tissue and healthy tissue from wound margins to achieve a healthy bleeding wound bed	• Selective • Useful for hard eschar and to debride large areas	• Requires specialist training • Usually requires an anaesthetic and an operating theatre, consequently time-consuming and resource heavy	• Excision of devitalised or infected host tissue is likely to also remove biofilm (Metcalf et al, 2014) • Indirect clinical evidence: in a retrospective analysis, patients with venous leg ulcers had significantly higher rates of wound area reduction following surgical debridement versus no surgical debridement (Cardinal et al, 2009)
Larval (biosurgical) May be performed by a generalist nurse or specialist practitioner	Larvae of the green bottle fly (*Lucilia sericata*) are placed loose or bagged in the wound where they ingest devitalised tissue and microbes	• Selective and rapid • Reduces pain, bacteria and malodour	• Unsuitable for dry, excessively moist or malignant wounds, wounds that communicate with a body cavity/organ or near major blood vessels • Patients may decline in health	• *In vitro*: larval therapy significantly reduced total bacterial load (planktonic and biofilm bacteria) on pig skin biofilms after 24 hours; scanning electron microscopy showed that biofilm was eliminated completely within 24–48 hours of exposure to the larvae (Cowan et al, 2013) • Indirect clinical evidence: 67 of 68 patients with chronic wounds treated with larval debridement achieved >90% debridement of necrotic tissue in 2–10 days; most wounds healed with follow-up wound care (Campbell & Campbell, 2014)
Ultrasonic May be performed by a generalist with specialist training or a specialist practitioner	Ultrasound energy breaks up the devitalised tissue and biofilm; most devices include a built-in irrigation system to wash out the fragments	• Immediate and selective effect	• Requires specialist equipment and training	• *In vitro*: ultrasound enhanced the effectiveness of antimicrobial agents against planktonic and biofilm bacteria (Korzendorfer & Hettrick, 2014; Crone et al, 2015) • Indirect clinical evidence: ultrasonic-assisted debridement was effective in debriding and stimulating healing in two cases (Butcher & Pinnuck, 2013)
Hydrosurgical May be performed by a generalist with specialist training or a specialist practitioner	A high-pressure jet of saline is used as a cutting implement	• Relatively quick and selective	• Requires specialist equipment and training • May be painful	• *In vivo*: a study using an animal model of biofilm found that the total amount of bacteria (taken to be a measure of both planktonic and biofilm bacteria) at day 2 after hydrosurgical debridement was significantly lower than with no debridement (Nusbaum et al, 2012) • Indirect clinical evidence: diabetic foot ulcers in a series of 15 patients were successfully debrided; 12 healed after split thickness grafting and 3 by secondary healing (Hong et al, 2014)

BEST PRACTICE STATEMENT 8

BPS 8. ONCE BIOFILM IS SUSPECTED TREATMENT SHOULD INCLUDE STRATEGIES TO REDUCE MICROBIAL LOAD BY USING AN ANTIMICROBIAL DRESSING OR A TOPICAL ANTISEPTIC PREPARATION FOR 2 WEEKS

Role of antimicrobials in biofilm treatment

In addition to disruption and removal of biofilm (see BPS 7, pages 20–23), biofilm-based wound care includes the use of topical antimicrobial agents because efforts to disrupt and remove biofilm, such as vigorous/active cleansing and debridement, are unlikely to remove all biofilm from a wound (Rhoads et al, 2008; Bianchi et al, 2016; Fletcher et al, 2016). Biofilm may grow or reform from:

- Remnants of biofilm
- Planktonic bacteria released from residual biofilm
- Other microbes remaining in the wound
- Newly introduced microbes (Philips et al, 2010)

The principles of minimising biofilm reformation are therefore:

- Protection of the wound from further contamination by other microbes, e.g. through the use of a dressing
- Reduction of the number of microbes, e.g. using topical antimicrobial agents (Keast et al, 2014; Fletcher et al, 2016; Bianchi et al, 2016).

Studies of biofilm have shown that after disruption it takes about 72 hours for antimicrobial numbers to reach pre-disruption levels (Wolcott et al, 2010a). Consequently, topical antimicrobials should be used after biofilm disruption (Wounds UK, 2013).

Table 8. Agents used in antimicrobial dressings and/or topical antiseptic preparations and effects on biofilm (Cowan, 2016; Wounds UK, 2013; IWII, 2016)

Agent	Antibiofilm effects	Formulation(s)	Notes
Antimicrobial enzymes (glucose oxidase and lactoperoxidase) (Cooper, 2013)	• Inhibits biofilm formation	• Gel also containing alginate	• Intended to be left in the wound bed under a suitable dressing • Avoid in patients with allergy to any of the components
Honey (Cooper et al, 2011)	• Inhibits biofilm growth and colony formation	• Impregnated dressings; liquid	• Avoid in patients with allergy to bee venom • Select products that have been gamma irradiated to sterilise • Blood glucose monitoring may be necessary in patients with diabetes
Iodine (Wound Healing and Management Node Group, 2012; Thorn et al, 2009)	• Inhibits development and reduces viability of biofilm	• Povidone or cadexomer iodine: impregnated dressings; powder; ointment	• Contraindicated in patients with thyroid or renal disease, or who are sensitive to iodine
Octenidine dihydrochloride (Braun et al, 2011)	• Inhibits growth and aids removal of biofilm	• Gel and solution (both also contain the surfactant ethylhexylglycerin)	• Avoid in patients with sensitivity to octenidine or wounds with exposed cartilage • Solution can be used as a soak during cleansing • Gel can be applied to the wound bed and left *in situ* under a suitable dressing
Polyhexamethylene-biguanide (PHMB) (Butcher, 2012; King & Barrett, 2016)	• Effective against planktonic and biofilm bacteria	• Gel and solution (both also contain the surfactant betaine) • Impregnated dressings	• Avoid in patients with sensitivity to PHMB • Solution can be used as a soak during cleansing • Gel can be applied to the wound bed and left *in situ* under a suitable dressing
Silver (International Consensus, 2012b; Hedger, 2015; Metcalf et al, 2016)	• Some dressings have shown biofilm inhibitory effects; silver may reduce bacterial adhesion	• Ionic silver and nanocrystalline silver: impregnated dressings; paste • Silver sulfadiazine: cream, impregnated dressings	• Avoid in patients sensitive to silver • Silver sulfadiazine should be avoided in patients sensitive to sulphonamide antibiotics

Antimicrobial agents

Antimicrobial agents act by killing or inhibiting the growth of microbes. The main types of antimicrobial agents used in wound care are:

- Antiseptics – non-selective agents that are used topically on skin or in wounds; development of resistance is unusual; generally formulated as antimicrobial dressings or topical antiseptic preparations
- Antibiotics – selective agents that act against bacteria that may be used topically or systemically; topical use is not recommended for the treatment of biofilm or wound infection because of the increasing risk of development of resistance (WUWHS, 2008; Wounds UK, 2013).

Some antiseptics may have toxic effects on human cells and practitioners need to balance the risk/benefit ratio of use in each wound (Wounds UK, 2013).

Using topical antiseptics

Topical antiseptics for use in wounds come in a variety of formulations: liquids, creams, gels, powders, sprays and antiseptic-impregnated dressings (often known as antimicrobial dressings). Some formulations are intended for use for short periods, e.g. a solution for use during cleansing, and others are intended to be left in contact with the wound for up to several days, e.g. an antimicrobial dressing.

In general, treatment of biofilm involves the use of an antiseptic agent continuously between the cycles of biofilm disruption/removal, often in the form of an antimicrobial dressing or a topical antiseptic preparation, such as an antiseptic-containing gel, held in place by a suitable dressing. To maximise the impact on wound microbial load, an antiseptic solution can be used during cleansing (WUWHS, 2008). See Box 6, page 12, for definitions of antimicrobial dressings, topical antiseptic preparations and irrigation/cleansing solutions.

Selection of the antimicrobial formulation may need to take into account factors such as known allergies or sensitivities, pain response, wound exudate level, need for odour control, anticipated change frequency, availability and cost. Table 8 (page 24) summarises the types, modes of action and formulations of the main topical antiseptics used in the UK in wound care.

Reassess after two weeks

Antimicrobial dressings and topical antiseptic preparations should not be used indefinitely (WUWHS, 2008). Response to the use of the dressing or preparation should be assessed after 2 weeks. If the wound has improved, the dressing or preparation should be discontinued. See pages 26–27 for more information on applying the topical antiseptic 2-week challenge in the management of biofilm.

Systemic antibiotics

Systemic antibiotics are generally not indicated in wounds where biofilm is suspected to be the cause of delayed healing because of the inherent resistance of biofilm microbes (Rhoads et al, 2008). However, microbes capable of infection are released from biofilm and other microbes present within the wound may cause spreading infection and systemic illness. In these situations, systemic antibiotics may be indicated and should be selected and administered in line with local policy (WUWHS, 2008).

For further reading see page 33.

KEY POINTS

1. Biofilm-based wound care uses antimicrobial dressings or topical antiseptic preparations to reduce the risk of biofilm regrowth alongside disruption and removal of the biofilm
2. A dressing will protect the wound from contamination by additional microbes
3. The active agents in antimicrobial dressings or topical antiseptic preparations kill or suppress the growth of microbes
4. Topical antiseptics are usually applied in the form of antimicrobial dressings or a gel held in place by a suitable dressing
5. Antiseptic solutions may also be used for wound cleansing
6. The choice of product(s) to treat the wound for biofilm may need to take into account other needs, such as high absorption if exudate levels are high
7. Antimicrobial dressings and topical antiseptic preparations should not be used indefinitely: their use should be reviewed after 2 weeks (the '2-week challenge')
8. The details of the antimicrobial dressing or topical antiseptic preparation selected should be documented
9. For the knowledge and skills needed to reduce microbial load, see Knowledge and Skills Self-assessments 4, 5 and 6, page 29.

BEST PRACTICE STATEMENT 9

BPS 9. WOUNDS SHOULD BE RE-EVALUATED AFTER 2 WEEKS OF BIOFILM-BASED WOUND MANAGEMENT. IF THE WOUND HAS NOT RESPONDED AND BIOFILM IS STILL SUSPECTED CONSIDER A SECOND OR THIRD ROUND OF TREATMENT USING A DIFFERENT REGIMEN

The 2-week challenge

The concept of the 2-week challenge was developed to avoid indefinite use of antimicrobial dressings or topical antiseptic preparations and to prompt review of the wound (International Consensus, 2012b). Figure 6 provides an example of biofilm-based wound care using mechanical debridement and the 2-week challenge.

After 2 weeks of use of an antimicrobial dressing or a topical antiseptic preparation, the wound should be assessed for signs of improvement or deterioration (see Table 5, page 14). If after 2 weeks the wound has:

- **Improved** – the topical antiseptic should be discontinued and standard care for the wound type continued with regular reassessment; in some circumstances, e.g. in a wound at high risk of infection, it may be justifiable to continue the antimicrobial dressing or topical antiseptic preparation in line with local protocols with further regular reviews (International Consensus, 2012b; Wounds UK, 2013).
- **Not improved** – reassess the patient and the wound and amend management plan as needed; the antimicrobial dressing or topical antiseptic preparation in use should be discontinued and a different type of antiseptic agent should be considered for a further 2-week challenge followed by reassessment (Table 8, page 24)
- **Deteriorated** – reassess the patient and the wound to determine the reason for deterioration; change the management plan accordingly and refer as necessary. If treatment with an antimicrobial dressing or topical antiseptic preparation remains appropriate, consider switching to another type (Table 8, page 24).

Where the decision is made to change the type of antiseptic agent, the principles of the 2-week challenge still apply and the wound should be reassessed 2 weeks after start of the new treatment. If appropriate, a third type of antiseptic agent can be used for a further 2 weeks. This should continue for a maximum of three cycles, i.e. three, 2-week challenges using three different antiseptic agents. If there is no healing progression after the third cycle, consider specialist referral (Morris et al, 2016).

Deterioration of the wound during treatment with an antimicrobial dressing or a topical antiseptic preparation should trigger reassessment of the patient and the wound.

It is important to remember that even when the wound is improving and progressing towards healing, some biofilm is likely to remain in the wound and may interfere with healing later in the healing process.

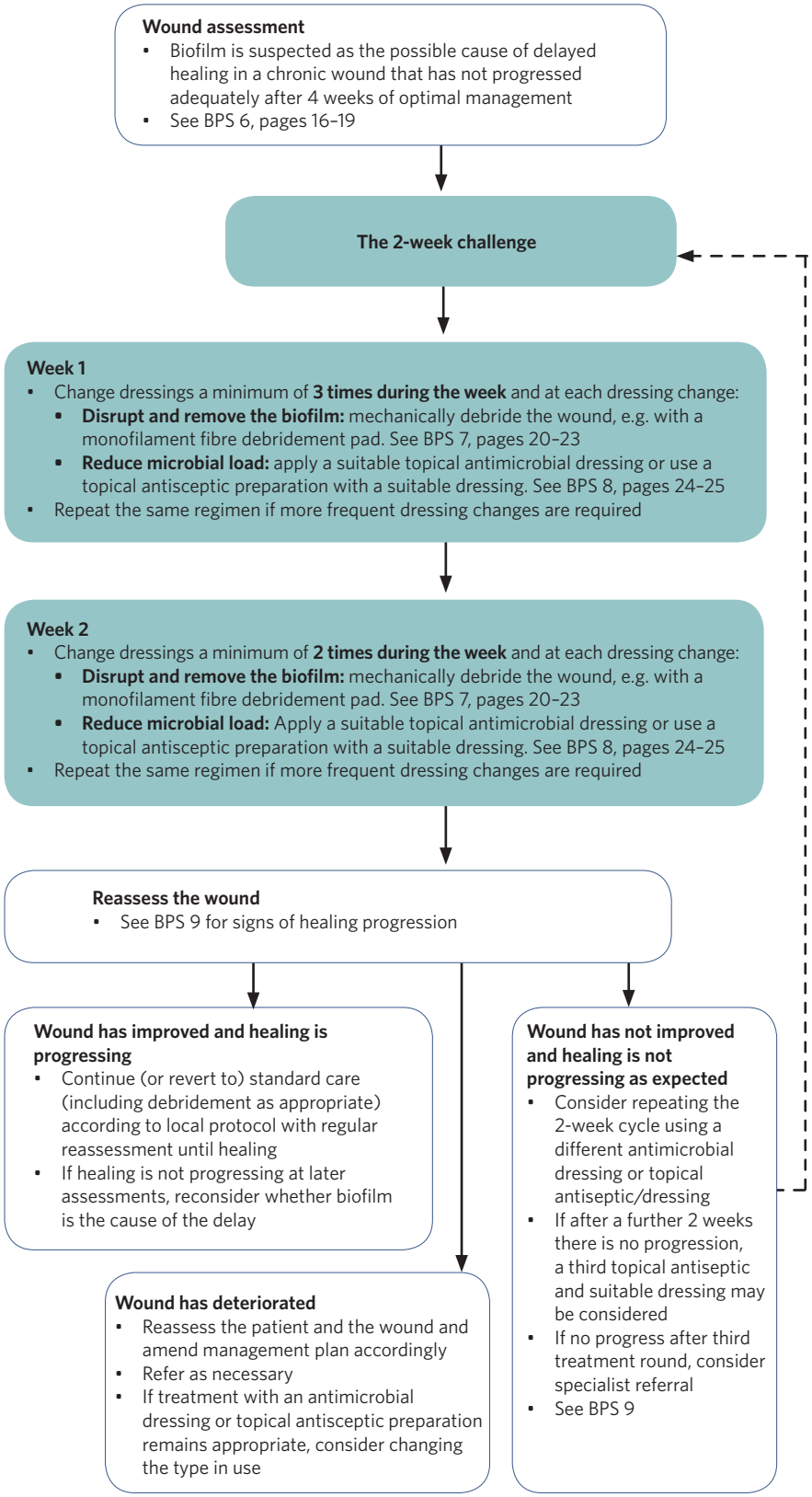


Figure 6: Biofilm-based wound management: the use of mechanical debridement in the context of the 2-week challenge

KEY POINTS

1. A wound being treated for biofilm should be reassessed after 2 weeks of treatment with an antimicrobial dressing or topical antiseptic preparation
2. If the wound has:
   - improved, the antimicrobial/or topical antiseptic preparation should be discontinued, (unless further use is justified) and standard care for the wound type continued
   - has not improved, a further 2-week challenge with an antimicrobial/ topical antiseptic preparation containing a different type of antiseptic agent should be considered
   - has deteriorated, the patient and the wound should be fully reassessed and specialist referral considered
3. A maximum of three cycles of the 2-week challenge may be used
4. If the wound is not improving after the third cycle, consider specialist referral
5. Even in wounds that are improving, biofilm may remain and affect future healing
6. Results of the reassessment and the details of any changes antimicrobial/topical antiseptic preparation use should be reported.

SECTION 3: KNOWLEDGE AND SKILLS SELF-ASSESSMENT FOR WOUND AND BIOFILM MANAGEMENT

The principles underlying wound management — assessment, planning, treatment and evaluation — can be viewed as a cycle that repeats until the wound is healed (Figure 7). At each stage, clinicians need to decide whether their knowledge and skills are sufficient for effective and safe implementation. If they are not, the patient should be referred to a clinician with the necessary knowledge and skills. In practice, escalation is often not the end of a clinician's involvement in management of the wound as other aspects of treatment may be within their skill set.

Figure 7 links to the more detailed Knowledge and Skills Self-assessments that can be found in Figure 8. The self-assessments will encourage individuals to identify areas for further development and to seek appropriate training. In addition, early recognition that escalation may be necessary will aid a patient's timely progress through the wound management cycle and hopefully reduce time to healing.

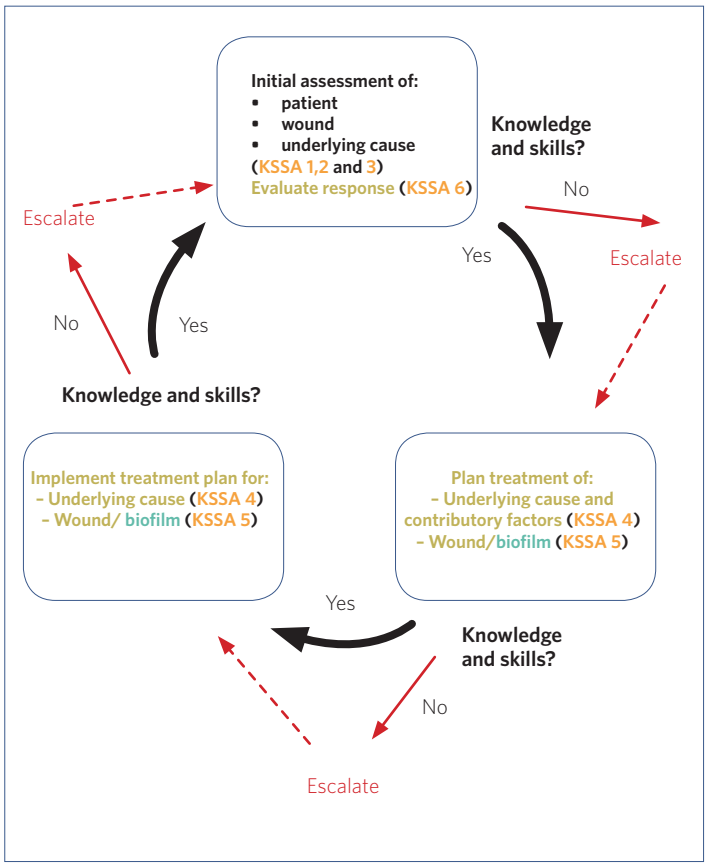


Figure 7: Principles of wound management with knowledge and skills self-assessment

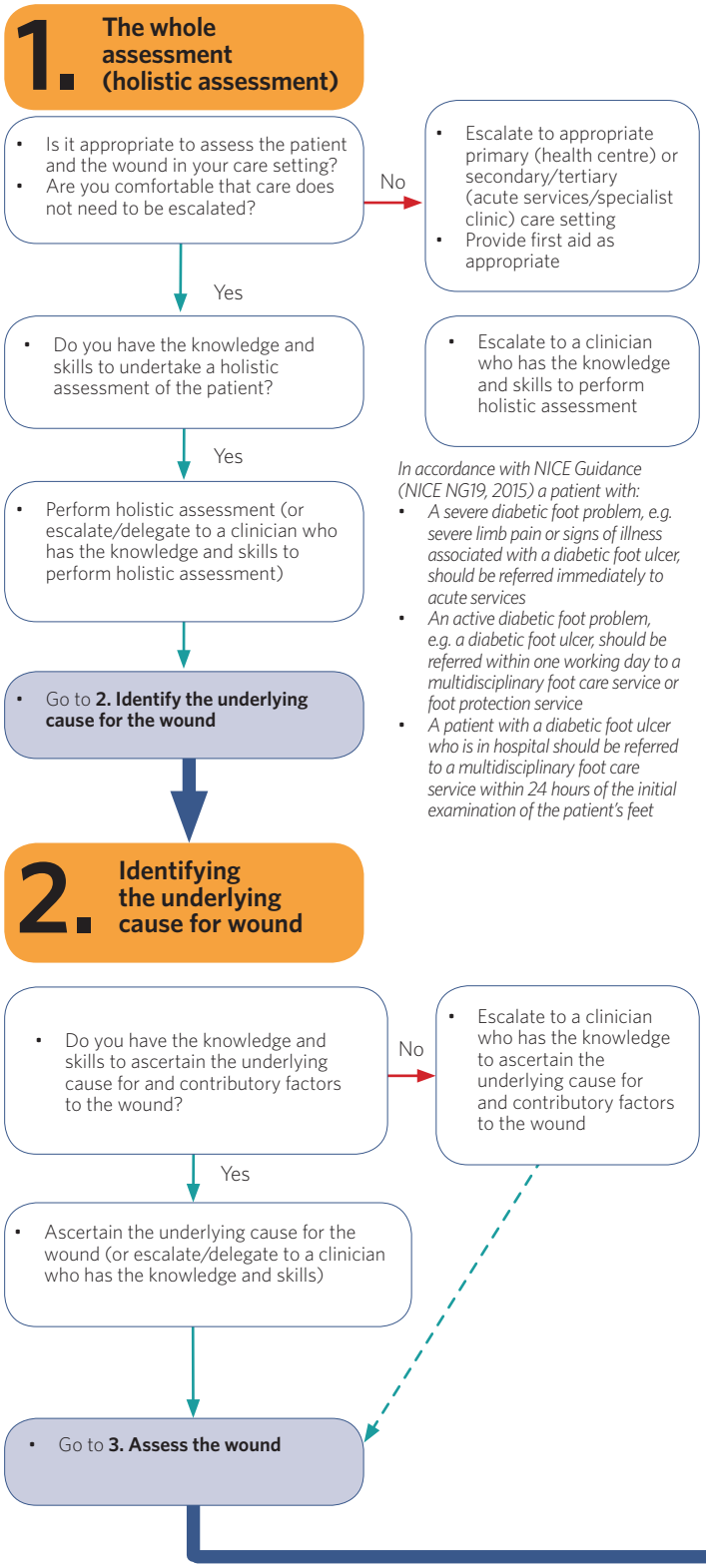
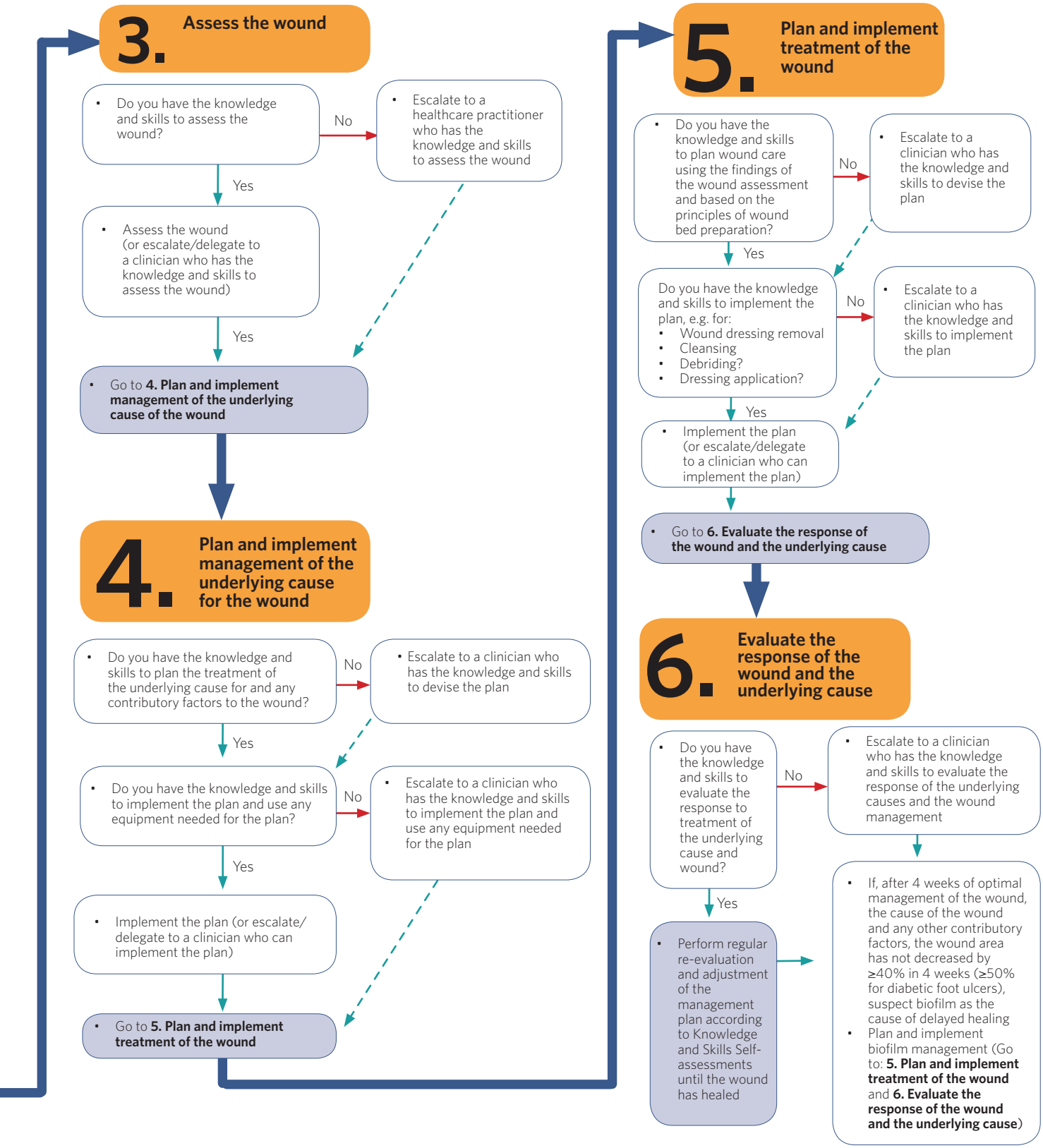


Figure 8: Knowledge and skills assessments


3. Assess the wound
Do you have the knowledge and skills to assess the wound?
No
Escalate to a healthcare practitioner who has the knowledge and skills to assess the wound
Yes
Assess the wound (or escalate/delegate to a clinician who has the knowledge and skills to assess the wound)
Yes
Go to 4. Plan and implement management of the underlying cause of the wound
4. Plan and implement management of the underlying cause for the wound
Do you have the knowledge and skills to plan the treatment of the underlying cause for and any contributory factors to the wound?
No
Escalate to a clinician who has the knowledge and skills to devise the plan
Yes
Do you have the knowledge and skills to implement the plan and use any equipment needed for the plan?
No
Escalate to a clinician who has the knowledge and skills to implement the plan and use any equipment needed for the plan
Yes
Implement the plan (or escalate/delegate to a clinician who can implement the plan)
Go to 5. Plan and implement treatment of the wound
5. Plan and implement treatment of the wound
Do you have the knowledge and skills to plan wound care using the findings of the wound assessment and based on the principles of wound bed preparation?
No
Escalate to a clinician who has the knowledge and skills to devise the plan
Yes
Do you have the knowledge and skills to implement the plan, e.g. for:
Wound dressing removal
Cleansing
Debriding?
Dressing application?
No
Escalate to a clinician who has the knowledge and skills to implement the plan
Yes
Implement the plan (or escalate/delegate to a clinician who can implement the plan)
Go to 6. Evaluate the response of the wound and the underlying cause
6. Evaluate the response of the wound and the underlying cause
Do you have the knowledge and skills to evaluate the response to treatment of the underlying cause and wound?
No
Escalate to a clinician who has the knowledge and skills to evaluate the response of the underlying causes and the wound management
Yes
Perform regular re-evaluation and adjustment of the management plan according to Knowledge and Skills Self-assessments until the wound has healed
If, after 4 weeks of optimal management of the wound, the cause of the wound and any other contributory factors, the wound area has not decreased by ≥40% in 4 weeks (≥50% for diabetic foot ulcers), suspect biofilm as the cause of delayed healing
Plan and implement biofilm management (Go to: 5. Plan and implement treatment of the wound and 6. Evaluate the response of the wound and the underlying cause)

APPENDIX 1: WOUND BIOFILM STRUCTURE AND LIFE CYCLE

For some time, the presence of biofilm has been implicated in a wide range of diseases, e.g. chronic tonsillitis, chronic sinus problems, urinary tract infections and dental decay.(Costerton et al, 1999). However, it is only relatively recently that biofilm in wounds has been recognised as being potentially pathogenic (Percival et al, 2015).

Biofilm has been defined as '*an aggregate of microbial cells adherent to a living or non-living surface, embedded with a matrix of extracellular polymeric substance of microbial origin*' (Hall-Stoodley et al, 2012).

The extrapolymeric substance (EPS) is the protective coating that helps the biofilm resist the patient's immune system and antimicrobial treatments (Bjarnsholt et al, 2016). It is produced by the embedded microbes and is made up of polysaccharides, proteins, glycolipids and free DNA (Bjarnsholt et al, 2016).

The EPS firmly attaches the biofilm to a surface, e.g. the wound bed (Phillips et al, 2010). In fact, biofilm is also found just below the wound surface (Schultz et al, 2016).

Biofilm is not uniformly distributed across a wound bed and exists in separate islands. Although a wound may contain biofilm derived from several different microbial species, individual patches of biofilm may contain only one species (Bjarnsholt et al, 2016).

The microbes in a biofilm may communicate with each other by releasing signalling molecules in a system called quorum sensing (Keast et al, 2014). Biofilm microbes are generally less metabolically active than free (planktonic) microbes. This dormant state provides some of the resistance to antimicrobial treatments (Phillips et al, 2010).

Biofilm development starts with the attachment of planktonic microbes to the wound bed (Figure 8). When mature, a biofilm sheds planktonic bacteria, microcolonies and fragments of biofilm, to form new areas of biofilm in the wound, to cause overt infection or to disperse more widely (Phillips et al, 2010). While in the planktonic state, the bacteria are metabolically active and vulnerable to the effects of the patient's immune system and antimicrobial treatments.

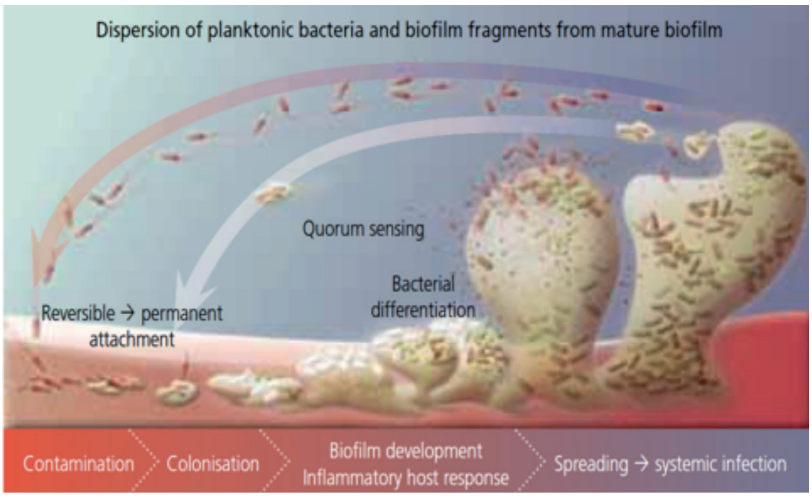


Figure 8. Biofilm formation and maturation (Phillips et al, 2010)

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FURTHER READING

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