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Foreword

The aim and mission of the International Wound Infection Institute (IWII) is to provide accessible, evidence-based information to support the prevention, identification and management of wound infection.

This fourth edition of *Wound Infection in Clinical Practice* was developed by members of the IWII Board and Committee, supported by our medical writer and methodologist, Dr Emily Haesler, Chair of the IWII Education and Evidence Subcommittee. Its development included a systematic review of the literature, a Delphi consensus process, external peer review and the integration of relevant IWII publications. This rigorous process was undertaken to ensure that the document reflects contemporary evidence, international expert consensus and practical clinical experience.

Our international and multidisciplinary author group includes academics, scientists, nurses, medical practitioners and pharmacists. The document was also reviewed by experts from a range of disciplines, including podiatry, infectious diseases, surgery and experts in paediatrics. This breadth of expertise reflects the importance of collaborative and multidisciplinary approaches to the prevention and management of wound infection.

IWII is a volunteer organisation committed to making wound infection education accessible worldwide. Our publications are free to download and are translated into multiple languages so that the information can be widely shared and implemented in everyday practice. This document is intended to support specialist clinicians in education, advanced clinical practice, and the development of policies and procedures. It is also designed to provide generalist clinicians with current information presented in clear, understandable language, together with practical and informative recommendations.

We have listened to feedback received following previous editions and during the development of this document. New and expanded content includes neonatal and paediatric considerations, the assessment of clinical signs across diverse skin tones, evolving concepts and paradigms, and medicated and non-medicated approaches to wound infection management. Evidence published since the third edition in 2022 has further informed our understanding of wound infection, biofilm, prevention, assessment and management.

We encourage readers to implement and evaluate the guidance presented in this document and to share their experiences with IWII, professional organisations and the wider wound care community. We also invite you to support the IWII mission by becoming a member and participating in our newsletters, publications, educational activities and free webinars.

Terry Swanson, Chair, International Wound Infection Institute
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Summary of the recommendations

1. Implement and support an organisational-level antimicrobial stewardship program to provide guidance, monitoring and education on antimicrobial use in wound care.
2. Establish a therapeutic relationship with the individual and their carers to support holistic wound infection assessment, shared decision making and a person-centred approach to preventing and treating wound infection.
3. Assess the individual's risk for wound infection and develop a management plan to address modifiable risk factors.
4. Identify wound infection based on clinical signs and symptoms. When indicated, use microbiological investigations to identify causative microbes and/or perform haematological and radiological investigations to assess severity of infection.
5. Select tissue biopsy as the preferred method to collect a wound specimen for diagnostic investigations. When tissue biopsy is not available, perform a wound swab using Levine technique.
6. Select either sterile/surgical aseptic technique or clean/standard aseptic technique when performing a wound dressing procedure. Conduct a risk assessment that considers the individual, the wound and environmental considerations to guide technique selection.
7. Implement standard precautions when conducting a wound dressing procedure.
8. Therapeutically cleanse the wound bed and wound edge, the periwound skin and the surrounding skin when the wound dressing procedure is performed.
9. Perform debridement when clinically indicated. The method and frequency of debridement should be selected based on the individual's clinical history and risk factors, assessment of the wound, goals of care, clinician's scope of practice, individual preferences and available resources.
10. Use an antiseptic as part of a comprehensive wound infection management plan when wound infection is confirmed or suspected. Consider using an antiseptic in individuals at high risk for invasive wound infection based on a risk assessment.
11. Select an antiseptic based on:
    - Antimicrobial spectrum and resistance,
    - Properties of the active ingredient,
    - Cytotoxicity, pH and allergenicity,
    - Goals of care and other individual factors, and
    - Resources, availability and organisation/wound care service policies.
12. Commence systemic antibiotic therapy urgently when there are signs and symptoms of systemic or spreading wound infection. Perform a risk assessment and use clinical judgement to determine whether commencement of systemic antibiotic therapy is appropriate for treating signs and symptoms of overt local wound infection.
13. Use an age-related approach to identify and mitigate risk for wound infection, to identify and assess wound infection and to select appropriate wound infection treatments in neonates, infants and children.

Chapter 1

Supporting best practice in wound infection

Supporting best practice in wound infection requires a deep understanding of the individual, the wound, the microorganisms present, and the environment in which care is delivered. Prevention, early identification and timely intervention depend on coordinated, evidence-based practice and effective communication between the individual, their carers and the multidisciplinary team.

Wound infection is influenced by the complex interaction of individual, microbial and environmental factors. Best practice therefore requires holistic assessment, clinical reasoning, accurate diagnosis and ongoing evaluation. While advances in research continue to improve our understanding of wound infection, the fundamental principles remain unchanged—prevent infection whenever possible, recognise clinical deterioration early, address the underlying cause, and provide timely, appropriate interventions that meet the needs of the individual.

The individual with the wound is the central member of the healthcare team. Their experiences, concerns, goals and preferences should guide shared decision-making. No single healthcare professional possesses all the knowledge and skills required to prevent and manage wound infection. Optimal outcomes are achieved when clinicians work collaboratively, recognising the contribution of each discipline and communicating effectively across healthcare settings.

This document has been developed to support clinicians with varying levels of wound care knowledge and experience. Specialist clinicians **[see Figure 1]** may use it to support advanced practice, education and the development of policies and clinical pathways, while generalist clinicians may use it as a practical guide to evidence-based prevention, identification and management of wound infection.

Effective team communication, timely referral and coordinated care are fundamental to best practice. Every clinician has a responsibility to recognise deterioration, practise antimicrobial stewardship, and seek additional expertise when assessment or management is beyond their scope of practice. By working together and placing the individual at the centre of care, clinicians can improve outcomes, reduce avoidable complications and contribute to preserving the effectiveness of antimicrobial therapies for future generations.

Principles of best practice

Best practice in wound infection includes:

- Placing the individual and their priorities at the centre of care,
- Undertaking a holistic assessment of the individual, wound and care environment,
- Identifying and addressing factors that increase infection risk or delay healing,
- Establishing the wound aetiology and an accurate clinical diagnosis,
- Recognising subtle/covert and overt signs and symptoms of infection,
- Intervening early when deterioration is observed or an increasing microbial burden is suspected,
- Implementing appropriate wound bed preparation, therapeutic wound and skin cleansing and debridement (as needed),
- Selecting medicated and non-medicated interventions according to clinical need,
- Integrating antimicrobial stewardship throughout prevention and management,

- Monitoring the response to treatment and modifying the plan when expected outcomes are not achieved, and
- Referring or escalating promptly when specialist intervention or a higher level of care is required.

The chapters that follow build on these principles, providing evidence-based guidance on the prevention, identification and management of wound infection across diverse healthcare settings. Together, they offer a practical framework to support consistent, person-centred care and improve outcomes for individuals with wounds.

Figure 1. The role of health disciplines in collaborative, person-centred wound infection care

Chapter 2
Antimicrobial resistance and stewardship

2026 updates and key points

- Antimicrobial stewardship is essential in wound care to minimise unnecessary antimicrobial exposure, reduce the selection and spread of antimicrobial resistance and maintain effective treatment options.
- Antimicrobials should be used only when clinically indicated, alongside accurate diagnosis, appropriate source control and optimisation of host factors and the wound environment through measures such as cleansing, debridement and exudate management.
- Effective stewardship requires coordinated action, including evidence-based prescribing and antiseptic use, regular review and monitoring, multidisciplinary antimicrobial stewardship programs, surveillance, and ongoing education of healthcare professionals.

Introduction

Antimicrobials is an all-encompassing umbrella term and is not synonymous with antibiotics. An antimicrobial agent is any natural, semi-synthetic, or synthetic substance that kills or inhibits the growth or activity of microorganisms. Antimicrobial agents comprise a broad group that includes antibiotics (antibacterials), antivirals, antifungals, and antiparasitic drugs, as well as antiseptics (solutions and dressings),[2] as illustrated in **Figure 2**. In addition, there are products and devices with antimicrobial effect that are registered for use in wound infection management.

Antimicrobial agents have transformed modern medicine by enabling effective treatment of infectious diseases and substantially reducing infection-related morbidity and mortality. In wound care, antimicrobial agents are an important component of infection management when used appropriately and alongside optimisation of host defences and the wound environment. This includes measures such as therapeutic wound cleansing, debridement and exudate management. Together, these strategies aim to reduce microbial burden, support wound healing and limit progression along the International Wound Infection Institute's Wound Infection Continuum (IWII-WIC) from contamination and colonisation through local infection to spreading or systemic infection and sepsis **[see Figure 5: IWII-WIC]**.

What is antimicrobial resistance, tolerance and persistence

Antimicrobial resistance is the ability of a microorganism to grow in the presence of an antimicrobial agent at concentrations that would normally inhibit or kill susceptible organisms. Resistance may be intrinsic or acquired through genetic mutation or horizontal acquisition of resistance determinants and is typically associated with an increase in the minimum inhibitory concentration (MIC). Acquired resistance may arise *de novo* through spontaneous genetic mutations or via acquired resistance mechanisms under selective pressure from antimicrobial use.[3] Infections caused by resistant microorganisms may be more difficult to treat and are associated with treatment failure, prolonged infection, delayed healing, and poorer clinical outcomes.[4] Microorganisms resistant to multiple classes of antimicrobials are referred to as multidrug-resistant (MDR) organisms.

Antimicrobial tolerance describes the ability of a microbial population to survive transient exposure to an antimicrobial agent without a corresponding increase in the MIC. In contrast to resistance, tolerant organisms remain susceptible according to conventional MIC testing, but demonstrate reduced or delayed killing by the antimicrobial. Tolerance may arise through physiological adaptations including reduced metabolic activity and stress responses, as might be the case in microorganisms growing within biofilms.

Persistence is a form of phenotypic survival of a subpopulation of microorganisms that survive antimicrobial exposure despite being genetically susceptible. Following removal of antimicrobial pressure, these surviving microbes resume growth and produce a population that remains susceptible to the antimicrobial.

Bacterial resistance occurs principally through four molecular mechanisms:[5]

- Enzymatic inactivation or degradation of the antimicrobial agent.
- Modification of the target of the antimicrobial agent reducing binding or antimicrobial activity.

- Reduced intracellular accumulation through decreased cell permeability.
- Active efflux, whereby the antimicrobial agent is transported out of the bacterial cell.

As outlined in **Figure 2**, AMR is widespread mainly following antibiotic treatment, but also antifungal and antiparasitic treatments. The principles of appropriate use of antimicrobial agents and microbial-binding dressings in the prevention and management of wound infection are discussed in Chapter 10.

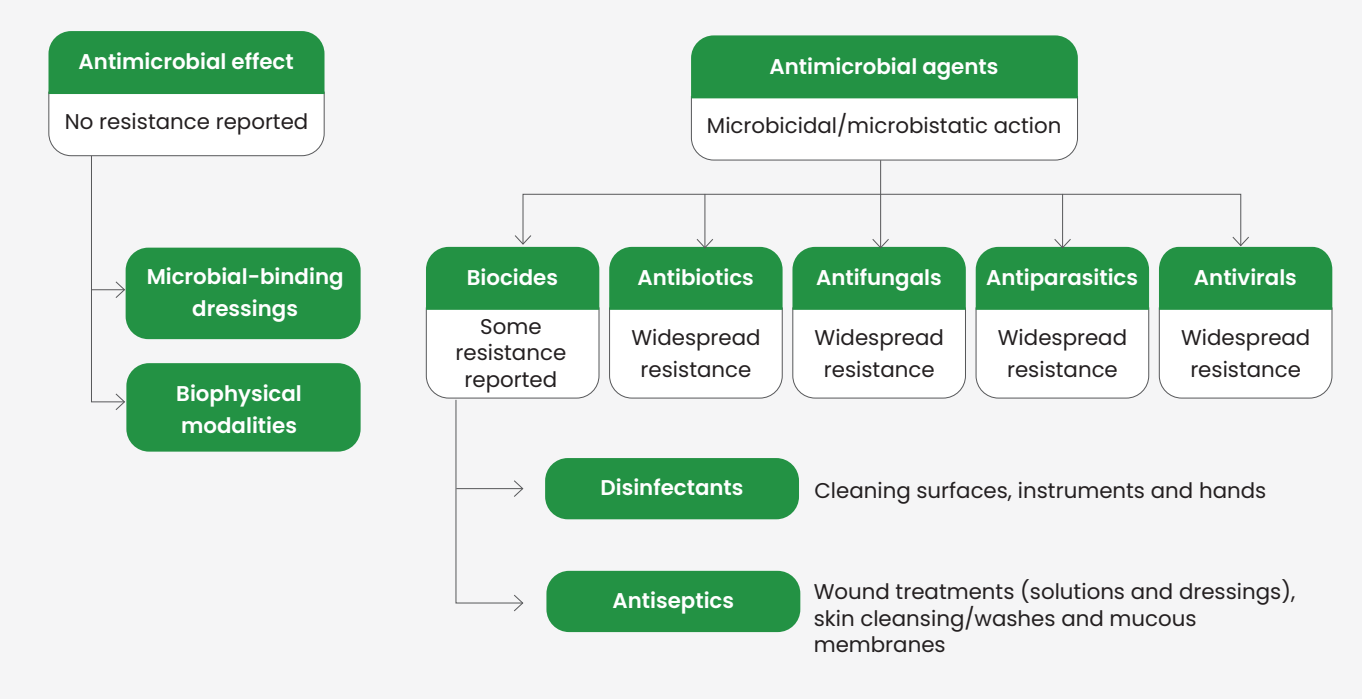


Figure 2. Classification of antimicrobial agents[6, 7] (adapted from Idensohn et al., 2026[8] with permission)

Antimicrobial resistance is driven by a range of social and economic factors, including:[4, 9]

- Overuse of antimicrobial agents in humans and food-producing animals,
- Using antimicrobial agents inappropriately,
- Inadequate prevention and control of infection and disease in all settings, but particularly in large facilities (e.g., healthcare and farms),
- Inadequate access to affordable, quality medicines, vaccines and diagnostics,
- Lack of access to clean water, sanitation and hygiene,
- Lack of awareness and knowledge regarding antimicrobial agents and their use,
- Inadequate enforcement of legislation, and
- Limited access to timely and appropriate diagnostics.

Despite progress in some settings,[10] AMR is developing more rapidly than new antimicrobial agents are becoming available. The global burden of AMR has been projected to reach up to 10 million deaths annually by 2050.[11] AMR also imposes a substantial economic burden through increased healthcare utilisation, prolonged hospitalisation, more complex treatment requirements and additional healthcare costs worldwide.

Antimicrobial resistance in the context of wound infection and treatment

The decision to use antimicrobial agents to prevent or treat wound infection should consider multiple clinical factors, including the potential contribution of antimicrobial use to the development and spread of AMR [see Chapter 10]. In chronic wounds, microorganisms frequently exist within biofilms, where they demonstrate increased tolerance to antimicrobial agents and host immune responses. Furthermore, the close proximity of microorganisms within biofilms facilitates horizontal gene transfer between bacteria, enabling the exchange of resistance genes, thereby accelerating the spread of AMR in the wound environment.[12]

Inappropriate use of antimicrobial agents is a major driver of the emergence and dissemination of antimicrobial resistant pathogens within both hospital settings and the wider community. For example, it is estimated that 30–50% of antibiotics prescribed in hospitals are unnecessary or inappropriate.[13] The problem is further compounded by the non-prescription supply of antimicrobial agents, which has been identified as a significant concern in developing countries.[14] Factors contributing to this practice include inadequate national medicines regulation, limited access to qualified pharmacists, commercial pressures on pharmacy staff, consumer demand, inappropriate prescribing practices and limited awareness of AMR.[14]

Mounting evidence suggests that the pattern of excessive use of topical and systemic antibiotics is an issue in the context of wound management.[15] Individuals with chronic/hard-to-heal wounds are more likely to receive antibiotics compared to people who do not have a chronic/hard-to-heal wound.[15] Given the high rate of chronic/hard-to-heal wound recurrence, repeated antibiotic treatment may result in substantial cumulative lifetime exposure, creating selective pressure within microbial populations and increasing the likelihood of AMR. Antibiotics are also frequently prescribed for individuals with non-healing wounds without clear clinical justification, without adequately addressing the underlying wound aetiology,[16–18] and with limited evidence of clinical benefit.[19] These findings indicate considerable scope to reduce unnecessary antibiotic use in wound care. A meta-analysis examining prophylactic topical antibiotics for acute, uncomplicated wounds found that, although topical antibiotics reduced surgical site infection (SSI) compared with placebo or no treatment, the absolute benefit was small.[19] Other evidence and antimicrobial stewardship (AMS) guidance caution against routine topical antibiotic use because of limited additional benefit and the potential to select for resistant organisms. Topical antibiotics should not be routinely used for the prevention or management of wound infection and should be reserved for specific clinical indications where there is a clear evidence-based rationale. Where topical antimicrobial treatment is indicated, an appropriate antiseptic should generally be preferred to a topical antibiotic.

Systemic antibiotics are not routinely indicated for all localised wound infection. Inappropriate antibiotic selection or use in wound care may contribute to AMR by creating sustained selective pressure, particularly when therapeutic concentrations at the wound site are inadequate. This may occur because biofilm-associated microorganisms exhibit reduced susceptibility to antimicrobial killing through multiple mechanisms, including antimicrobial tolerance, persistence and altered metabolic activity, thereby limiting the effectiveness of systemic antibiotics.[20, 21] Effective delivery also depends on an adequate vascular supply, which may be compromised in individuals with chronic/hard-to-heal wounds.[22] Systemic antibiotic therapy should therefore be individualised according to the individual's clinical presentation and supported by appropriate diagnostic investigations where indicated. Antibiotics should be used only where there is a clear clinical indication, alongside adequate source control, with the individual's clinical response and need for ongoing therapy regularly reviewed.

Emerging evidence indicates that reduced susceptibility or resistance to some topical antiseptics (and disinfectants) can occur, although this appears to be uncommon. Resistance and potential cross-resistance with antibiotics have been reported particularly for some

agents, including chlorhexidine and triclosan.[23-25] These findings support the application of AMS principles to the use of antiseptics for wound care, and to the use of disinfectants in healthcare and more broadly. However, they should not discourage clinically appropriate antiseptic treatment. Antiseptics should be selected according to the individual and wound, used at an effective concentration for an appropriate duration, and reviewed regularly to avoid unnecessary or prolonged exposure.

Although silver has broad-spectrum antimicrobial activity and resistance appears less common than antibiotic resistance, silver resistance is a recognised concern. Silver resistance genes have been identified in bacteria isolated from wounds, although their presence does not necessarily confer complete phenotypic resistance to silver-containing dressings. Increasing and prolonged exposure to silver may provide selective pressure favouring resistant organisms. For this reason, silver dressings should not be used indefinitely or as routine prophylaxis. Their use should be based on a clear clinical indication, reviewed regularly, and discontinued when signs of local infection or increased microbial burden have resolved.[26]

The rising prevalence of antifungal resistance[4, 27, 28] should prompt clinicians to remain alert to its potential relevance in wound care,[4] particularly in individuals at high risk of wound contamination with fungal elements (e.g., those with tinea pedis or onychomycosis).[29, 30] Fungi can form biofilms within chronic wounds, which may reduce susceptibility to antifungal treatment and contribute to persistence of infection.[31] Fungal communities may also interact with bacterial populations within polymicrobial biofilms, potentially increasing treatment complexity and supporting persistent microbial reservoirs.[32] Antifungal resistance may arise through mechanisms including genetic mutation and selection pressure associated with antifungal exposure. The widespread use of broad-spectrum antifungal agents, including triazoles in human medicine and agriculture, has contributed to increasing global concern regarding antifungal resistance.[33, 34] The clinical significance and prevalence of antifungal resistance in wounds remain incompletely characterised. Antifungal resistance should be considered particularly in high-risk individuals, including those with diabetes, recurrent or persistent infection, repeated antimicrobial exposure, immunocompromise, or failure to respond to appropriate therapy.

More judicious antibiotic and antifungal use in wound practice will contribute significantly to a reduction in antibiotic and antifungal resistance and reduce both the poor health outcomes and economic burden associated with AMR. Reviewing wound care practice and aligning wound infection prevention and management with the goals and principles of AMS is essential to addressing the global problem of AMR.[35]

What is antimicrobial stewardship?

Antimicrobial stewardship is a multidisciplinary strategy that promotes targeted and responsible use of antimicrobial agents (particularly antibiotics) through appropriate diagnosis, antimicrobial selection, dosing optimisation, treatment monitoring, and de-escalation or discontinuation when indicated. The objectives are focused on improving clinical outcomes, reducing adverse effects, preserving antimicrobial effectiveness, and preventing the emergence and spread of AMR.[36, 37]

While escalating emergence of AMR involving a widening range of pathogens is stimulating renewed research and development into alternative infection management strategies[38] [see Chapter 14], there is an ongoing need at international, national, organisational, professional and general public levels to implement strategies to reduce the risk of AMR. In recognition of the severity of this challenge, several organisations develop and review policies and strategies to combat AMR. This includes the World Health Organization (WHO), the Centres for Disease Control and Prevention (CDC), and the European Centre for Disease Prevention and Control (ECDC). In particular, the WHO has established a comprehensive global strategy.[39]

In healthcare settings, AMS is implemented through locally agreed, implemented and coordinated programs designed to decrease the spread of infections caused by multidrug-resistant organisms, and improve clinical outcomes by encouraging appropriate and optimised use of all antimicrobials.[40] Antimicrobial stewardship programs (AMSPs) exert

The World Health Organization AWaRe (Access, Watch, Reserve) program[1] provides recommendations for antimicrobial stewardship management programs on selecting antibiotic regimens in primary care and hospital settings.

a significant and measurable influence on the management of infection (including wound infection).[41] The aim of AMSPs is to reduce unnecessary antimicrobial use,[36, 37] and inappropriate prescribing, and to promote access to the most appropriate treatment. Formal AMSPs emphasise accurate diagnosis, appropriate antimicrobial selection, preference for narrow-spectrum agents where suitable, optimisation of route, dose and duration of therapy, and regular review of treatment response and prescribing practice. Increasingly, AMS in wound care also encompasses the appropriate selection, duration and monitoring of topical antiseptic use.[42, 43] By promoting judicious, evidence-based antimicrobial use, AMSPs can help reduce selective pressure for resistance, improve clinical outcomes and minimise avoidable healthcare costs associated with inappropriate antimicrobial treatment.[44]

Antimicrobial stewardship in preventing and managing wound infection

Given the identified issues of AMR associated with wound care, the imperative to address AMS in a coordinated and collaborative manner in all wound care services is evident.

Recommendation 1

Implement and support an organisational-level antimicrobial stewardship program to provide guidance, monitoring and education on antimicrobial use in wound care.

(Underpinning evidence: Level 1 evidence[39, 45, 46])

Evidence shows that AMSPs can improve antimicrobial use across a range of healthcare settings.[45, 46] A large systematic review and meta-analysis[45] found that implementation of AMSPs in tertiary hospitals, primary care and residential care was associated with a 10% reduction in overall antibiotic prescribing (95% confidence interval [CI] 4% to 15%; 17 studies) and a 28% reduction in antibiotic consumption (95% CI 8% to 44%; 34 studies). Decision-support interventions produced some of the strongest effects.[45] A systematic review of 55 studies conducted in intensive care settings similarly found that AMSPs were associated with reduced antibiotic use, shorter treatment duration and lower antimicrobial costs.[46] Effective approaches included multicomponent stewardship programs, prospective audit and feedback, procalcitonin-guided protocols,[47] prescribing and de-escalation guidelines, and diagnostic stewardship.[46]

Specific to wound management, a comprehensive scoping review[39] identified several wound-focused studies[48, 49] showing that AMSPs can reduce antibiotic use while maintaining or improving important clinical outcomes. Before-and-after evaluations in surgical settings reported reductions in surgical site infection (SSI) rates of up to 19.7% following AMSP implementation.[48] In a wound-specific service, implementation of an AMSP was associated with an approximate 40% reduction in escalation of antimicrobial therapy, from 63% before implementation to 37% afterwards, without an associated increase in length of stay or all-cause mortality among individuals with chronic/hard-to-heal wounds.[50] Wound-focused AMSPs described in the review[39] included pharmacist-led stewardship with audit and feedback,[48] antibiotic treatment guidelines[50] and selective reporting protocols for wound culture results.[50]

Effective antimicrobial stewardship initiatives

If AMS guidelines are standardised and routinely integrated into clinical practice through coordinated AMSPs, they have the potential to reduce antimicrobial use and simultaneously improve wound management outcomes.[39] Several specific strategies may be beneficial to support wider adoption of AMS principles within chronic wound management.[39] These include strengthening health professionals' education and training on AMS concepts, conducting regular audits coupled with constructive feedback, and fostering greater professional ownership of stewardship initiatives.[39] **Table 1** provides a comprehensive overview of initiatives at the government, organisational and clinical level that have been identified as important components of multi-intervention AMSPs.

Facility level antimicrobial stewardship initiatives

Organisation/facility-based guidelines, drug formularies and clinical decision pathways should be based on national and international guidance and facility-level data (e.g. microbial sensitivity patterns) to support and facilitate implementation of AMS by clinicians managing wound infection.[1, 7, 51–54] Laboratory based and clinical investigations into AMR patterns in microorganisms provide critical evidence that can inform the refinement of a facility-level AMSP, supporting efforts to minimise the development and spread of AMR.[55]

Monitoring and clinical auditing of the use of antimicrobial agents underpins evaluation of AMSPs and informs quality improvement in managing wound infection. To ensure a multidisciplinary and multifaceted approach to AMS, an overarching committee responsible for auditing, monitoring and providing feedback on facility-level antimicrobial use should be a critical focus for health services that provide wound care.[39, 46, 51] The active involvement of nurses in AMSPs has been consistently emphasised in the literature,[41, 58] highlighting the importance of ensuring their involvement in facility-level monitoring initiatives.

Table 1: Antimicrobial stewardship initiatives[1, 7, 51–57]

Government level initiatives
• Promote global regulation of prescription and supply of antimicrobial agents. • Support global initiatives focused on reducing AMR. • Promote awareness of AMR across the human health, animal health and public sectors. • Support and stimulate ongoing research on AMR and development of new antimicrobial agents.
Organisation/health service level initiatives
• Provide adequate funding and resources to support AMS. • Provide regular education to all stakeholders on AMR and AMS. • Develop institutional policies and procedures on use of antimicrobial agents in wound care based on global guidance. • Implement best clinical practice in wound infection prevention and treatment. • Facilitate accurate diagnosis of wound infection through appropriate policies, resources and care pathways. • Convene a multidisciplinary AMS committee responsible for optimising antimicrobial use. Membership could include infectious diseases specialists, clinical microbiologists, pharmacists, infection prevention specialists, surgeons and clinicians from high-use specialties, nurses and wound care specialists. • Monitor facility-level trends in microbial susceptibility and adapt prescribing guidelines accordingly. • Audit antimicrobial prescribing and patterns of use, including indication, wound type, agent selection, route, dose and duration of treatment. • Monitor and publish incidence of wound infection, patterns of antimicrobial agent use and their effectiveness.
Clinical level initiatives
• Optimise therapeutic cleansing, debridement and exudate management. • Consider using products and devices with antimicrobial effect that are registered for use in wound infection management. • Use antimicrobial agents to treat wounds exhibiting clinical signs and symptoms of infection. Only consider prophylactic antimicrobial therapy if a wound is at very high risk of infection. • Select empirical antibiotic therapy based on the likely pathogenic profile and with narrow spectrum activity where possible. Reserve broad-spectrum antibiotics for more resistant bacterial infections. • Continue antibiotic treatment for an appropriate duration to prevent development of resistance. • Monitor therapeutic response to guide ongoing selection and use of wound infection treatments. • Review and (when appropriate) delist allergies and sensitivities.

Antimicrobial stewardship compliant wound care

As noted in the clinical-level initiatives in **Table 1**, clinicians' decisions in the delivery of wound care are central to effective AMS. This document provides guidance on clinical management strategies that support AMS practice, including:

- Holistic assessment of the individual [see Chapter 3 and Chapter 13],
- Comprehensive wound assessment [see Chapter 5],
- Accurate diagnosis of wound infection [see Chapter 6],
- Wound bed preparation and management to promote an environment less conducive to microbial proliferation [see Chapter 9], and
- Appropriate selection and/or prescribing of antimicrobial agents and products and devices with antimicrobial effect that are registered for use in wound infection management [see Chapter 10 and Chapter 11].

Embed the principles of antimicrobial stewardship in the context of wound care into the curriculum of health science programs.

Comprehensive and ongoing education is an important component of AMS, equipping health professionals with the knowledge and skills to contribute to facility-level stewardship initiatives, provide wound care consistent with AMS principles, and educate individuals with wounds and their families about AMR.[52] Integrating wound care into medical, nursing and other relevant health science curricula may strengthen knowledge of wound assessment, prevention, infection management, tissue repair, evidence-based wound care and wound-focused AMR, while also supporting multidisciplinary collaboration and improved patient outcomes.[59] These principles should also be reinforced through continuing professional development. Advanced wound care education, including postgraduate certification and specialist training, should be encouraged to further develop clinical expertise.[60]

Chapter 3
Holistic assessment and management

2026 updates and key points

- Establish a therapeutic relationship with the individual to facilitate a holistic approach to assessment and management of individuals at risk of, or with wound infection.
- Work collaboratively with the individual and their support network to establish wound infection goals and the acceptable management strategies to achieve those goals.
- Use a holistic approach in assessing the individual and the full range of factors that influence their wound, their risk of infection and their healing ability. Person-centred wound assessment and management models facilitate holistic wound care.
- A person-centred approach to managing wound infection focuses on optimising the wound, factors related to the individual's ability to heal, and the local environment in which the wound is being treated.

Introduction

Wound infections prolong the inflammatory response and stall or reverse the healing process,[61, 62] impacting individuals, care providers, healthcare systems and society. The individual's immune defences are a key determinant of whether microbial burden progresses to clinical infection. In turn, wound infection can adversely affect physical, psychological and social functioning and reduce quality of life.[63, 64] Promoting the individual's overall health, immune function and wellbeing is therefore integral to both the prevention and management of wound infection. A person-centred approach that reflects the individual's needs, priorities, preferences and circumstances is fundamental to achieving optimal outcomes.

The goal of holistic care in wound infection is to shift the interaction between the individual and infecting microorganisms in favour of the individual by:

- Identifying and addressing systemic, local, psychosocial and environmental factors that may increase susceptibility to infection or delay its resolution,
- Establishing realistic and achievable care goals and treatment options that are acceptable to the individual and, where appropriate, their nominated support network, and
- Developing a comprehensive, individualised plan for the prevention and management of wound infection that aligns with the individual's preferences, priorities and overall goals of care.

Establishing a therapeutic relationship with the individual

A fundamental principle of holistic assessment and management is active engagement of the individual and their support network in the care process.[68, 69] Central to this is the development of a therapeutic relationship (also referred to as a therapeutic alliance): a collaborative, trusting and respectful partnership between the healthcare professional and the individual receiving care. Establishing this relationship is a core component of person-centred care.[70] Arising from psychology but recognised across health disciplines, a substantial body of evidence indicates that establishing a therapeutic relationship between an individual and their healthcare provider is associated with enhanced clinical outcomes. This is because such a relationship underpins a person-centred approach that drives holistic assessment and care delivery. An effective therapeutic relationship consists of:[71, 72]

- A constructive partnership between the individual and their wound care clinician,
- Collaborative goal setting, and
- Mutual agreement on the required interventions to reach the goals.

In the context of healthcare delivery, a therapeutic relationship is characterised by a warm and empathetic approach, mutual trust and respect, constructive communication and negotiation.[65, 67] Establishing this type of partnership can promote feelings of safety and support, psychological wellbeing and empowerment.[54, 66, 73] This foundation is key to the other components of the therapeutic relationship in which the individual actively contributes to goal setting and agrees on a plan to achieve those goals.[70]

Recommendation 2

Establish a therapeutic relationship with the individual and their carers to support holistic wound infection assessment, shared decision making and a person-centred approach to preventing and treating wound infection.

(Underpinning evidence: Level 1 evidence,[65, 66] Level 3 evidence[67] and Level 5 evidence[68-70])

Ask. Listen. Understand the impact of the wound on quality of life and wellbeing.

Specific to wound care, a therapeutic relationship has been associated with improved adherence to wound care, enhanced psychological resilience, decreased pain perception and increased engagement of the individual in decision-making and satisfaction with care, enhanced self-care skills, and improved clinical outcomes.[67-69] The therapeutic relationship is particularly significant in contexts in which individuals and their support networks are likely to experience high levels of emotional distress such as palliative wound care,[73] paediatric settings, and intensive care. However, the benefits of therapeutic relationships are evident in all wound care contexts, including community-based care.[72]

Holistic assessment of the individual with or at risk of wound infection

Numerous models are available to guide a whole-person, holistic approach to wound assessment **[Table 2]**. Some of the frameworks extend to person-centred wound care planning. Designed to support therapeutic relationships and person-centred approaches to wound care, these models can be used when working with individuals with wounds at any stage on the wound infection continuum **[Figure 5: IWII-WIC]**.

Table 2: Person-centred wound assessment and management models

Care model	Model aims	Key model features
Wounds UK Best Practice Statement on improving holistic assessment[68]	Encourage wide-ranging assessment that considers the impact of all aspects of the person's health and wellbeing on the healing process.	Each best practice statement is emphasised by an accompanying 'Patient Expectation' that indicates what people with wounds can expect in their care.
The Infection Management Pathway[74] incorporating the T.I.M.E. Clinical Decision Support Tool[75]	• Promotes comprehensive assessment and care continuity. • Facilitates clinical decision making and best practice among non-wound care specialists. • Supports antimicrobial stewardship.	Uses the mnemonic A-B-C-D-E: • **A**ssess the person, and their wound. • **B**ring in a multi-disciplinary team. • **C**ontrol underlying barriers to healing. • **D**ecide appropriate treatment. • **E**valuate outcomes and reassess goals.
The Adult Burns Patient Concerns Inventory[76]	Improves clinician–patient–family communication and empowers patients to identify concerns, supporting targeted, patient-centred consultations.	58-item holistic outpatient assessment tool Domains include: • Physical and functional wellbeing • Psychological, emotional, and spiritual wellbeing • Social care and social wellbeing • Treatment-related concerns
Wound Healing Strategies to Improve Palliation[77]	Provides a palliative approach to assessment and ongoing care review when wound healing is not achievable.	When complete healing is not feasible, use the mnemonic S-P-E-C-I-A-L: • **S**tabilising the wound • **P**reventing new wounds • **E**liminate odour • **C**ontrol pain • **I**nfection prophylaxis • **A**dvanced, absorbent wound dressings • **L**essen dressing change
Universal Model for the Team Approach to Wound Care[78]	Promotes patient advocacy and development of management plans that reflect patient goals, perceived needs, and appropriate services.	• Multidisciplinary wound care framework • Person with a wound remains the focus • Wound navigator coordinates care and referrals • Multidisciplinary team identifies healthcare options aligned with patient needs
TIMERS: expanding wound care beyond the focus of the wound[79]	Provides a 10-step pathway for wound management, including maintenance-focused care for palliative wounds.	• **T**issue (non-viable or deficient) • **I**nfection/inflammation • **M**oisture imbalance • **E**dge of wound (non-advancing or undermined) • **R**egeneration/repair of tissue • **S**ocial factors affecting wound healing trajectory.

Table 2: Person-centred wound assessment and management models (Continued)

Care model	Model aims	Key model features
Wound Balance[70, 80]	Expands the TIMERS framework by emphasising balance among the patient, wound, and care practices.	• Patient balance • Wound balance • Care practice balance
Wound Bed Preparation 2021[81]	Facilitates person-centred wound assessment and establishes goals of healing, maintenance, or palliation.	• Treatment of the cause • Address patient-centred concerns • Regularly assess ability to heal • Local wound care, and appropriate debridement with pain control • Assess and manage wound infection • Moisture management • Evaluate the rate of healing • Assess edge effect • Ensure organisational support

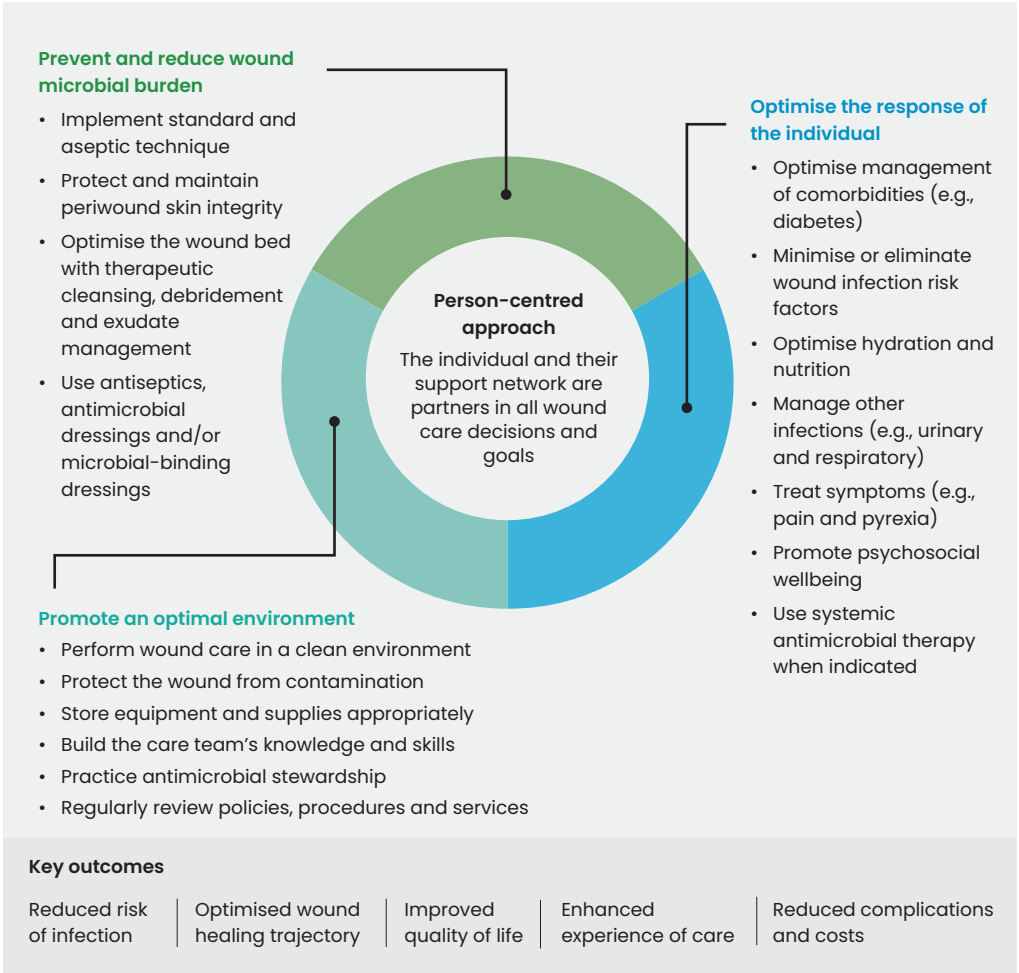
In addition to a comprehensive clinical assessment of the wound [see Chapter 5], a host of other factors that contribute to the individual's experience of wound infection should be comprehensively assessed. These factors are often the same factors that contributed to the development of the initial wound and include:[68, 70, 82-84]

- The history of the individual and their wound.
- Comorbidities and their management.
- Nutritional status.
- Factors that influence the inflammatory and immune response.
- Factors that influence local tissue healing.
- Psychosocial factors and wellbeing.
- Access to resources, education and support.

Understanding the impact of each of these domains facilitates identification of factors of significance to the individual person and their wound.[85] **Figure 3** provides a mnemonic and framework for holistic assessment of a person with or at risk of a wound infection. **Figure 4** provides an overview of holistic management.

Figure 3. Whole person wound infection assessment[84] (Adapted with permission)

Figure 4. Holistic wound infection prevention and management



Chapter 4
Wounds at risk of infection

2026 updates and key points

- Risk of wound infection is influenced by characteristics of the individual, their wound, and the environment.
- Evaluation of a wound infection risk should be based on knowledge of risk factors and an individualised, holistic assessment.
- Strategies to prevent wound infection should address the individual's modifiable risk factors.
- Perioperative care bundles that included evidence-based interventions are effective for reducing rates of surgical site infections.
- Most of the research on wound infection risks and effectiveness of preventive strategies addresses surgical site infection. More studies on preventing wound infection are required.

Introduction

All open wounds contain microorganisms, but infection occurs only when the balance between host defences and the wound microbiota is disrupted. Microorganisms may then proliferate, express virulence factors, invade tissue and trigger a harmful inflammatory response. Progression to infection is influenced by host susceptibility, microbial burden and virulence, polymicrobial interactions and biofilm formation. Biofilms can promote persistent inflammation, delayed healing, reduced antimicrobial susceptibility and increased risk of wound infection.[61, 86, 87]

Factors associated with risk of wound infection

Assessment of wound infection risk requires a holistic evaluation of the individual, the wound and the care environment. Risk is multifactorial and reflects the interaction of host-related, wound-related and environmental factors. Host factors that influence the development of wound infection are systemic and multifactorial and encompass many variables. **Table 3** summarises the key factors associated with increased risk of wound infection.

Risk factors may also differ between acute and chronic wounds. For example, the risk of surgical site infection (SSI) is influenced by factors such as the type and duration of surgery, together with host and environmental factors.[88–91] Although much of the available evidence on wound infection risk is derived from studies of surgical site infection, many of the identified host and environmental risk factors are also relevant to other wound types.

Assessing wound infection risk

Assessment of an individual's risk of wound infection is important for identifying potentially modifiable factors and informing management decisions. For example, it may influence decisions on duration of antimicrobial treatment in an individual requiring treatment for wound infection. Clinical judgement should be based on an individualised assessment and knowledge of recognised host, wound and environmental risk factors.[115]

Formal risk assessment tools **[Table 4]** may complement clinical judgement, but most focus on surgical site infection, often within specific surgical populations. Many of the formal risk assessment tools do not comprehensively assess host, wound and environmental factors, and their psychometric properties are often insufficiently established. Consequently, these tools should be used alongside, rather than in place of, comprehensive clinical assessment and professional judgement.

Recommendation 3

Assess the individual's risk for wound infection and develop a management plan to address modifiable risk factors.

(Underpinning evidence: Level 1 evidence[111–114])

Table 3: Factors associated with increased risk of wound infection

Individual (host) risk factors[86, 89, 92-108] (Also see Chapter 13 for additional factors in paediatric populations)

- Poorly controlled diabetes (i.e., hyperglycaemia)
- Peripheral neuropathy (sensory, motor and autonomic)
- Neuroarthropathy
- Radiation therapy or chemotherapy
- Conditions associated with hypoxia and/or poor tissue perfusion (e.g., anaemia, cardiac disease, respiratory disease, peripheral arterial disease, hypovolaemia, renal impairment or rheumatoid arthritis)
- Immune system disorders (e.g., acquired immune deficiency syndrome)
- Connective tissue disorders (e.g., Ehlers-Danlos syndrome)
- Immunosuppressant/corticosteroid use
- Malnutrition or obesity
- Serum albumin <35 g/L
- Frailty
- Alcohol, smoking or illicit drug use
- Poor adherence with treatment plan
- Inadequate personal hygiene

Wound risk factors[90, 91, 95, 98, 101, 106, 109, 110]

Acute wounds	Chronic wounds	Acute and chronic wounds
• Dirty wounds • Traumatic injuries • Operation is classified as dirty/ contaminated • Pre-procedural hair removal using a razor or other method that causes skin trauma • Operative factors (e.g., prolonged surgery, blood transfusion or induced hypothermia)	• Duration of wound • Large wounds • Anatomical location (e.g., near perineum or sacrum)	• Foreign body presence (e.g., drains, sutures or wound dressing fragments) • Haematoma • Necrotic or sloughy wound tissue • Impaired tissue perfusion • Increased exudate and oedema that is not adequately managed • Wounds over bony prominences or probing to bone • Involvement of tissue deeper than skin and subcutaneous tissues (e.g., tendon, muscle, joint or bone)

Environmental risk factors[101, 106, 107]

- Unhygienic environment (e.g., dust, unclean surfaces, or presence of mould/mildew)
- Hospitalisation (due to increased risk of exposure to antibiotic resistant microorganisms)
- Inadequate hand hygiene and aseptic technique
- Inadequate management of moisture (e.g., due to exudate, incontinence or perspiration)
- Interface pressure that is inadequately off-loaded

Preventing wound infection

Prevention of wound infection focuses on reducing modifiable risk factors through an individualised, holistic approach. Goals of care and an individualised management plan should be established collaboratively with the individual, their family and the healthcare team, with preventive strategies addressing relevant host, wound and environmental risks.

Most evidence on interventions to prevent wound infection comes from studies conducted in surgical settings and explores perioperative care bundles. Several systematic reviews[111-114] of studies at low to moderate risk of bias demonstrated the benefits of perioperative care bundles. In a comprehensive analysis of 26 studies exploring surgical bundles, a 45% reduction (95% confidence interval [CI] 27–59%) in SSI was demonstrated.[111] Reduction in SSI rates is also demonstrated in meta-analyses based on surgery types, including a 54% reduction (95% CI 8–78%, 14 studies) for cranial neurosurgery,[112] a 71% reduction (95% CI 69–83%, 21 studies) for caesarean sections,[113] a 47% reduction (95% CI, 30–60%, 14 studies) for colorectal surgery[111] and a 71% reduction (95 % CI 59–80%, 5 studies) for gynaecological surgery.[111] A review of bundles used in paediatric cardiac surgery reported a 30–70% reduction in SSI rates.[114]

Table 4: Sample of tools available to assess the risk of wound infection			
Risk assessment tool	**Wound type**	**Risk variables**	**Predictive power**
Australian Clinical Risk Index[116]	Surgical site infection following cardiac surgery	Includes diabetes and BMI as risk variables	Low in all types of cardiac patient[117]
Brompton and Harefield Infection score[118]	Surgical site infection following cardiac surgery	Includes gender, diabetes, BMI, cardiac function and emergency vs elective surgery status	Moderate[118]
Malunion of the Sternum score[119]	Surgical site infection following cardiac surgery	Includes age, gender, BMI, previous surgery and diabetes as risk variables	Moderate[119]
National Nosocomial Infections Surveillance Risk Index[120]	Surgical site infection in surgical wounds	Includes type of surgery, pre-anaesthetic score and surgery duration.	Low in all types of cardiac patient[117]
Perth Surgical Wound Dehiscence Risk Assessment Tool[121]	Wound dehiscence in surgical wounds	Includes comorbidities, smoking, previous surgery, age and BMI as risk variables	Moderate[121]
Wounds At Risk (WAR) score[122, 123]	All wounds	Comorbidities, medications, wound exposure to contamination sources, age, wound duration, wound aetiology, wound dimensions, wound anatomical location	Not reported
Wound Infection Calculator[124]	Postoperative wound infection following spinal surgery	Includes gender, BMI, smoking, physical status score, level of surgical invasiveness	High[124]
Wound Infection Risk Assessment and Evaluation tool[125]	Community-based wounds	Comorbidities, immune status, smoking, medications, nutrition, antibiotic therapy	Not reported

These findings are consistent across high income and middle-to-low income settings,[126] and reduction in wound infection rates was more likely when the bundle included evidence-based interventions.[127] Including more interventions in a bundle does not necessarily increase its effectiveness,[127] which is relevant because compliance influences the bundle's impact.[111] The World Health Organization (WHO) provides guidance on effective components to include in perioperative care bundles.[128]

Evidence does not support prolonged systemic antibiotic prophylaxis, and routine antiseptic use is generally not indicated unless there is an increased infection risk (e.g., pre-surgical antisepsis).[1, 8, 44, 126] Clinical decisions should balance antimicrobial stewardship with the individual's infection risk, clinical signs and symptoms, and response to strategies such as therapeutic cleansing, debridement, exudate management, and products and devices with antimicrobial effect that are registered for use in wound infection management.

Chapter 5
Identifying and assessing infection in a wound

2026 updates and key points

- The International Wound Infection Institute's Wound Infection Continuum (IWII-WIC) has been updated via a consensus process to include treatment guidance.
- The IWII-WIC is recognised as a conceptual framework of wound infection progression and the presentation of signs and symptoms indicative of wounds progressing along the dynamic continuum.
- Clinicians must have a comprehensive understanding of the signs and symptoms of wound infection to make a clinical diagnosis of infection and commence appropriate treatments.
- Clinicians must be cognisant of factors that can influence the presentation of wound infection and potentially mask or complicate the recognition and differentiation of wound infection.
- Wound assessment tools, including the IWII-WIC, can be used to facilitate and enhance clinical assessment, monitoring and documentation of wound infection.

Introduction

Wound infection occurs when microorganisms, in planktonic or aggregated forms, invade and proliferate within a wound to a level that triggers a local, spreading and/or systemic host response. As microorganisms multiply, they may express a range of virulence factors, including biofilm formation, that enable them to evade or overcome host defences. This can result in tissue damage, persistent inflammation and impaired wound healing.[129, 130] Although host immune defences usually control or eliminate invading microorganisms, this response may be inadequate when host immunity is compromised[131] or when microorganisms successfully evade host defence mechanisms. In these circumstances, intervention may be required to support host defences and reduce or eliminate the microbial burden.

The IWII Wound Infection Continuum

The IWII Wound Infection Continuum [IWII-WIC, see **Figure 5**] is a widely recognised educational tool that provides a conceptual framework for understanding the impact of microorganisms on the host, the wound and wound healing. The IWII-WIC was developed and subsequently revised through formal Delphi consensus processes involving IWII experts, informed by clinical wound presentation and contemporary scientific understanding of wound infection. The continuum illustrates the dynamic progression of microbial presence and its increasing impact on the host, from contamination and colonisation through local infection (covert and overt), to spreading and systemic infection.[132-134]

Stages of the IWII Wound Infection Continuum (IWII-WIC)

The stages of infection as currently presented on the IWII-WIC were agreed by wound infection experts in 2016 in a consensus process.[133, 135] Microbial burden exists on a dynamic continuum, with multiple host, wound and microbial factors influencing whether infection progresses and how readily signs and symptoms can be clinically recognised **[Table 5]**. Clinical interventions aim to prevent or halt progression along the continuum by reducing microbial burden and supporting host defences.

The IWII-WIC includes five conceptual stages and lists the signs and symptoms commonly exhibited by the individual and the wound as infection develops. Definitions for these five stages were agreed on in an international consensus process.[136] The IWII-WIC:

- Contamination,
- Colonisation,
- Local infection (covert/subtle and overt/classic),
- Spreading infection, and
- Systemic infection.

Contamination refers to a stage of microbial adhesion (i.e., reversible attachment) in which the microorganisms within the wound are presumed to not be proliferating (i.e., the microbial burden is in a dormant or lag phase of growth). No significant host reaction is evoked and no delay in wound healing is clinically observed.[136] The host defences destroy microorganisms through a process called phagocytosis.[137, 138]

Colonisation refers to a stage in which the microorganisms present within the wound are generally irreversibly attached and presumed to be undergoing limited proliferation. In a colonised wound, no significant host reaction is evoked, and no delay in wound healing is clinically observed.[136] Due to the protective function of the skin microbiome, all open

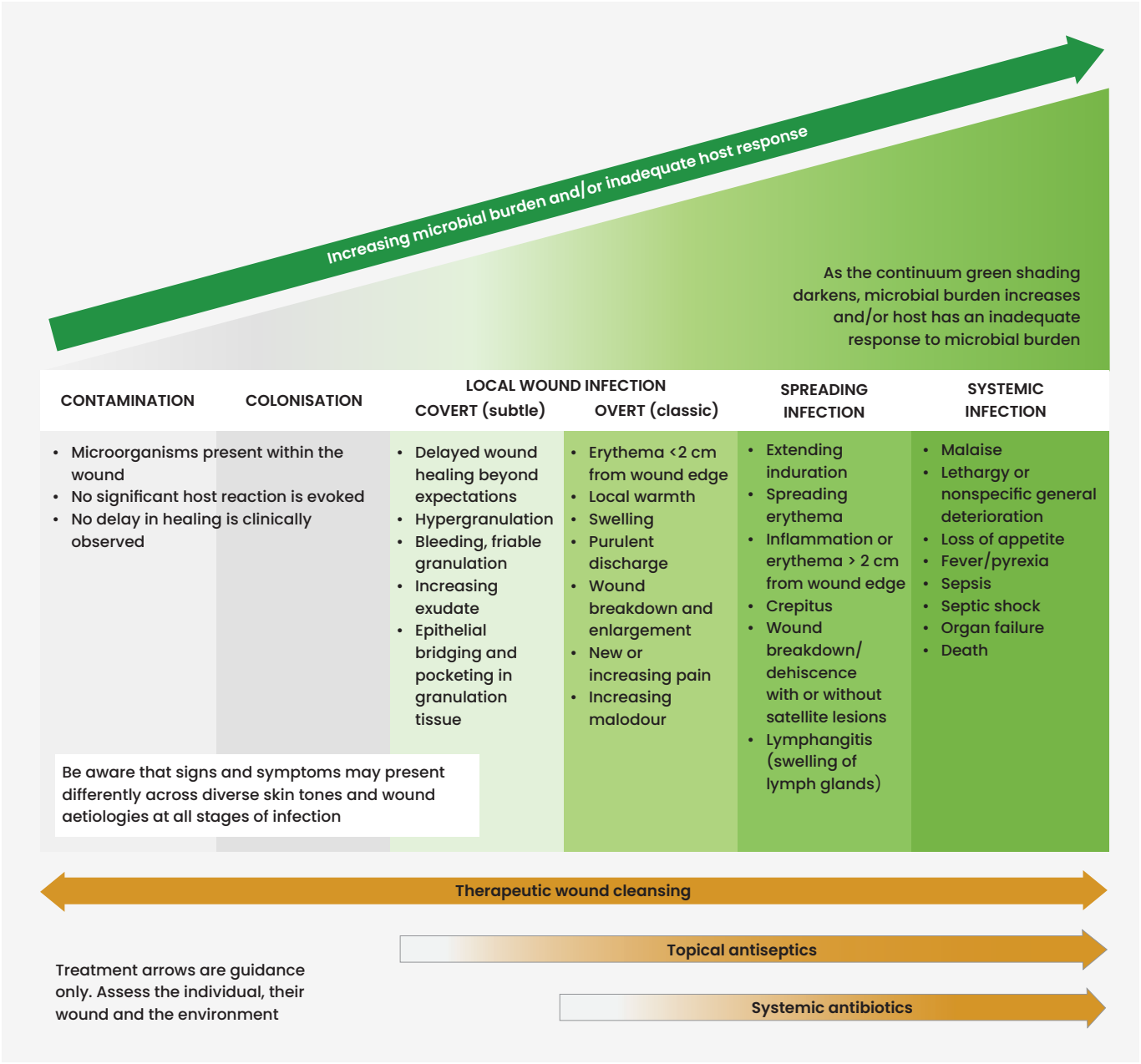


Figure 5. IWII Wound Infection Continuum (IWII-WIC)

wounds are colonised with microorganisms at the time of skin breakdown,[139] but at this stage the virulence appears to be low and the microbial load may be low as there is little proliferation and social interaction. Microorganisms that colonise a wound may also arise from environmental exposure.

Local infection refers to a stage of infection in which microorganisms are present and proliferating within the wound, evoking a host response, often including a delay in wound healing. Local infection is contained within the wound, wound edges and the immediate periwound region and often presents as covert/subtle signs and symptoms[133, 135] that may not be immediately recognised as a sign of infection. This is particularly relevant with respect to biofilm, which is often associated with chronic low-grade infection with subtle manifestations and delayed healing.[140, 141]

Covert/subtle signs and symptoms of wound infection include one or more of:[20, 142–145]

- Delayed wound healing beyond expectations,
- Hypergranulation,
- Bleeding, friable granulation,
- Increasing exudate, and/or
- Epithelial bridging and pocketing in granulation tissue.

As local wound infection progresses, overt/classic signs and symptoms that are traditionally associated with infection generally become evident and are more recognisable as an indicator of wound infection. However, these symptoms may be masked in individuals due to a large range of factors, including skin tone, poor vascular perfusion and compromised immunity **[Table 5]**.

Overt (classic) signs and symptoms of wound infection include one or more of:[142–147]

- Erythema (< 2 cm beyond the wound edge) or change in skin tone,
- Local warmth,
- Swelling,
- Purulent discharge,
- Wound breakdown and enlargement,
- New or increasing pain, and/or
- Increasing malodour.

Spreading infection describes the stage of infection in which there is invasion of the surrounding tissue by infective microorganisms that have spread from a wound. Microorganisms proliferate and spread to a degree that signs and symptoms extend beyond 2cm from the wound edge.[148, 149] Spreading infection may involve deep tissue, muscle, fascia, organs or body cavities.

Spreading infection signs and symptoms include one or more of:[142, 144]

- Extending induration,
- Spreading inflammation or erythema (> 2 cm from the wound edge),
- Crepitus,
- Wound breakdown/dehiscence with or without satellite lesions, and/or
- Lymphangitis (red streaks spreading toward lymph nodes and swelling of lymph glands).

Systemic infection refers to the stage of infection in which microorganisms spread throughout the body via the vascular or lymphatic systems, evoking a host response that affects the body as a whole. In the context of wound infection, microorganisms spread from a locally infected wound. Systemic inflammatory response can also be triggered by a local wound infection through other pathways, for example, through the release of toxins or a dysregulated immune response.

Systemic signs and symptoms of infection include one or more of:[142, 145]

- Malaise,
- Lethargy or nonspecific general deterioration,
- Loss of appetite,
- Fever/pyrexia,
- Sepsis,
- Septic shock,
- Organ failure, and/or
- Death.

Clinical assessment of wound infection

Continuous, accurate, holistic assessment of the individual and their wound is essential for effective wound treatment.[125, 150] Early recognition and timely treatment of wound infection can support healing, improve clinical outcomes and reduce the burden on the individual, their support network and healthcare services.[74, 151–153] Assessment findings should be documented consistently over time, with serial wound photography used where appropriate to support monitoring of wound progression and response to treatment.

Table 5: Factors influencing the clinical assessment of wound infection[147, 158, 160, 161]

Factor	Potential impact on signs and symptoms of wound infection	Clinical impact
Medium and dark skin tones (Baseline skin tone should be included in wound and skin assessment)	• Surgical site infection may present with less obvious visual changes in individuals with dark skin tones.[162] • Colour changes may present as purple, deep red, brown, grey or darker or lighter than the surrounding skin, rather than as bright erythema.[162]	• Potentially delays clinical recognition and diagnosis • Infection may be under-recognised if assessment relies heavily on erythema
Neonatal skin and tissue immaturity	• Presence of vernix caseosa in neonates, fragile/thin skin, physiological oedema	• Infection may be under-recognised
Immunosuppression (e.g., steroids, chemotherapy, transplant medications, HIV)	• Reduces inflammatory response	• Erythema, warmth, swelling, and purulence may be absent or minimal • Infection may be advanced before becoming clinically apparent
Diabetes mellitus	• Pain perception may be reduced • Microvascular disease impairs inflammatory signs	• Classic symptoms such as pain and erythema may be muted (clinicians should ask about symptoms of neuropathic pain) • Infection may be advanced before becoming clinically apparent.
Advanced age	• Diminished immune and inflammatory responses	• Fever, leukocytosis, and local inflammatory signs may be absent
Peripheral arterial disease/ tissue ischaemia	• Poor perfusion limits delivery of inflammatory cells and mediators	• Reduced erythema, warmth, and exudate
Recent antibiotic therapy	• May transiently reduce bacterial burden and attenuate inflammatory response without fully eradicating infection	• Partial treatment may mask clinical progression
Anti-inflammatory medications (e.g., NSAIDs, corticosteroids)	• Suppress fever, pain, swelling, and erythema	• Delays recognition of worsening infection
Heavy wound colonisation without invasion	• Produces malodour and exudate resembling infection	• Risk of over-diagnosis and inappropriate antibiotic use
Inflammatory wound conditions (e.g., pyoderma gangrenosum, vasculitis)	• Mimic infection through erythema or skin tone changes, pain, and tissue breakdown	• Can lead to misdiagnosis and unnecessary antimicrobial treatment
Postoperative inflammation	• New surgical wounds exhibit physiological erythema or skin tone changes (usually not >1cm beyond wound edge), warmth, oedema, and pain	• Makes differentiation between normal healing and infection challenging
Oedema or lymphatic impairment	• Causes swelling and erythema or skin tone changes independent of infection	• Decreases specificity of infection signs
Neuropathy	• Decreases pain sensation	• Infection can progress with little or no reported discomfort
Atypical or anaerobic organisms	• May cause subtle or unusual clinical presentations	• Traditional diagnostic criteria become less reliable
Concurrent systemic illness (sepsis, autoimmune disease, malignancy)	• Alters immune response and inflammatory markers	• Local signs may not correlate with infection severity

Table 5: Factors influencing the clinical assessment of wound infection[147, 158, 160, 161] (continued)		
Factor	**Potential impact on signs and symptoms of wound infection**	**Clinical impact**
Moisture-associated skin damage, incontinence-associated dermatitis and dermatitis	• Produce erythema, exudate, and discomfort that resemble infection	• Can result in false-positive diagnosis
Communication	• Challenges in communicating (e.g., due to language barriers, cognitive or physical impairment, age) reduce the ability to communicate concerns	• Can delay identification of early signs and symptoms of wound infection

Photographic guides to support clinicians in recognising different stages of wound healing [Table 8] and signs and symptoms of wound infection [Table 9] are included in this chapter.

Assessment begins with a holistic evaluation of the individual's risk of wound infection, including relevant host factors and wound history [see Chapter 3]. This assessment must include a clinical assessment of the wound, including its anatomical location and the appearance of the wound bed, wound edges, periwound area and surrounding skin.[154, 155]

Clinical signs and symptoms of wound infection may have limited diagnostic accuracy and reliability.[156–158] However, diagnostic investigations may be time-consuming, costly or not readily accessible. Clinicians should therefore use clinical judgement within the context of a comprehensive assessment, incorporating visual inspection, patient-reported symptoms and physical examination. This should include palpation to assess skin temperature, tissue consistency and oedema, as well as observation for changes in skin tone and other signs and symptoms described in the IWII-WIC.[159] Wound infection should be suspected in the presence of multiple indicative signs and symptoms rather than relying on the presence of any one individual sign or symptom.

Suspect wound infection in the presence of multiple indicative signs and symptoms rather than the presence of any single sign or symptom.

Factors that influence presentation of wound infection

Infection in acute wounds, such as surgical, traumatic and burn wounds, may be readily recognisable in otherwise healthy individuals.[159] In contrast, identifying infection in chronic/hard-to-heal wounds can be more challenging and may require specific clinical knowledge and experience.[159] Covert or subtle signs of local infection may predominate or be masked by underlying conditions [Table 5].[74, 134, 153] Microbial burden does not always produce overt clinical signs and symptoms,[134] and the presentation of wound infection can vary considerably between individuals according to host, wound and other contextual factors [see Table 5].[156–158]

Considerations in assessing wound infection in specific wound types

Wound aetiology should be considered when evaluating both the risk of wound infection and the way infection may present. Both wound aetiology and aetiology-specific risk factors can influence the likelihood of wound infection. Wound infection may present in more subtle ways in older or immunocompromised people, potentially delaying its recognition and treatment [Table 6]. These cumulative factors can lead to treatment delay and progressive infection.

Diabetes is particularly important because it contributes to both wound development and increased susceptibility to infection. Diabetes-related foot ulcers may progress rapidly to deep infection, which can be difficult to detect clinically and may require procedures such as deep probing, radiological investigations or surgical exploration for identification.[109, 161]

Wound infection assessment tools

A wound infection assessment tool can support systematic clinical evaluation. Various scoring systems, diagnostic criteria and classification systems have been developed to assist in identifying infection in specific wound types (e.g., the Centers for Disease Control and Prevention criteria for surgical site infection).[169] However, many available tools were not developed specifically for wound infection assessment or have undergone limited

Table 6: Wound infection assessment in specific wound types

Type of wounds	Aetiology-specific considerations
Surgical site infection (occurs within 30 days of surgery)	• Daily visual wound assessment (where possible depending on the type of wound dressing applied following surgery) and vital sign assessment.[163] • Early indicators of wound infection: ◦ Increased wound edge distance (lack of approximation). ◦ Increased wound exudate.[163] ◦ Increased heart rate.[163] ◦ Increased morning tympanic temperature.[163] ◦ Increasing pain. • Wound edge colour (e.g., erythema or changes in skin tone) and induration are not reliable indicators of wound infection and may present differently depending on the individual's skin tone.[163] Increasing erythema or skin tone changes and induration, especially > 72 hours after surgery could indeed indicate infection.
Pressure ulcers/injuries	• Associated with spreading infection (e.g., cellulitis) and increased markers for infection.[164, 165] • Full thickness pressure ulcers/injuries (i.e., Category/Stage 3 or 4 pressure ulcers/injuries) are more likely to exhibit signs of infection, particularly erythema and purulent exudate.[164, 165] • Observe for indirect indicators of systemic infection (e.g., anorexia, delirium and/or confusion).[164]
Diabetes-related foot ulcers	• Usual signs of sepsis may not be present in individuals with diabetes.[166] • Probing to the bone with a sterile metal probe or instrument to diagnose diabetes-related foot osteomyelitis is inexpensive, accessible and relatively safe.[109] • Plain X-rays and biomarkers of infection (e.g., ESR, CRP and/or PCT) can be used to assess severity and progression of osteomyelitis.[109] • An increase in temperature in one area of the foot identified using palpation, infrared or digital thermometry (if accessible) combined with photographic assessment may be of value in the initial assessment of infection when performed via telemedicine.[109]
Chronic leg ulcers	• Distinguish chronic inflammation from wound infection. • Larger ulcers (≥ 10 cm^2) and those with more wound bed slough are more likely to exhibit signs of infection.[167] • Although heavy wound exudate is an indicator of possible wound infection, it should be considered in the context of lower leg oedema and compression use and whether leg volume reduction has been achieved.[167] • Depression, chronic pulmonary disease and anticoagulant use may be related to wound infection.[167]
Skin tears	• Distinguish trauma-related inflammation from infection.[168] • Early indicators of infection include: ◦ Increased wound edge distance (lack of approximation). ◦ Increased wound exudate. ◦ Increasing pain. ◦ Failure of skin to adhere/loss of skin over the wound. • Mechanism of injury should be considered (tetanus vaccination / booster may be required).[168]

psychometric evaluation. **Table 7** summarises commonly used wound infection assessment tools and classification systems and their reported measurement properties. Because no single clinical sign or symptom can reliably confirm or exclude wound infection,[74] most tools use combinations or checklists of indicative signs and symptoms, many of which are reflected in the IWII-WIC. Some tools also incorporate scoring or ranking systems to support interpretation and clinical decision-making.

Table 7: Wound infection assessment tools

Assessment tool	Wound type	Description	Psychometric testing
ASEPSIS[170]	Developed for cardiac surgery, may be applied to other types of surgical wounds.	• A method of assessing wound • healing that defines characteristics that are awarded points. • Includes objective assessment criteria. • Points are given for:[170, 171] ◦ **A**dditional treatment. ◦ **S**erous discharge. ◦ **E**rythema. ◦ **P**urulent exudate. ◦ **S**eparation of the deep tissues. ◦ **I**solation of bacteria. ◦ **S**tay duration (time spent as an inpatient).	• Sensitivity and specificity of a range of total ASEPSIS scores (score >10 to score >40) in predicting hospitalisation, antibiotic therapy and surgery are reported.[171] • Good inter-rater reliability.[172]
Clinical Signs and Symptoms Checklist (CSSC)[158]	Variety of wound types.	• Includes 12 clinical signs and symptoms of infection. • Includes five classic signs/symptoms of wound infection. • Includes seven secondary signs and symptoms of wound infection.	• Sensitivity and specificity of individual signs and symptoms reported in different populations[158, 173] (range sensitivity 0.18 to 0.81; specificity 0.56 to 1.00).[158] • Positive and negative predictive values of individual signs and symptoms reported in different populations.[158, 173]
Infection Management Pathway[74]	All wound types.	• Standardises the assessment and diagnosis of causes of delayed healing related to local infection and biofilm. • Provides a treatment plan based on which signs/symptoms of infection are present. • Pathway is commercially positioned.	Testing not reported
IWII Wound Infection Continuum (IWII-WIC)	All wound types.	• Presents clinical signs/symptoms as indicators of different wound infection stages.[135] • Conceptual model and teaching tool.[133]	• Includes clinical signs and symptoms validated in other assessment tools.
NERDS and STONEES[174]	Chronic wounds.	• Mnemonics for signs and symptoms of superficial (NERDS) and deep (STONES) infection. • Diagnose superficial infection in the presence of ≥ 3/5 clinical signs/symptoms of superficial infection (NERDS).[174] • Diagnose deep infection in the presence of ≥ 3/5 clinical signs/symptoms of deep infection (STONEES).[174]	• Sensitivity and specificity of individual signs and symptoms of superficial infection (NERDS) reported (range sensitivity 0.32 to 0.70; specificity 0.47 to 0.86).[175] • Sensitivity and specificity of individual signs and symptoms of deep infection (STONEES) reported (range sensitivity 0.37 to 0.87; specificity 0.44 to 0.89).[175] • Sensitivity and specificity of 2-4 signs/symptoms from NERDS or STONEES reported.[175]
Therapeutic Index for Local Infections (TILI) score[176]	Acute and chronic/ hard-to-heal wounds.	• Six indirect criteria for local wound infection, presence of all criteria indicates antimicrobial treatment should be commenced. • Three direct indications; presence of ≥ 1 criterion indicates antimicrobial treatment should be commenced. • Available in multiple languages.	Testing not reported

Table 7: Wound infection assessment tools (Continued)

Assessment tool	Wound type	Description	Psychometric testing
Wound Infection Risk Assessment and Evaluation tool (WIRE)[125]	Community-based wounds.	• Detects early wound infection. • Identifies systemic infection based on clinical presentation.	Testing not reported
WIfI System[177]	Diabetes-related foot ulcers	• Originally developed as part of PEDIS classification.[178, 179] • Defines the presence and severity of foot infection in a person with diabetes on four levels of severity. • Requires clinical examination and standard blood and imaging tests. • Stratification aligns with therapeutic decisions.	• Predictor of healing and time to healing.[178] • Valid as an indicator of risk of amputation.[179, 180] • Low inter-rater reliability.[179]

Table 8: Signs of healthy wound healing

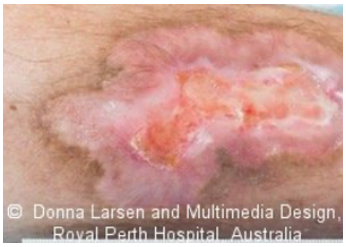
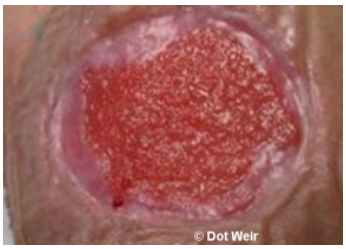
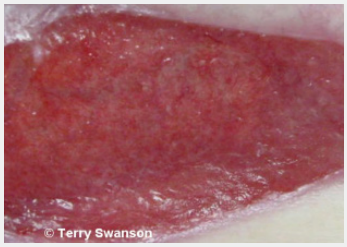
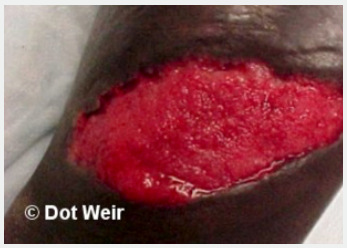
Wound bed tissue	Appearance	Clinical presentations
Epithelial tissue	Appears pink to lavender or pearly white and indicates the wound is viable and healthy. Note that epithelialisation will not occur in an unhealthy wound bed.	 
Granulation tissue	Appears red and moist and is well-vascularised, occurring in the reconstruction or proliferative phase of healing and indicates the wound is viable and healthy.	 

Table 9: Presentation of unhealthy tissue and signs and symptoms of wound infection

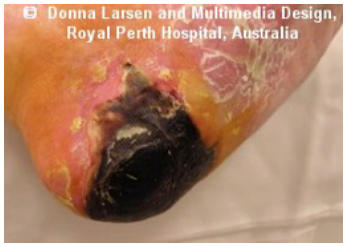
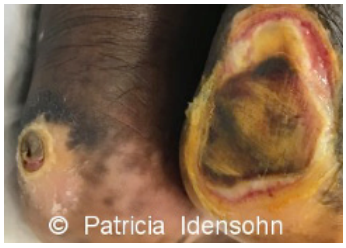
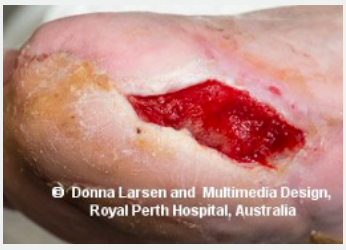
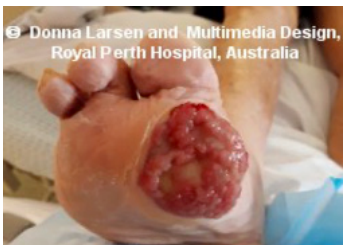
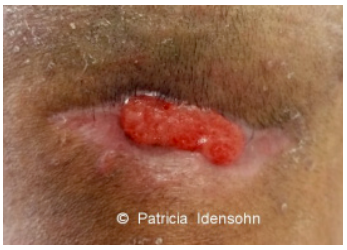
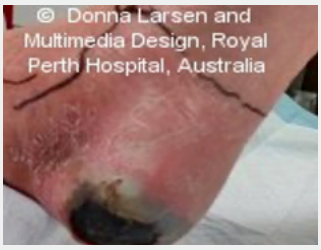
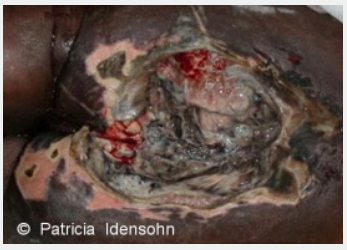
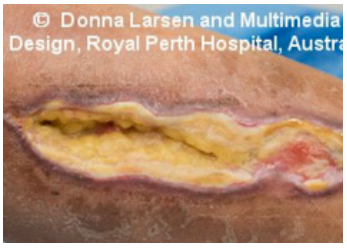
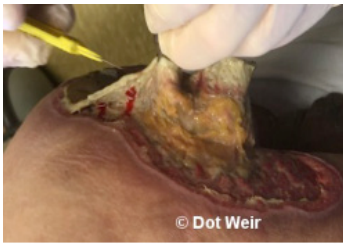
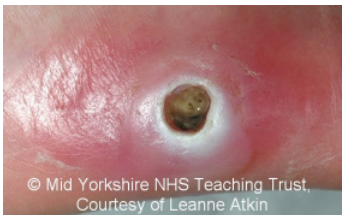
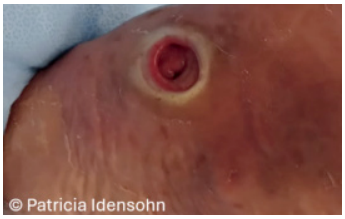
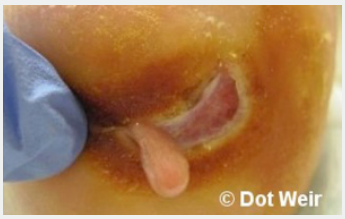
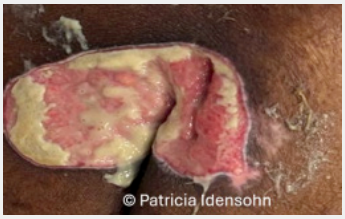
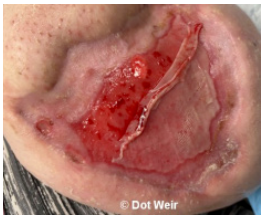
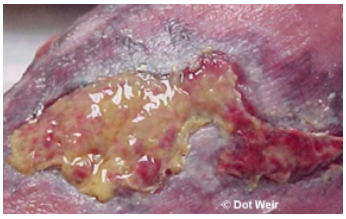
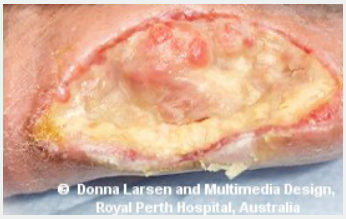
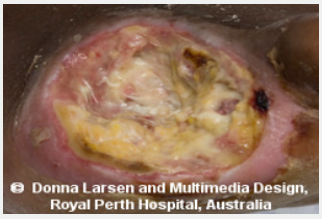
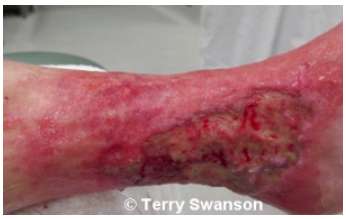
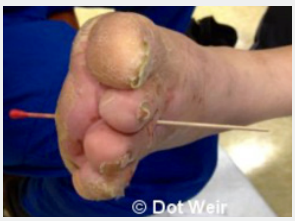
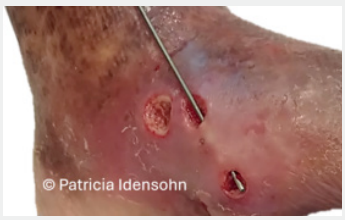
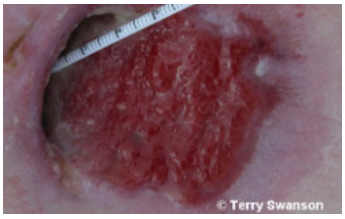
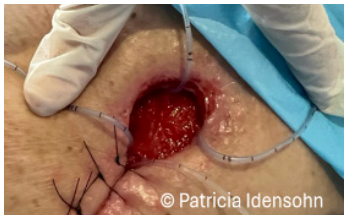
Wound bed tissue	Appearance	Clinical presentations
Eschar	Appears black and dry, indicates the presence of extensive dead tissue that will prevent wound healing.	 
Friable granulation tissue (unhealthy granulation)	Fragile tissue that bleeds easily.[136]	 
Hypergranulation tissue	Appears red, uneven and granular, usually growing above the level of the surrounding skin. Occurs in the proliferation stage and indicates that the tissue has overgrown. Associated with high microbial burden or friction to the wound.	 
Infected necrotic tissue	Can commence as red lumps or bumps that spread to a bruise-like appearance with a centre dark/ dusky region that turns black. Skin may break and ooze exudate. There will be evidence of surrounding erythema.	 
Non-viable adipose tissue	Non-viable body fat and loose connective tissue that appears white, brown or yellow (colour varies by hydration), and may appear like fat molecules or droplets. Can be confused with slough.	 
Osteomyelitis (high risk due to exposed bone)	Osteomyelitis is an infection of the bone that occurs through infection of the bloodstream or from a wound that allows bacteria to directly reach bone, for example through direct exposure of the bone	 

Table 9: Presentation of unhealthy tissue and signs and symptoms of wound infection (Continued)

Wound bed tissue	Appearance	Clinical presentations
Periwound erythema	Superficial reddening of the periwound skin (in these photographs, appears with maceration)	 
Purulent exudate	Thick, milky fluid, often with an offensive odour, and containing white blood cells, microbes, and tissue debris.	 
Proteinaceous, mucilaginous or coagulum (loosely adherent slough)	Loosely adherent surface substance of various colourings that appear gelatinous.	 
Slough	Adherent tissue that appears yellow, brown or grey and indicates presence of devitalised tissue (i.e., dead cells) and debris that will impede wound healing.	 
Spreading erythema	Diffuse reddening of the surrounding skin and beyond	 
Tunnelling	A sinus tract (channel or open passage) that extends from the wound bed underneath the surface of the skin.	 
Undermining	Wound edge is separated from the healthy tissue around causing a pocket to form underneath the surface	 

Chapter 6
Diagnosis of Wound Infection

2026 updates and key points

- Diagnosis of wound infection should be made based on clinical signs and symptoms of wound infection.
- Microbiological investigations are used to identify causative organisms. Haematological and radiological testing helps to assess severity of infection.
- The technique used to collect a sample for microscopy, culture and sensitivities is important to the reliability of the results. Tissue biopsy is the preferred technique.
- Microbiological culture and sensitivities are only used when an antibiotic/antifungal is indicated (i.e. spreading or systemic infections).

Introduction

A comprehensive wound assessment supports the early recognition and diagnosis of wound infection and timely initiation of treatment. Clinicians should therefore understand both the factors that increase infection risk and the clinical signs and symptoms of infection. Diagnosis is primarily clinical and includes assessment for the classic signs of infection: warmth, pain, swelling and erythema.[110, 181–183] In chronic/hard-to-heal wounds, infection may present with covert or slowly emerging signs and symptoms. This reflects the dynamic nature of microbial burden and the frequent presence of polymicrobial biofilm communities, which may not produce overt signs of infection (see Chapter 5).[181]

Clinical assessment can usually provide a reliable diagnosis of wound infection. However, in complex or chronic/hard-to-heal wounds, clinical signs and symptoms of infection are not always readily apparent. Clinicians should recognise the limitations of relying on classic signs, particularly in individuals with chronic/hard-to-heal wounds, immunocompromise, poor perfusion, peripheral neuropathy or diabetes-related foot disease, multiple comorbidities, exposed bone or anticipated surgery.[160] Signs and symptoms of wound infection may be absent, muted or delayed, and their presentation may differ across skin tones.[160, 181, 184] Investigations can support confirmation of infection and inform decisions regarding antimicrobial therapy, including systemic antibiotics.

Diagnosing wound infection

All open wounds contain microorganisms in both planktonic and biofilm forms. Therefore, a wound should only be cultured after making a clinical diagnosis of wound infection based on signs and symptoms, or when there remains a high clinical suspicion of wound infection, for example if there is failure of the wound to improve or to heal despite optimal care.

Microbiological analysis of a wound specimen can be performed to provide useful information, including identification of microbes and antimicrobial susceptibility patterns to direct treatment.[109, 160, 183, 187–189] This includes ensuring empiric therapy is appropriate to the pathogen.[109, 186]

Recommendation 4

Identify wound infection based on clinical signs and symptoms. When indicated, use microbiological investigations to identify causative microbes and/or perform haematological and radiological investigations to assess severity of infection.

(Underpinning evidence: Level 3 evidence[183] and Level 5 evidence[109, 149, 185, 186])

Microbiological analysis has important limitations and should always be interpreted within the clinical context.[160] Its usefulness is influenced by the organisms present, the type of specimen collected, the sampling technique and the diagnostic method used. Wound swabs predominantly recover microorganisms from the wound surface and may underrepresent organisms located deeper within wound tissue. Because microorganisms are not distributed uniformly throughout a wound, any sampling method may fail to obtain a representative specimen.[160] In addition, detection of microorganisms confirms their presence but does not, in isolation, distinguish wound colonisation from wound infection.

Interpretation of laboratory results when multiple microorganisms are isolated, is a major diagnostic challenge.[160] The presence of microbes does not confirm infection, because chronic/hard-to-heal wounds are almost always colonised with multiple species of bacteria, so presence of microorganisms should be expected in any wound sample.[160] To facilitate the

Table 10: Indications for initiating microbiological analysis of a wound specimen[109, 149, 185, 186]

- Acute or chronic wounds with signs of overt, spreading or systemic* infection
- Chronic wounds with delayed healing despite an appropriate and holistic wound care regimen
- Infected wounds that have failed to respond to antimicrobial intervention, or are deteriorating despite appropriate antimicrobial treatment
- Identify causative pathogens, guide the selection of antimicrobial treatment (or adjustment if the individual does not respond to empiric treatment) and support antimicrobial stewardship
- In compliance with local protocols for the surveillance of drug-resistant microbial species
- Wounds where the presence of certain species would negate a surgical procedure (e.g., beta-haemolytic streptococci in wounds prior to skin grafting)

* In individuals showing signs of sepsis, blood cultures are also indicated, and other likely sites of infection should be considered. Collect other samples for microbiological analysis as relevant (e.g., specimens of urine, sputum or the tip of a central venous line catheter).

To avoid false-positive results, use an inert wound cleanser and debride the wound (if required) prior to collecting a specimen from the base of the wound

best sampling of microbes within the wound bed, specimens for microbiology should only be taken after aseptic therapeutic wound cleansing and/or debridement.[141, 155]

The isolation of organisms does not reflect microbial interactions, virulence or biofilm behaviour. Additionally, many commonly used investigative techniques cannot identify biofilm.[160] Therefore, the decision to take a wound specimen for microscopy, culture and sensitivity testing should be made carefully, and only when the individual has overt or greater signs and symptoms of infection, or when a wound fails to heal despite optimal care, especially in immunocompromised individuals.

Biofilm detection and quantification

Biofilm detection and quantification in wounds remain challenging because there is no widely available bedside test that definitively confirms biofilm presence. Clinical suspicion is therefore usually based on wound characteristics, response to treatment and failure to progress despite appropriate care, while laboratory confirmation may require specialised techniques.[140] Microscopy, including scanning electron microscopy and confocal microscopy, can demonstrate biofilm structure, while culture and molecular techniques such as quantitative PCR can characterise or quantify microbial populations. However, these methods do not always distinguish planktonic organisms from true biofilm communities.[190] Consequently, biofilm assessment should combine clinical evaluation with appropriate microbiological or specialised diagnostic methods where available, rather than relying on any single test.

Wound specimen collection techniques

The method of specimen collection from the wound influences accuracy and clinical relevance of microbiological results.[160] The following methods are commonly used to collect a sample from the wound for microbiological analysis:[191]

- Tissue biopsy or curettage,
- Wound swab, and
- Needle aspirate (wound exudate/pus collection), noting this is used mainly for sampling deep space/abscess infections rather than open wounds.

Different sampling techniques vary in their ability to detect pathogens. Not all culture techniques will detect all clinically relevant pathogens.[192] Preferred specimen collection techniques are outlined in **Table 11**.

Tissue biopsy

Tissue biopsy or curettage is generally considered the preferred method for obtaining a wound specimen for microbiological analysis because it samples microorganisms within deeper wound tissue and may provide both qualitative and quantitative information. Studies suggest that tissue and aspirate specimens more closely reflect microorganisms associated with wound infection than surface swabs.[183, 193, 196, 198] Using aseptic technique, the wound should first be cleansed and, where appropriate, debrided to remove surface contaminants and non-viable tissue. A tissue specimen is then obtained from viable wound tissue using punch biopsy, incisional biopsy or curettage.[191] The precise sampling site and technique should be selected

Table 11: Diagnostic utility for specific pathogen types[109, 186, 192–194]		
Pathogen type	**Preferred specimen**	**Clinical consideration**
Aerobic bacteria	• Tissue biopsy • Needle aspirate • Wound swab (Levine technique, refer to **Figure 6**)	• Most specimen techniques will readily culture typical aerobic wound pathogens
Anaerobic bacteria	• Tissue biopsy	• Anaerobes rapidly lose viability when exposed to oxygen • Contact laboratory to confirm appropriate collection and transport media • Surface wound swabs have poor recovery rates • Consider in malodorous, necrotic, or ischaemic wounds (do not biopsy necrotic tissue)
Fungal and atypical mycobacteria	• Tissue biopsy • Needle aspirate for deep space abscesses or joint infections	• Histopathology and fungal culture require an adequate tissue volume and sample from deeper tissues • May require specialised culture media. Contact laboratory to confirm appropriate collection and transport media • Molecular diagnostics may assist in more rapid identification of mycobacterial and fungal infections, given long culture times. Cultures should still be done to obtain MICs which guide antibiotic dosing • Suspect in chronic/hard-to-heal wounds that do not progress to healing with standard debridement and antimicrobial therapy

according to the wound type, suspected depth and nature of infection, and the purpose of the microbiological investigation. Punch or incisional biopsy provides the highest diagnostic yield and allows histopathological examination and stains if needed (when ruling out mycobacterial or fungal infections, stains can be ordered for rapid results, although a negative result does not indicate lack of infection).

Tissue biopsy is painful, can potentially cause further tissue damage and feasibility can vary according to the anatomical location of the wound and laboratory capability. The procedures require specialist equipment and a skilled clinician and can be costly. Therefore, tissue biopsy is not routinely performed in many clinical settings.[191]

Recommendation 5

Select tissue biopsy as the preferred method to collect a wound specimen for diagnostic investigations. When tissue biopsy is not available, perform a wound swab using Levine technique.

(Underpinning evidence: Level 1 evidence,[195, 196] Level 2 evidence,[197] and Level 3 evidence[183])

Wound swabs

In many clinical and geographic settings, a wound swab is the more readily available method for collecting a wound sample for culture and sensitivities.[109, 189] This method of sample collection is simple, non-invasive, accessible and relatively inexpensive.[189, 199] In a study[183] in which clinicians and microbiologists diagnosed wound infection based on clinical presentation and the results from either tissue biopsy or wound swabs, agreement between biopsy and swab varied between 77% and 96%.[183] In this study, variability between individual assessors was more significant than the agreement based on wound specimen techniques, highlighting the importance of competency in recognising clinical presentation of wound infection.[183] The results suggest that although tissue biopsy is the gold standard, a correctly performed wound swab is adequate for most wounds in the absence of availability and/or feasibility of tissue biopsy.[183]

There are several important limitations to wound swabbing. First, microbiological analysis of a wound swab can only identify more superficial wound microorganisms and not the microbes in the deep tissues of the wound.[197] This is significant because biofilm is known to penetrate and proliferate deep within chronic/hard-to-heal wounds.[200] A sample of superficial wound microflora will identify expected microorganisms that colonise all wounds, and the microbiological analysis may not differentiate colonisation from infection.[109, 160] Secondly, not all microorganisms collected on a wound swab will survive during transportation to the laboratory, further influencing the accuracy of wound swab results.[191] Additionally, the collection technique and equipment used to collect the sample are shown to influence the quality of the wound sample.[191, 201]

The Levine technique is the preferred method for taking a wound swab. Studies have demonstrated that this method achieves a more reliable sample of the wound microflora compared to the Z-swab technique.[189, 193, 196] The Levine technique includes expression of fluid from the tissue, targeting deeper microorganisms than those directly on the surface of the wound. This is more likely to collect microorganisms causing infection rather than superficial contaminants. **Figure 6** provides an overview of the Levine technique. The procedure is performed after cleansing the wound with an inert (chemically inactive) and sterile solution (e.g., sterile normal saline) to reduce the collection of superficial contaminants.

When a wound swab is indicated, the specimen should be collected using an appropriate technique and placed promptly into the transport medium specified by the receiving microbiology laboratory. Where both culture and direct microscopy are required, separate specimens (i.e., two swabs required) may be requested according to local laboratory protocols. If anaerobic infection is suspected, appropriate anaerobic sampling and transport methods are required, with tissue or aspirate preferred where feasible. Direct microscopy, including Gram staining, can provide rapid preliminary information about microorganisms and inflammatory cells present in the specimen, supporting early interpretation while definitive culture and susceptibility results are pending.[186]

The type of swab (nylon flocked swabs or traditional cotton, rayon, and polyester fibre swabs) used to take a specimen for culture can impact findings and interpretation because there are important differences in their specimen collection performance, analytical reliability, and diagnostic utility.[202, 203] Flocked swabs use perpendicular nylon fibres that enhance sample uptake and facilitate efficient release into transport media, resulting in improved recovery of cells, microorganisms, and nucleic acids. This superior recovery can translate to higher analytical sensitivity, particularly for molecular diagnostics. In contrast, traditional fibre swabs may retain portions of the collected specimen within the fibre matrix, reducing the specimen available for analysis.[203–205] Overall, swab selection should be guided by clinical objectives, required diagnostic sensitivity, local pathology/microbiology service protocols and cost considerations.

Further microbiological investigations

Further microbiological investigations may be indicated when there is suspicion of spreading wound infection or systemic infection. These include blood cultures and where clinically indicated, cultures from bone or deep tissue obtained during surgery. Blood cultures should be obtained in individuals with suspected bacteraemia, sepsis or severe wound infection associated with systemic signs and symptoms of infection. Blood cultures should ideally be collected before initiation of systemic antimicrobial therapy, provided this does not delay treatment. Routine blood cultures are not recommended for uncomplicated localised wound infections because of their low diagnostic yield.

Haematological and biochemical investigations

Haematological and biochemical investigations are valuable adjuncts in the assessment of wound infection, although they should not be used in isolation to establish a diagnosis. The value of these tests lies in corroborating clinical findings, identifying complications/severity of infection, and guiding management decisions.

Figure 6. Taking a wound swab for culture using the Levine technique

The white blood cell count (WBC) with differential is commonly used to evaluate the presence of infection.[191] An elevated WBC accompanied by neutrophilia may indicate an acute inflammatory or infectious process. However, a normal WBC does not exclude infection, particularly in chronic wounds, older adults, or individuals who are immunocompromised, where the inflammatory response may be diminished.[206] The erythrocyte sedimentation rate (ESR) is a non-specific marker of inflammation that typically rises more slowly than other inflammatory markers. Consequently, it is more useful in the assessment of chronic or deep-seated infections and for monitoring trends in disease progression over time.[206]

Biochemical markers such as C-reactive protein (CRP) and procalcitonin can provide additional information regarding systemic inflammation but are non-specific markers. CRP levels increase in response to inflammatory stimuli from both infectious and non-infectious

causes and may be useful in monitoring the effectiveness of treatment.[191] Procalcitonin rises more rapidly, particularly in bacterial infections, and has demonstrated value in the diagnosis and monitoring of systemic or spreading infections. However, procalcitonin levels may remain within normal limits in patients with localised wound infections, limiting its utility as a sole indicator of wound infection.[207]

Radiological investigations

Radiological investigations are also used to explore and corroborate clinical assessment rather than an isolated diagnostic investigation. Radiological investigations are unable to diagnose the microbial species or antimicrobial sensitivities.[208] They are usually unable to identify early wound infection as there is generally no anatomical change in the absence of clinical signs indicative of more advanced infection such as fluid accumulation or bone, tissue and organ changes.[208] Therefore, these investigations are primarily used to assess the extent of complications of wound infection (e.g., osteomyelitis). An overview of imaging techniques is provided in Table 12.

Table 12: Comparison of radiological investigations used in wound infection diagnosis[191, 209-211]

Imaging technique	Purpose and uses	Clinical considerations
Plain x-rays	• Identify presence of osteomyelitis or abscess • Detect foreign bodies • Detect gas in soft tissue	• Widely available • Only detects radiopaque foreign bodies and bone involvement • Limited soft tissue contrast • Radiation exposure
Magnetic resonance imaging (MRI)	• Identify presence of osteomyelitis or abscess • Detect foreign bodies • Detect gas in soft tissue	• Superior soft tissue analysis • No radiation risk • Detects early bone changes • Accessibility and cost
Computerised tomography (CT)	• Identify presence of osteomyelitis or abscess. • Detect foreign bodies • Detect gas in soft tissue	• Detects bone and deep soft tissue pathology • Radiation exposure • Limited soft tissue analysis • Accessibility and cost
Ultrasound	• Identify presence and extent of abscess, fluid collection or haematoma • Foreign body detection	• Real-time imaging often accessible at bedside • No radiation risk • Differentiates abscess from cellulitis • Limited depth penetration • Operator-dependent and requires training
Nuclear medicine scans e.g. positron emission tomography (PET), single photon emission computerised tomography (SPECT), bone scans	• SPECT/CT offers superior specificity for osteomyelitis	• Used when MRI is contra-indicated • Used in presence of metal implants • Accessibility and cost

Table 13: Types of microbiological examination[212-215]			
Type	**Mechanism**	**Resolution limit (maximum magnification)**	**Clinical considerations**
Light microscopy	Visible light	0.2 µm (1500x)	• Detects planktonic microorganisms • Primarily used on isolated cultures or sections of tissue • Gram stain used to establish presumptive species identification • Impossible to obtain definitive identification of microbial species • Low-cost and readily available
Fluorescence microscopy (Fluorescence In Situ Hybridization; FISH)	Ultraviolet light	0.1 µm (2000x)	• Targeted species and their relative locations can be mapped with fluorescent dyes/labels • Detection is limited to organisms recognised by the probes used. • Use is limited to microbial cell suspensions and thin tissue sections • Cost is a limitation
Confocal laser scanning microscopy (CLSM)	Laser beam coupled to a light microscope	0.1 µm (2000x)	• Detects planktonic microorganisms and biofilm • Species can be identified and their relative locations mapped with fluorescent dyes/labels • Tissue blocks can be examined and images obtained at regular depths can be reconstructed to generate 2D or 3D structure of whole specimen • Only fluorescent structures are observed • Fluorescence decays relatively quickly • Cost is a limitation
Scanning electron microscopy (SEM)	Electrons beamed onto specimen at an angle and deflected electrons collected	10 µm (500,000x)	• Detects planktonic microorganisms and biofilm • Cannot examine living material • Minimal time required for sample preparation • Specimen surface layer images provide insight into 3D structure • Dehydration of samples may cause changes • Cost is a limitation
Transmission electron microscopy (TEM)	Electrons beamed through a thin section of specimen	0.2 µm (5,000,000x)	• Detects planktonic microorganisms and biofilm • Images detail internal cellular structures or organisms • Cannot examine living material • Specimen preparation is lengthy, and may introduce artefacts • Cost is a limitation
Polymerase chain reaction (PCR)	Amplifies specific regions of DNA	0.1 and 10 kilobase pairs	• Can confirm genes of interest from bacteria, toxins, viruses and other microorganisms • Rapid and highly specific • Identifies non-cultivatable or slow growing micro-organisms (e.g. mycobacteria, anaerobes, viruses) • Superior sensitivity versus traditional cultures for detecting polymicrobial growth

Microscopy

Different microscopy techniques may be used to support microbiological assessment, although their availability varies between clinical settings **[Table 13]**. Direct microscopic examination, such as Gram staining, can provide rapid preliminary information about the presence and morphology of microorganisms and inflammatory cells within a specimen. This may assist early clinical decision-making while awaiting culture and antimicrobial susceptibility results, which commonly require 24–48 hours.[194]

Microbiology results should not be interpreted in isolation. They should be considered alongside the individual's clinical context, wound characteristics, ongoing signs and symptoms of infection and overall clinical judgement.[183, 194] If appropriate, consult a microbiologist or an infectious disease expert.

Routine culture-based microbiology identifies only a proportion of the microorganisms present within a wound.[186] Where fungal, mycobacterial or anaerobic infection is suspected (e.g., following wound exposure to environmental contaminants or a wound that is not responding to treatment) investigations should be specifically requested and, where necessary, discussed with the microbiology laboratory.

Adjunct diagnostics

Point of care theranostic tools

Fluorescence imaging and wound pH assessment are emerging point-of-care adjuncts that may provide additional information about the wound environment. Bacterial fluorescence imaging uses violet light to stimulate endogenous fluorophores produced by some bacteria. Under appropriate imaging conditions, red fluorescence is associated predominantly with porphyrin-producing bacteria, while cyan fluorescence may be associated with P. aeruginosa.[156, 216] The technique provides immediate information about the presence and spatial distribution of elevated bacterial burden, allowing areas of concern within the wound bed and periwound tissues to be visualised at the point of care.[156, 216]

Fluorescence imaging may supplement clinical assessment because wounds with clinically significant bacterial burden do not always demonstrate obvious clinical signs of infection. In a multicentre study of 350 wounds, 82% had clinically relevant bacterial loads, while assessment based on clinical signs and symptoms failed to identify 85% of these wounds.[216] Addition of fluorescence imaging increased detection of elevated bacterial burden approximately fourfold, and the findings also influenced wound bed preparation and treatment decisions.[216] The ability to identify the location of bacterial fluorescence may be useful for targeting therapeutic cleansing and debridement and selecting the most appropriate site for microbiological sampling. A comparison of fluorescence-guided and clinically directed sampling demonstrated that fluorescence can improve identification of sampling locations containing clinically relevant bacterial burden.[217] Fluorescence imaging may also support wound infection management across different skin tones.[184]

However, fluorescence imaging does not diagnose wound infection or identify biofilm specifically. It detects fluorescence associated with porphyrin-producing bacterial burden only and cannot distinguish planktonic bacteria from bacteria within biofilm. It also does not provide definitive species identification or antimicrobial susceptibility results. Fluorescence findings should therefore be interpreted alongside the individual's clinical presentation, comprehensive wound assessment and, when indicated, microbiological investigation.

Wound pH may provide useful information about the biochemical environment of a wound and its healing trajectory. Intact skin is normally acidic, whereas chronic/hard-to-heal and infected wounds tend to have a more alkaline pH, often above neutral.[218, 219] Alkaline conditions may favour bacterial proliferation and are associated with increased protease activity, impaired oxygen release and disruption of normal healing processes.[218, 219] Conversely, a shift towards a more acidic environment has been associated with progression towards healing. Recent clinical profiling has also shown that chronic/hard-to-heal wounds have significantly higher pH values than acute or healing wounds, with pH tending to decline over time in wounds that are healing.[220, 221] For this reason, serial wound pH measurement used alongside clinical assessment may be more informative than a single reading. However, wound pH is influenced by wound aetiology, exudate, infection, wound dressings and topical treatments, and no single pH threshold can currently diagnose wound infection. It should therefore be considered an adjunct biomarker rather than a stand-alone diagnostic test[221] and may also be used to inform treatment choices [see Chapter 10].

Genomic sequencing

Molecular diagnostic techniques are increasingly being used to complement conventional microbiological methods for identification of microbes particularly in complex and chronic/hard-to-heal wounds.[222] These techniques can detect microorganisms that are difficult or impossible to culture using standard laboratory methods and may improve pathogen

identification in individuals who have received prior antibiotic therapy or when routine cultures are negative despite a high clinical suspicion of infection.

Unlike culture-based methods, genomic sequencing identifies microbial nucleic acids directly from clinical specimens. Metagenomic approaches characterise the collective microbial DNA within a wound, providing information on microbial diversity, potential pathogens, antimicrobial resistance genes and virulence-associated determinants. Genomic sequencing can inform strategies to manage wound infection in more complex, chronic/hard-to-heal wounds[222-224] and to inform local antimicrobial stewardship.[225]

Despite these advantages, genomic sequencing has important limitations that currently restrict its routine clinical use. Detection of microbial DNA does not distinguish viable from non-viable microorganisms and cannot reliably distinguish colonisation and infection.

Chapter 7
Wound biofilms

2026 updates and key points

- Current scientific understanding of microbial behaviour within wounds remains limited. There is broader recognition of the infectious wound microenvironment as a broader concept than biofilm alone.
- Diagnostic confirmation of presence of microorganisms in biofilm forms may not be required, unless there is clinical interest in the species of microorganisms that might inform treatment strategies.
- Therapeutic wound cleansing, debridement and exudate management optimise the wound microenvironment rather than targeting biofilm in isolation

Introduction

Breach of the skin barrier exposes normally sterile underlying tissues to microorganisms. The initial microbial composition of an acute wound may be influenced by the mechanism of injury and the surrounding environment (e.g., bite wounds may contain oral flora, water-exposed wounds may contain aquatic microorganisms, soil-contaminated injuries may introduce environmental microbes). However, chronic wounds typically evolve over time and regardless of the initial insult, their colonising microbes become increasingly dominated by endogenous microorganisms derived from the individual's skin and mucosal microbiota. Healthy human skin hosts a diverse ecosystem of bacteria and fungi primarily beneficial in nature. As the environment changes from the relatively dry acid mantle of intact skin to a moist and inflammatory environment of a wound, shifts in microbial metabolism, proliferation and composition generally occur.[86, 226, 227] These changes in the wound microenvironment influence microbial behaviour and interactions with the host.

Wound biofilm

In their native environments, many species of microbes preferentially grow attached to surfaces or in a biofilm. Early research has provided evidence regarding an association with biofilms and wounds in general and the concept of disease progression.[228, 229] Three landmark studies published in 2008 provided convincing evidence that biofilms develop within wounds and are associated with delayed healing.[230-232] In one of these studies, a prospective exploration using scanning electron microscopy established that 60% of chronic wounds contained biofilm, compared to 6% of acute wounds.[232]

Since then, a rapidly expanding body of scientific literature has attempted to describe the impact of biofilm on wound progression and healing. In 2017, a meta-analysis of nine chronic wound studies showed almost 80% of the chronic wounds sampled contained biofilms, leading the authors to conclude that biofilms are ubiquitous in a chronic wound.[233] However, other studies have positively identified biofilm in only 30% of chronic wounds sampled.[234] Although substantial advances have been made in understanding wound biofilm, important questions remain regarding their development, spatial distribution, persistence and the precise mechanisms by which they contribute to delayed healing and wound infection.[235]

Although biofilms are frequently detected in chronic wounds, their presence alone does not establish a direct causal relationship with impaired healing. Rather, current evidence supports a strong association between biofilm, persistent inflammation, delayed healing and an increased risk of wound infection. However, the relative contribution of biofilm compared with other host, wound and environmental factors remains an area of ongoing investigation. Specifically, the following remain unclear:

a. To what extent do biofilms directly drive delayed wound healing, persistent inflammation and infection?
b. Does the chronic wound environment preferentially promote biofilm formation and persistence?
c. How do host factors, microbial interactions and wound microenvironment collectively influence biofilm development and pathogenicity?
d. Which individuals and wound types are most likely to benefit from biofilm-targeted interventions?

Microorganisms are often difficult to eradicate from wounds, particularly when wound infection is established or microorganisms are organised within biofilm.[236] This persistence is attributed, in part, to the increased tolerance exhibited by biofilm-associated microorganisms to topical and systemic antimicrobial agents, together with protection from host immune responses.

Recognition of microbial persistence and biofilm tolerance has contributed to contemporary concepts of the chronic/hard-to-heal wound and has informed strategies aimed at disrupting biofilm while optimising the wound healing environment.[106, 237]

Application of bench research on biofilms to the clinical wound environment

Much of our understanding of wound biofilms has been derived from laboratory (*in vitro*) models. Although these models have provided important mechanistic insights into biofilm formation, structure and antimicrobial tolerance, caution is required when translating these findings directly to the clinical wound environment.

Sophisticated laboratory models are now available that reproduce many characteristics of wound biofilms and have greatly advanced our understanding of microbial behaviour.[238] However, these models cannot fully replicate the complexity of the clinical wound environment. Differences between laboratory models and clinical wounds are reflected in microbial gene expression, metabolism, interspecies interactions and antimicrobial susceptibility, even when laboratory biofilms are established using microorganisms isolated from human wounds.[236, 239-241]

Clinical wounds represent a highly dynamic infectious microenvironment characterised by oxygen gradients, local hypoxia, altered pH, nutrient limitation, host inflammatory mediators and slow-growing or metabolically inactive microbial populations.[232, 242] These conditions differ substantially from most laboratory systems and should be considered when interpreting experimental findings and applying them to clinical practice.

Continued integration of basic science, translational research and clinical observation has substantially advanced our understanding of wound infection and biofilms. Ongoing collaboration between laboratory and clinical research remains essential to refine the wound infection continuum, improve diagnostic approaches and develop more effective strategies for preventing and managing wound infection.

What is known (and unknown) about wound biofilms?

As described in the literature, *in vitro* biofilms are initiated by planktonic microorganisms and follow a defined developmental cycle. The *in vitro* hallmark of biofilms is the presence of a self-produced matrix of extracellular material composed of polysaccharides, proteins, extracellular DNA and supporting cross-linking metal ions such as calcium, magnesium and iron.

However, this knowledge may not directly translate to biofilm behaviour within a wound. How biofilms develop in chronic and acute wounds is still unknown. Observations have confirmed that in many wounds, microorganisms are present not only as aggregates but also as single cells; however, the relative contribution of these different microbial states to inflammation, tissue damage and delayed repair remains poorly defined.[243, 244]

This uncertainty is important clinically because the wound should not be viewed simply as a surface colonised by biofilm. Chronic/hard-to-heal wounds contain a complex mixture of host tissue, inflammatory cells, exudate, necrotic material, slough and microorganisms in different physiological states.[245] Slough should not be viewed simply as non-viable tissue covering the wound bed. Rather, it may represent a complex host-derived microenvironment in which bacteria can be embedded, protected from host defences, metabolically altered, or spatially separated from immune cells and antimicrobial agents. However, it remains unresolved to what extent microorganisms within slough behave as classic biofilm, loose aggregates, or predominantly single cells. Often there will be multiple different bacterial or fungal species present.[246, 247] Understanding the interplay between co-existing microbial species in chronic/hard-to-heal wounds and potential mechanisms that explain the increased tolerance of biofilm to the host and traditional antimicrobial agents continues to be explored.[248]

Wound biofilms can be embedded in both viable and non-viable tissues, and the wound dressing itself.[61] Therefore, the microenvironment is central to understanding wound microbiology. Oxygen tension, pH, nutrient availability, redox conditions, exudate composition and host-derived matrix components may all influence microbial behaviour. These factors can shape whether microorganisms grow, become metabolically inactive, tolerate antimicrobial

exposure, or interact with host inflammation. From this perspective, the clinical challenge extends beyond biofilm alone. Rather, it is the interaction between microorganisms, host tissue, inflammatory responses, slough and the local physicochemical environment that creates an infectious microenvironment capable of sustaining microbial persistence, promoting tolerance and increasing the risk of wound infection.[242] Although microorganisms undoubtedly contribute extracellular components within wounds, the relative contribution of microbial-derived matrix compared with host-derived proteins, DNA and tissue components remains uncertain.[61]

Whether polymicrobial aggregates form after microorganisms enter the wound or are introduced already organised remains uncertain.[238] Some studies have identified bacterial aggregates on both healthy skin and acute epidermal wounds,[249, 250] suggesting that in at least some clinical situations, biofilm may be established prior to its introduction to a wound. More research is required to confirm and fully understand this mechanism.

Importantly, neither planktonic microorganisms nor biofilm cause a wound to initially occur. Rather, wound aetiology, tissue injury and host factors create the environment in which microorganisms establish and persist. Furthermore, complete eradication of microorganisms is neither achievable nor desirable, as healing wounds normally harbour diverse microbial communities. The clinical objective is therefore not microbial sterility, but restoration of microbial balance and reduction of microbial burden to a level that allows progression of healing.

Current evidence supports two clinically relevant concepts:

- Persistent microbial communities, including biofilms, are associated with delayed healing and an increased risk of wound infection; and
- Interventions that disrupt these communities and optimise the wound environment can facilitate progression towards healing.

These assumptions are applied in the understanding of chronic/hard-to-heal wounds and their treatment[106, 237] and clinical strategies to maintain biofilm and microbial burden at levels the host defences can manage.

Identifying biofilms in a wound

Our understanding of how biofilms are identified within wounds has evolved alongside advances in biofilm science and contemporary concepts of chronic wound healing. Early theories[251–253] proposed that macroscopic visual appearance of the wound (e.g., observation of a fibrinous, necrotic and/or slimy surface substance) could identify the presence of biofilm. However, subsequent research has demonstrated that biofilms cannot be observed by the naked eye in biological systems such as a chronic wound, even though they are most likely present.[233, 254, 255] Advanced diagnostic techniques, which are not readily available in most clinical settings, are required to demonstrate biofilm in wound tissue.[233]

Research on wound samples shows that while biofilm may be the underlying cause of the appearance of some wounds,[230, 233] visible wound bed changes are not conclusive indicators of biofilm presence. Further, many wounds that appear to be healthy to the naked eye are shown via laboratory investigation to contain biofilm.[256]

At present, there is no universally accepted clinical gold standard for identifying biofilm within wounds. Although advanced imaging and microscopy techniques can demonstrate biofilm in research settings, these methods are not routinely available in clinical practice. Furthermore, confirmation of biofilm is not required for most treatment decisions, as wound management should be guided primarily by comprehensive clinical assessment rather than laboratory confirmation of biofilm. However, in some cases, the species of microorganisms present in the wound might be of clinical interest and inform treatment strategies.

Consider whether it is clinically necessary to diagnostically confirm biofilm in a non-healing wound.

If a wound is hard-to-heal and is not responding to standard protocols of care, tolerant microorganisms should be considered as one possible explanation. This includes microorganisms present as biofilm aggregates, embedded communities, or single cells within

Indicators that biofilm might be present in a wound

- Failure /recalcitrance to optimal wound management and appropriate antimicrobial treatment
- Delayed healing or deterioration on cessation of antimicrobial treatment
- Signs of low-level chronic inflammation
- Friable granulation / hypergranulation

the wound microenvironment. In the absence of laboratory-confirmed diagnosis, criteria that are indicative of possible presence of a wound biofilm are listed in the call out box.#

Clinical indicators should always be interpreted within the broader context of the individual, including wound aetiology, vascular status, inflammation, immune function, nutritional status, local environmental factors (such as pressure and shear) and the quality of wound management. Failure of a wound to respond despite optimisation of these factors may increase suspicion that microbial persistence, including biofilm, is contributing to delayed healing.

What does this mean for treating wounds?

Microorganisms organised as biofilms, microbial aggregates or other persistent communities may exhibit increased tolerance to topical and systemic antimicrobial agents. Consequently, successful management of wound infection should not rely solely on antimicrobial agents but should be used in conjunction with optimisation of the host, therapeutic wound cleansing, debridement, exudate management and products and devices with antimicrobial effect that are registered for use in wound infection management. Debridement is a fundamental component of wound bed preparation in chronic/hard-to-heal wounds. It removes slough, debris, necrotic tissue and other material that may harbour microorganisms, while also reducing inflammatory mediators and host-derived matrix components. As a result, debridement reduces microbial burden, disrupts microbial aggregates and biofilm-like structures, and improves the local wound environment.[62, 257-259]

Selection of the debridement method, intensity and frequency should be individualised according to the person's condition, wound characteristics, treatment goals and evidence of ongoing microbial proliferation or persistence. The primary objective is to debride the wound sufficiently to reduce microbial burden, remove barriers to healing and maintain a wound environment that supports tissue repair.

Importantly, debridement should be viewed as one component of comprehensive wound management rather than a biofilm-specific intervention. Optimal outcomes require simultaneous management of the underlying wound aetiology, restoration of tissue perfusion where appropriate, control of oedema, optimisation of nutrition and management of systemic factors, together with therapeutic cleansing, debridement, exudate management and antimicrobial stewardship. Together, these interventions modify the wound microenvironment and support progression towards healing.

These criteria were agreed in a formal Delphi process conducted by IWII in 2026 (to be published) with 20 wound infection experts from clinical, scientific and academic backgrounds around the world. The Delphi process involved three rounds of voting and discussion to reach agreement on clinical indicators. This updates the Delphi process and consensus agreement that IWII published in 2016 and 2022 and reflects our evolving understanding of tolerant microorganisms, biofilm and the wound microenvironment. In 2026, the following criteria were removed from the consensus list of indicators of possible biofilm presence: low-level local erythema (a sign of inflammation therefore already represented), increased wound exudate/moisture (too non-specific) and covert/secondary signs of infection (no expert consensus).

Chapter 8
Principles of aseptic technique in wound care

2026 updates and key points

- Aseptic technique should be practised within the framework of infection prevention and control. Adherence to standard precautions, effective hand hygiene, appropriate use of personal protective equipment, environmental controls, and clinician competency are fundamental to preventing microbial transmission and reducing the risk of wound infection.
- Use a risk-based approach to select an aseptic technique framework.
- Clean/standard aseptic technique does not appear to have inferior wound infection outcomes compared to sterile/surgical aseptic technique in varied wound care settings.
- Therapeutic wound and skin cleansing is a clinical procedure that includes cleansing of the wound bed, wound edge, periwound and surrounding skin.
- Developing standardised policies and procedures and supporting ongoing education are important roles of the local wound care service to promote competency and compliance with clinical practices that prevent and manage wound infection.

Introduction

Aseptic technique framework refers to the set of practices that are used to reduce the risk of introducing and spreading microorganisms during a wound dressing procedure (WDP). In the presence of any break in the skin (e.g., a wound) pathogens can be introduced from the:

- Surrounding environment (i.e., air, equipment and people, including the wound clinician),
- Surrounding skin (i.e., microflora that is usually present on the skin), and
- Other endogenous sources (e.g., the gastrointestinal or respiratory tracts).

In wound care, the aseptic technique framework should be selected according to the individual, the wound, the procedure and the care environment. The approach should protect the individual, clinician and environment while supporting wound healing, and should be practical and evidence-informed rather than ritualistic.

Aseptic technique frameworks for wound dressing procedures

The goal when performing any clinical procedure in which there is a break in the skin is to prevent introduction of pathogens.[260] Recognised frameworks for performing aseptic technique that are used in wound management to achieve this goal are sterile/surgical aseptic technique and clean/standard aseptic technique.[261-265]

Sterile/surgical aseptic technique

When performing a sterile/surgical aseptic technique, standard infection and control precautions are implemented, hands are cleansed with alcohol-based sanitiser or skin cleanser and running water and sterile gloves are worn. A sterile field is used with sterile equipment (including dressing pack, fluid well, scissors, forceps and cleansing solutions) and sterile wound dressings. Asepsis is maintained when preparing the wound dressing.[264, 266-268]

Clean/standard aseptic technique

Clean/standard aseptic technique is an adaptation of the above procedure that combines rigorous infection-prevention practice with selected clean, rather than fully sterile, items, as appropriate to the individual, the wound and what is possible in the clinical context.[261]

When performing a clean/standard aseptic technique, infection prevention and control standard precautions are implemented, hands are cleansed with an alcohol-based sanitiser or skin cleanser and running water, and non-sterile gloves are worn. Clean equipment, bowls, cleansing cloths and towels should be used for cleansing the periwound and the surrounding skin [Zones 2 and 3, **Figure 7**]. A basic dressing pack (i.e., a plastic tray with well, plastic forceps and gauze) is used. Sterile equipment (e.g., scissors, curette and forceps) and sterile fluid or potable water can be used to perform debridement.[261, 264, 267-270]

While some clinical guidance[268, 270] suggests that in the home care setting wound dressings could be cut with clean scissors and the unused dressing components stored between WDPs,[#] research has shown that non-sterile scissors remain contaminated after cleaning with disinfectant wipes.[271] Given the lack of consensus, wound dressings should be used and stored in accordance with the manufacturer's instructions. A risk-based approach should be applied

[#] This practice is considered acceptable in some clinical contexts (e.g. home-based care or resource-limited settings) where the scissors are cleansed with disinfectant and hot water, the wound dressings are for a single individual's use and are stored in a clean environment[270]

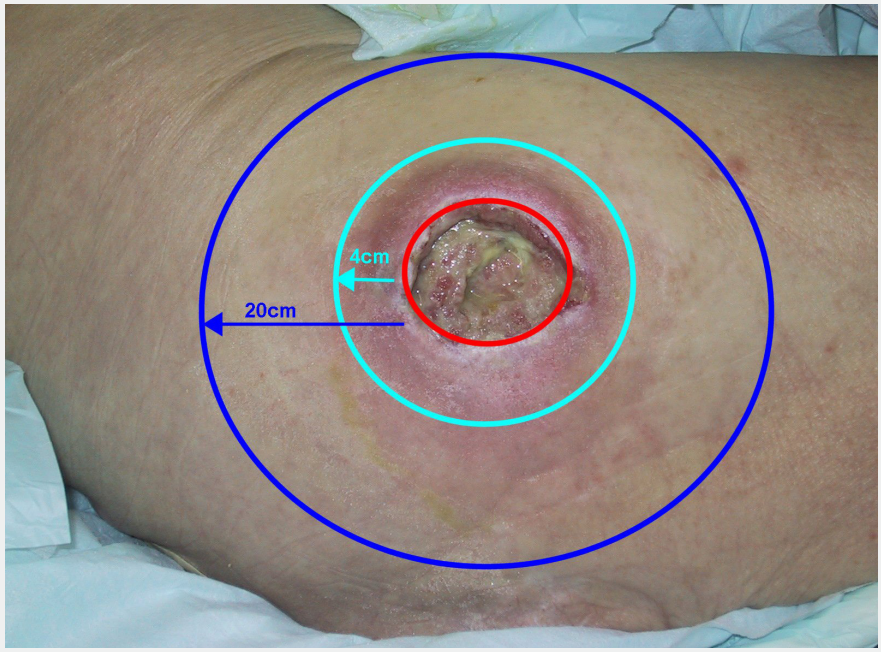


Figure 7. Therapeutic cleansing zones[155]

that considers the individual's clinical context, the wound, the care environment and relevant organisation/wound care service policies.

Principles to consider in selecting a technique for wound dressing procedures

1. Recognise that clean/standard technique is acceptable in many clinical settings

Recent evidence indicates that use of a clean/standard technique, rather than a sterile/surgical technique, does not increase the risk of wound infection across a range of clinical settings.[272] A systematic review and meta-analysis[272] found moderate-certainty evidence, with low risk of bias, that neither approach was inferior to the other for preventing wound infection. However, maintaining the conditions required for sterile/surgical technique can be difficult in uncontrolled or semi-controlled environments, such as home and community care.[261, 267] A pragmatic, context-sensitive approach should therefore be used, particularly outside highly controlled clinical environments such as wound clinics and tertiary healthcare services.[260, 261] Organisations/wound care services should develop local policies and procedures that specify the appropriate aseptic technique framework for WDPs, taking into account available resources, standards of care, the patient population and environmental risks.

Recommendation 6

Select either sterile/surgical aseptic technique or clean/standard aseptic technique when performing a wound dressing procedure. Conduct a risk assessment that considers the individual, the wound and environmental considerations to guide technique selection.

(Underpinning evidence: Level 1 evidence[269, 272])

2. Select an appropriate aseptic technique framework using a risk-based approach
Choose an aseptic technique framework based on a risk-based assessment of the individual, the wound, the procedure and the environment. A formal risk assessment to select between sterile/surgical or clean/standard technique should consider:[260, 264, 265, 268, 269, 273-277]

- Immune status of the individual,
- Size and location of the wound,
- Entry into anatomical cavities or organs,
- Extent of visualisation of the wound bed,
- Complexity of the procedure,
- Clinical setting, and
- Goal of care and preferences of the individual.

An evaluation of the clinical setting should consider:

- Environment in which the WDP will need to be performed,
- Availability of sterile versus clean equipment,
- Access to cleansing agents and wound dressings, and
- Scope of practice, confidence and competence of the clinician.

3. Always implement standard infection control precautions
Regardless of the aseptic technique framework that is selected, standard infection control precautions must be implemented.[278-280] These precautions provide the foundation for reducing contamination, transmission of microorganisms and healthcare-associated infection across all clinical settings.[281] Standard precautions are based on the principle that infection prevention measures should be applied consistently for every individual, irrespective of known or suspected infection status, and are informed by established scientific evidence.[278] The World Health Organization's global strategy for infection prevention and control highlights the principles of:[279, 280]

- Consistent implementation of hand hygiene at the point of care,
- Maintenance of environmental and system supports for safe practice (e.g., potable water, sanitation and hygiene), and
- Investment in staff education and competency in implementing infection prevention and control standard precautions and infection prevention and control.

Recommendation 7

Implement standard precautions when conducting a wound dressing procedure.

(Underpinning evidence: Level 1 evidence[278])

Hand hygiene, use of gloves and personal protective equipment (PPE) matched to the task (e.g., using a gown/apron, mask and eye protection if there is a risk of splash back or aerosolisation), safe waste management, and maintenance of a clean and organised clinical care space are all essential components of infection prevention and control standard precautions.[261, 264, 278, 279] Hand hygiene using an alcohol-based sanitiser or skin cleanser and running water, should be performed before and after:[261, 264, 279]

- Touching the individual's skin,
- Exposure risk to bodily fluids,
- Performing the WDP,
- Removing gloves, and
- Touching the individual's surroundings.

The immediate environment in which a WDP is performed should be clean, animal/pet-free, have low air flow (e.g., turn off fans/air conditioner), and the equipment workspace should be a clean, flat, non-porous surface that is disinfected before use.[269, 273, 282]

Consider therapeutic wound and skin cleansing as an essential component of the WDP
Current guidance places greater emphasis on therapeutic wound and skin cleansing.[155, 283] Cleansing should address all of the therapeutic cleansing zones: wound bed and wound edges (Zone 1), the periwound (Zone 2) and surrounding skin (Zone 3).[155] The surrounding skin and periwound (Zones 2 and 3) should be cleansed first using clean equipment and potable water or an appropriate cleansing solution. Next, the wound bed and wound edges (Zone 1) are cleansed using a technique, equipment and cleansing solution appropriate to the condition of the wound [see Chapter 9]. Cleansing should effectively remove debris, exudate and contaminants while protecting viable tissue, supporting wound healing, and minimising pain and discomfort.[155] Detailed guidance and recommendation are available in the *IWII's Therapeutic wound and skin cleansing: Clinical evidence and recommendations (2025)*.[155]

4. Match equipment to the aseptic technique framework
A crucial part of performing the WDP is matching the equipment used to the selected aseptic technique framework [see examples in **Table 14**]. Use sterile gloves for sterile/surgical aseptic technique and non-sterile gloves for clean/standard technique.[261] Instruments used for debridement or contact with key wound structures must be sterile.[261] When gloves or instruments become contaminated during the procedure, change them before proceeding to a clean or key part of care. Avoid re-using single-use items and follow manufacturer instructions for dressing storage, cutting and handling.[261]

Table 14: Equipment requirements for two different aseptic technique frameworks[264]

	Sterile/surgical technique	Clean/standard technique
Gloves	Sterile gloves (consider double gloves)	Non-sterile gloves
Field and equipment	Sterile field and sterile key equipment	Clean setup with sterile items used for key parts as required
Debridement instruments	Sterile	Sterile
Typical use	High risk individuals, deeper/complex wounds, exposed anatomical structures and/or complex procedures	Low risk individuals and wounds and settings where full sterility is not feasible
Core requirements	Maintain asepsis throughout all key parts	Minimise exposure to environmental contaminants with infection prevention and control standard precautions and a no-unnecessary-touch approach

5. Sequence the WDP to maintain asepsis
A safe WDP requires appropriate sequencing of the tasks required to apply infection prevention and control standard precautions, the selected aseptic technique framework and to achieve the goals of the WDP. As outlined in **Figure 8**, this includes:

- Performing hand hygiene,
- Reviewing the individual and wound history and clinical presentation,
- Preparation of the individual and the environment,
- Assembly of the equipment required for the WDP,
- Performing hand hygiene,
- Removal of the old wound dressing using appropriate gloves and equipment,
- Performing hand hygiene and donning appropriate gloves,
- Performing therapeutic wound and skin cleansing
- Establishing the sterile wound dressing field or maintaining sterile key parts,
- Performing hand hygiene and donning appropriate gloves,

- Performing debridement and post debridement cleansing as indicated,
- Reassessing the wound,
- Discarding gloves, performing hand hygiene and donning new gloves,
- Application of the new wound dressing with appropriate equipment,
- Appropriate disposal of waste,
- Removing gloves and performing final hand hygiene; and
- Documenting and communicating the wound assessment, evaluation and ongoing management plan.

A more detailed description of sequencing in a WDP is described in the IWII's *Therapeutic wound and skin cleansing: Clinical evidence and recommendations (2025)* that focuses more closely on cleansing technique.[155] Additionally, the ANTT® Clinical Practice Framework[264, 284] adds a useful operational lens by introducing the concepts of key sites (skin integrity breaks through which microorganisms can enter the body) and key parts (equipment that makes direct contact with the wound). After identifying key sites and key parts, the WDP should be performed in a way that protects key sites and key parts (i.e., key sites and key parts should only touch other key sites and key parts) and uses non-touch technique wherever feasible.[264, 284]

6. Document clinical reasoning when documenting the WDP
Good documentation should demonstrate that clinical reasoning underpinned by a risk-based approach was used to select an aseptic technique framework. This means that the considerations in the risk-based assessment and the conclusions made should be included in documentation. Comprehensive documentation should record consultation with and education given to the individual and their caregivers, assessment of the individual and the environment, assessment of the wound, the aseptic technique framework that was selected, the therapeutic wound cleansing and debridement that was performed, products used in the WDP, pain assessment and management, and concerns regarding the wound's progress towards healing, and the ongoing wound management plan. Using a clinical decision support tool or framework to guide documentation can facilitate accurate documentation of the clinical reasoning process.[285, 286]

7. Support clinical competency, and consistency with education and skills training
Effective aseptic technique depends on clinician competence as much as the framework and equipment that is used.[264, 287] Education should focus on reinforcing application of infection prevention and control standard precautions, selecting the correct aseptic technique framework, identifying key sites and key parts, understanding the importance of sequencing, using non-touch technique whenever possible, and escalating from a standard/clean aseptic technique approach to a more controlled sterile/surgical aseptic approach when the individual's risk factors or the technical complexity of the WDP increases. Clinical competency can be supported with appropriate education, standardised practice in the local wound care service, ongoing role modelling, and opportunities for regular practice and technique reinforcement.[287]

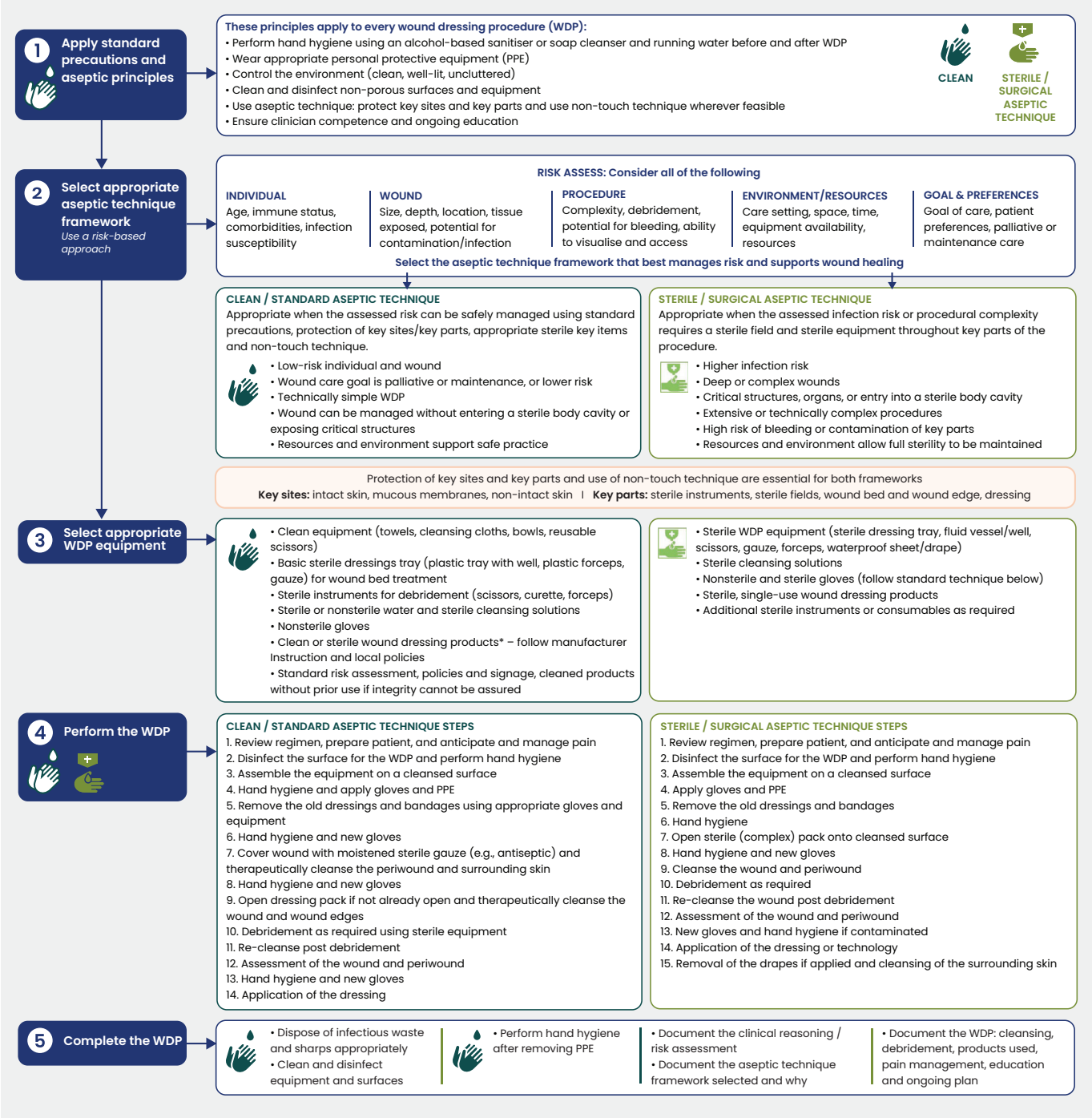


Figure 8. Wound dressing procedure using different aseptic technique frameworks[261, 264, 267-269, 273, 282]

Chapter 9
Contemporary wound bed preparation and management

Introduction

Aseptic technique framework refers to the set of practices that are used to reduce the risk of infection. Wound bed preparation (WBP) is a dynamic and ongoing process aimed at removing barriers to healing and optimising the wound environment to support tissue repair. Contemporary WBP has evolved beyond the management of non-viable tissue alone and now encompasses a broader understanding of the microbial, inflammatory and physiological factors that impair healing.[288, 289]

Current approaches draw on several complementary frameworks, including:

- Biofilm-Based Wound Care (BBWC),[258, 290]
- Continuous Integral Debridement,[283, 291]
- Therapeutic Wound and Skin Cleansing,[155]
- TIME,[148, 292]
- TIMERS,[293]
- Wound Balance,[294] and
- Wound Hygiene,[262, 295]

Despite differences in terminology and emphasis, these frameworks consistently advocate the following principles:

- Assess the individual and identify their risk factors for infection and delayed healing,
- Manage factors that delay healing and increase the risk of infection,
- Implement local strategies to optimise the wound microenvironment,
- Therapeutically cleanse the surrounding skin, periwound and wound,
- Debride the wound, wound edges and periwound of non-viable tissue when clinically indicated and within scope of practice,
- Re-cleanse following debridement,
- Apply antiseptics consistent with the clinical need and preferences of the individual,
- Manage factors that increase risk of infection (e.g., excess wound exudate), and
- Consider alternating antiseptics when clinically indicated to provide broader antimicrobial activity while supporting antimicrobial stewardship (AMS).

Local factors that delay wound healing and increase risk of infection

The overarching goal of WBP is to identify and address both microbial and non-microbial barriers to healing (see call out box). Effective WBP requires repeated assessment of these factors throughout the healing journey[283, 296] so that appropriate WBP can be planned, implemented and re-evaluated when progress towards healing is not as expected.

Local barriers to healing include: necrotic tissue, slough, biofilm, excessive exudate, inflammatory mediators, foreign material, callus, non-advancing wound edges and impaired periwound and surrounding skin integrity.

Therapeutic wound and skin cleansing

The IWII's *Therapeutic Wound and Skin Cleansing* clinical guidance[155] identifies therapeutic cleansing as a fundamental component of WBP. Therapeutic wound and skin cleansing is defined as the active removal of contaminants, debris, non-attached devitalised tissue, microorganisms, excess exudate and residual dressing materials from the wound, wound edge, periwound and surrounding skin. Unlike routine washing, therapeutic cleansing is performed with the specific objective of improving the wound environment and addressing local barriers to healing.[155]

Based on a systematic review of the evidence, the IWII makes several recommendations on when and how therapeutic wound cleansing should be performed as a component of WBP.

Recommendation 8

Therapeutically cleanse the wound bed and wound edge, the periwound skin and the surrounding skin when the wound dressing procedure is performed.

(Underpinning evidence: Level 3 evidence[297, 298] and Level 5 evidence[141, 148, 295, 299])

There is an association between more frequent therapeutic cleansing and faster healing of chronic/hard-to-heal wounds (pressure injuries specifically).[298] In clinical practice, regular therapeutic cleansing of the wound is advantageous, particularly for wounds that are more likely to be exposed to contaminants (e.g., faecal material).[298]

In many wounds, the need for therapeutic cleansing will be immediately evident from the visual condition of the wound bed, particularly when debris or non-viable tissue is present. However, even in healing wounds with healthy granulation tissue, there may be microbial contamination and non-visible debris (e.g., adhesive) in and/or around the wound. Several early clinical studies have demonstrated that wound cleansing can reduce the microbial burden to levels that enable the host to manage and prevent infection.[300, 301] Additionally, the process of cleansing the wound enables better visual assessment of the wound bed, manages exudate and odour, and promotes the individual's overall feeling of wellbeing.[302]

There has been ongoing debate as to whether a wound requires cleansing prior to taking a sample for diagnostic purposes.[189] There are no studies directly comparing the diagnostic accuracy of wound swabs or biopsies between cleansing or not cleansing the wound prior to collecting the sample. However, diagnostic studies that have compared the validity of different specimen collection methods include cleansing with an inert solution (e.g., sterile normal saline) as a component of swabbing and biopsy procedures.[193, 198, 303] Cleansing the wound with an inert solution loosens or removes surface contaminants and reduces the likelihood of false-positive results when taking a wound or tissue sample for microscopy, culture and sensitivity. Cleansing the wound also improves visibility of the wound bed to ensure that the sample is collected correctly.

The therapeutic cleansing zones include the following three zones: the wound bed and wound edge, the periwound and the surrounding skin. All areas within the therapeutic cleansing zone require therapeutic cleansing.[155] Use of a skin cleanser to cleanse the periwound skin has been shown to be associated with an immediate reduction in microbial counts.[297] The periwound may also have an accumulation of moisture in the region covered by the wound dressing.

Debridement

Debridement is a fundamental component of WBP. Debridement is defined as the removal of viable and non-viable wound components, including necrotic tissue, slough, microorganisms, biofilm, extracellular polymeric substances (EPS) and foreign material. Debridement reduces both microbial and non-microbial barriers to healing, facilitates wound assessment and promotes progression towards healing.

Recommendation 9

Perform debridement when clinically indicated. The method and frequency of debridement should be selected based on the individual's clinical history and risk factors, assessment of the wound, goals of care, clinician's scope of practice, individual preferences and available resources.

(Underpinning evidence: Level 1 evidence[304-308])

Regular debridement is recommended whenever clinically appropriate because barriers to healing frequently re-accumulate over time. For most wounds, debridement should therefore be considered an ongoing process rather than a single intervention.[283] Level 1 evidence[304-308] has demonstrated the effectiveness of debridement in promoting wound healing in a range of chronic/hard-to-heal wound types. Surgical debridement and sharp debridement provide some of the most rapid options for removing unviable tissue and are therefore favoured in severe infection or extensive necrosis,[304] when within the scope of practice of the clinician. Several reviews provide comprehensive head-to-head comparisons of different debridement methods.[304, 308] However, in clinical practice, a wide range of considerations are required when selecting a debridement method, as summarised in **Table 15**.

Complementing debridement

Although distinct interventions, therapeutic cleansing and debridement should not be viewed as separate activities. Rather, they function synergistically within WBP, with cleansing facilitating the removal of loose contaminants and debris and debridement addressing attached barriers such as biofilm, slough, callus and necrotic tissue. Together they contribute to reduction of microbial burden, removal of inflammatory stimuli and optimisation of the wound environment.[155, 283, 291] The integration of therapeutic cleansing into WBP reflects growing recognition that the wound, wound edge, periwound and surrounding skin function as a single wound ecosystem. Research demonstrates that microorganisms, biofilm and inflammatory burden frequently extend beyond the visible wound bed into adjacent tissues. Consequently, effective WBP should include therapeutic cleansing not only of the wound surface but also of the wound edge, periwound and surrounding skin.[155, 309]

A major focus of contemporary WBP is the management of biofilm. Understanding of wound biofilm continues to evolve and is discussed in greater detail in Chapter 7. Biofilm is present in an estimated 78% of chronic/hard-to-heal wounds and contributes to persistent inflammation, antimicrobial tolerance and delayed healing.[61, 233] Biofilm-associated microorganisms exist within a protective EPS matrix that limits host immune responses and reduces the effectiveness of host defences and many antimicrobial therapies.[242] Contemporary understanding also recognises that microorganisms are not confined to the wound surface. Biofilm aggregates may extend into deeper tissue, wound edges and periwound.[61, 310, 311] This highlights the importance of interventions that address the entire wound ecosystem rather than the wound bed alone, for example debriding hyperkeratotic or calloused tissue to restore a viable wound edge.[61, 310, 312] Because biofilm rapidly reforms following disruption, management requires repeated intervention. Therapeutic wound cleansing and debridement work synergistically to disrupt, detach and remove biofilm and its associated inflammatory burden, forming the cornerstone of biofilm-based wound care.[155, 283, 296]

Contemporary WBP is also informed by the International Wound Infection Institute Wound Infection Continuum **[IWII-WIC, see Figure 5]**.[141] This framework reinforces the importance of early intervention to manage increasing microbial burden before clinical infection develops, highlighting the role of therapeutic cleansing, debridement and appropriate antimicrobial stewardship in preventing infection and the progression of infection along the continuum.[310]

Emerging evidence suggests that the infectious microenvironment—including oxygen gradients, pH, moisture balance, nutrient availability and host immune responses—influences microbial behaviour, biofilm persistence and treatment responsiveness.[242] The zone model proposed by Kirketerp-Møller, Stewart and Bjarnsholt (2020)[61] further emphasises the complex interactions between bacteria, biofilm, host responses and local tissue conditions. Understanding and modifying this microenvironment is increasingly recognised as an important aspect of WBP and infection management.

Therapeutic wound and skin cleansing also contributes to the management of wound-associated inflammation. Chronic/hard-to-heal wounds often contain elevated concentrations of proteases, inflammatory cytokines and cellular debris[313] that perpetuate tissue damage and impair healing. Removal of these factors through cleansing and

Table 15: Debridement* methods for contemporary wound bed preparation[283, 304, 306, 307]

Method	Category	Examples	Mechanism / Role in WBP	Key Indications	Clinical considerations/ referral triggers
Surgical	Standalone / most invasive	Operating theatre debridement; hydrosurgery	Excision of devitalised tissue, biofilm, foreign material.	Extensive necrosis, deep infection, abscess	• Fast and efficient; source control • Requires specialist expertise • WBP needed
Selective sharp	Standalone	Scalpel, scissors, curette	Removes devitalised tissue and disrupts attached mature biofilm.	Hard-to-heal wounds with slough, necrosis, callus	• Rapid and precise • Assess pain, perfusion and bleeding risk
Conservative sharp	Standalone / maintenance/ adjunct	Sterile curette, scalpel or scissors	Removes loose non-viable tissue with minimal pain and bleeding.	Maintenance debridement	• Repeatable and low trauma • Escalate if necrosis, infection or non-progression persists
Mechanical	Standalone or adjunct	Debridement pads, monofilament pads, irrigation, negatively charged fibres	Physical disruption/removal of debris and biofilm.	Sloughy wounds; local infection	• Accessible and effective • Use alternatives to wet-to-dry dressings where possible
Technical	Standalone	Hydrosurgery, ultrasound/ ultrasonic, NPWTi-d	Removal/disruption of tissue and biofilm.	Complex wounds	• Efficient and precise • Requires equipment, training, referrals
Biological / larval	Standalone	Lucilia sericata larvae Lucilia cuprina larvae	Selective necrotic tissue and biofilm removal.	Necrotic/sloughy wounds	• Highly selective • Consider acceptability, pain, bleeding risk
Autolytic	Adjunct	Hydrocolloids, alginates, hydro-responsive dressings,	Supports endogenous breakdown of devitalised tissue.	Most wound types	• Selective and often painless, but slower • Caution with acute infection or ischaemia
Osmotic	Adjunct	Honey; hypertonic dressings	Softens and liquifies devitalised tissue.	DFU, VLU, pressure injury	• Supports autolysis • Monitor exudate and maceration
Enzymatic	Adjunct	Collagenase	Breaks down devitalised tissue.	Hard-to-heal wounds	• Selective • Avoid incompatible products
Oxidative	Adjunct	Cold atmospheric plasma	Reduces microbial burden and disrupts biofilm.	Infected wounds	• Antimicrobial potential • Requires equipment and training
Chemical	Adjunct	Desiccating gels	Desiccates slough and biofilm.	Selected wounds	• Useful alternative • Observe contraindications
Chemo-mechanical	Adjunct	Hypochlorite gel	Chemical softening followed by removal.	DFU and leg ulcers	• Facilitates loosening and subsequent removal of non-viable tissue • Rapid action
Surfactant	Adjunct	Surfactant cleansers	Facilitates removal of debris, microorganisms and biofilm components.	Most wound types	• Supports therapeutic cleansing • Check compatibility and sensitivities.

* Continuous integral debridement incorporates therapeutic wound and skin cleansing, biofilm management, wound edge/periwound care and repeat assessment throughout healing.

debridement helps restore a more favourable wound environment and supports progression through the normal phases of healing. This concept aligns with the contemporary understanding that WBP must address both microbial and non-microbial impediments to healing.[283, 291, 314]

The importance of systematic WBP is reflected in contemporary wound outcome data. Analysis of more than 19,000 wounds managed within community healthcare settings demonstrated that more than 40% were static, non-healing or deteriorating and that almost half had persisted for more than three months.[315] These findings reinforce the need for proactive and repeated WBP strategies that address tissue, inflammation, infection, biofilm, moisture balance and wound edge advancement throughout the wound healing trajectory.[315]

Therefore, contemporary WBP should be viewed as a continuous cycle of assessment, therapeutic wound and skin cleansing, debridement, biofilm management and optimisation of the wound environment.[291] Together, these interventions create a wound environment more conducive to healing by reducing microbial burden, controlling inflammation, removing physical barriers and supporting tissue repair.[79, 155, 283]

Exudate management

Effective exudate management is an integral component of wound bed management. Managing wound exudate plays a role in reducing wound infection risk without relying on antimicrobial activity or contributing directly to antimicrobial selective pressure. Excessive or poorly controlled exudate can cause periwound maceration and loss of skin barrier integrity, creating conditions that facilitate microbial penetration and wound deterioration. Exudate can contain microorganisms, inflammatory mediators and proteolytic enzymes that perpetuate inflammation and impair healing.[316-319] Maintaining an appropriate moisture balance, while containing and removing excess exudate, helps protect surrounding skin, support tissue repair and potentially reduce infection-related complications.

Some non-medicated wound dressings **[Table 16]** have physical properties that modulate wound moisture. Although these dressings are increasingly being used to reduce the risk of wound infection, most are not currently registered/licensed as a device specifically for infection management, and their properties do not imply an ability to kill or inhibit the action of microorganisms (unless combined with medicated additives).

Properties include one or more of the following:[7, 316, 317, 319-321]

- Sequestration: dressing materials pull and lock away microorganisms and other harmful components such as exudate and matrix metalloproteinases (MMPs), reducing their availability within the local wound environment.
- Immobilisation: dressing materials hold microorganisms and/or toxins in place, stopping them from moving around or spreading across the wound surface.
- Retention or modulation of wound moisture: continued holding of microorganisms and/or other components within dressing material after initial capture or binding limits their release back into the wound environment.

Additionally, properties of some of these non-medicated wound dressings also facilitate autolytic debridement.[317, 321, 322] Clinical evidence demonstrating direct infection related benefits is limited.[316, 319, 320, 322, 323]

Non-medicated exudate management dressings provide an option with no known AMR risk. They can be used as adjuncts within a multimodal approach to wound infection management that includes optimisation of host factors, therapeutic cleansing and debridement, and appropriate use of topical or systemic antimicrobial agents.

Ongoing assessment of the wound bed and exudate is required to ensure that the selected dressing remains appropriate. Some dressings designed to absorb or manage exudate may be unsuitable for wounds with insufficient moisture, as they can adhere to the wound surface and potentially disrupt healing. Conversely, wounds producing large volumes of exudate may

Table 16: Characteristics of wound dressings with exudate management properties		
Dressing Type	**Mechanisms of action**	**Clinical considerations**
Gelling fibre dressings (e.g. carboxy-methylcellulose, PVA dressing)	• **Sequestration:** forms gel with exudate • **Immobilisation:** entrapment within gel • **Retention:** maintains moist bed and prevents exudate pooling	• Absorbs and traps exudate and microbes • Reduces maceration • Supports autolytic debridement and wound bed preparation • Useful under secondary layer in exuding wounds • Good gelling and conformability • Gel can adhere if dried, not appropriate for low-exuding wounds
Moisture-managing wound dressings (e.g., hydrogel, hydrofibre, hydrocolloid, hydroconductive, Ringer's solution–activated polyacrylate dressing)	• **Sequestration:** hydrofibre/ hydrocolloid • **Immobilisation:** certain hydrofibre/gel/polyacrylate • **Retention:** Donates or absorbs moisture depending on product	• Traps exudate and microbes in absorptive types or donates moisture for autolysis in necrotic wounds • Selection of product depends on exudate level in infected wounds • Must match wound exudate: hydrogels unsuitable for heavy exudate
Superabsorbent dressings	• **Sequestration:** traps exudate when it gels • **Immobilisation:** entraps microbes in gel/polyacrylate, facilitating their removal from the wound surface • **Retention:** high absorption	• Removes exudate and suspended bacteria • Very high absorption prevents exudate pooling • Reduces maceration and malodour • Useful adjunct in heavily exuding infected wounds when combined with debridement/ • antimicrobial agents • Silicone SAP dressings are also indicated for low exuding/ dry wounds
Negatively charged fibre desloughing dressings	• **Electrostatic attraction:** negatively charged fibres bind with slough and microbial colonies, allowing removal.[317] • **Retention/adherence:** Interaction is sufficiently strong to adhere to slough, facilitating removal from the wound surface.[321]	• Liquefies slough (solid or gelatinous) allowing for ease of therapeutic cleansing and debridement • Bacteria (both live and dead) have been recovered from dressings after removal • Awareness of the appearance of the wound dressing residue is a point of education for carers

Table 17: Evaluating wound exudate[326]		
Exudate level	**Inspection of dressing on removal**	**Evaluation of wound bed**
None	Dry dressing	Dry wound bed
Scant	No measurable moisture on wound dressing	Wound bed moist
Small/minimal	<25% of wound dressing involved	Wound bed very moist
Moderate	25%–75% of wound dressing involved	Wound bed wet
Large/copious	>75% of wound dressing involved	Wound filled

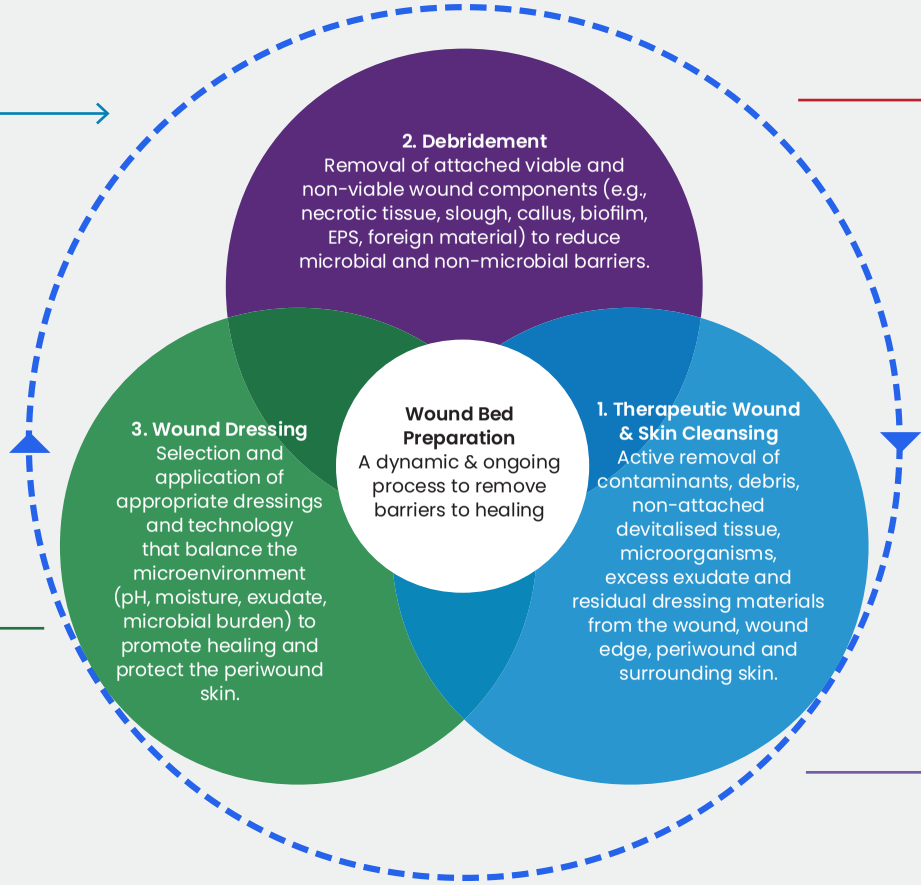
require frequent dressing changes even when highly absorbent dressings are used.[324, 325] **Table 17** provides a general guide to assessing exudate levels through inspection of the wound bed and used dressing. Clinical estimation of exudate volume can be imprecise and should be interpreted in the context of dressing type, absorptive capacity and frequency of dressing changes.

Assess & Plan

- Assess patient and wound
- Identify and manage factors that delay healing
- Determine position on the IWII Wound Infection Continuum
- Develop an individualised plan with patient goals and preferences
- Select appropriate cleansing, debridement and topical strategies

Support Healing

- Optimise moisture balance and tissue perfusion
- Protect periwound skin and manage exudate
- Encourage patient self-care and address lifestyle factors (nutrition, smoking, activity)
- Apply advanced dressings and adjunctive therapies as indicated
- Reassess and repeat WBP at each dressing change and throughout healing

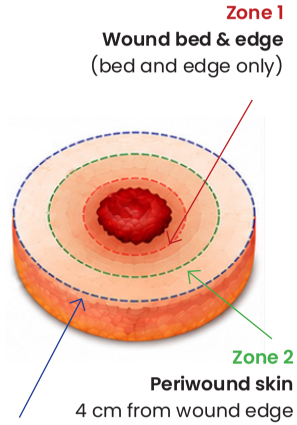


Infection Prevention & Management

- Monitor for increasing signs of infection and progression on the IWII Wound Infection Continuum
- Early intervention to prevent escalation to spreading or systemic infection
- Use appropriate systemic or topical antimicrobials when clinically indicated
- Apply antimicrobial stewardship principles
- Reassess regularly and escalate care when required

Zones for cleansing & debridement

Cleansing and debridement should include all zones to reduce microbial burden, remove inflammatory stimuli and optimise tissue health.



Key outcomes

Reduced microbial burden and biofilm	Reduced inflammation and exudate	Improved tissue health and perfusion	Enhanced granulation and epithelialisation	Prevent infection and promote healing

Repeat the cycle at each dressing change and throughout the healing journey.

Wound bed preparation is not a one-time event; it is an ongoing, dynamic process.

Figure 9. The integrated wound bed preparation

Chapter 10
Antimicrobial agents in wound infection

2026 updates and key points

- Most wounds heal on a normal trajectory and do not require the use of antimicrobial agents.
- Antimicrobial therapy is an adjunct to good wound care that consists of host optimisation, therapeutic cleansing, debridement and exudate management.
- Use of antiseptics should be evaluated based on the individual's risk factors and signs and symptoms of wound infection. Prophylactic antisepsis could be considered for individuals at high risk of wound infection.
- Systemic antimicrobial therapy is indicated for signs and symptoms of spreading and systemic infection and may also be appropriate for some individuals with overt signs and symptoms of wound infection and high-risk factors.
- Selection of an antiseptic should consider the individual and their wound (e.g., clinical history, wound presentation and risk factors), goals of care (e.g., need for symptom management), profile of the product (e.g., cytotoxicity, therapeutic index, pH, etc.) and resources and policies of the wound care service.
- All antimicrobial agents should be used judiciously and consistent with principles of antimicrobial stewardship, safety and compliance with policies and procedures of the wound care service.
- Monitoring and adjustment of treatment is required. This includes rapid escalation of treatment with progression of signs and symptoms of wound infection, continuing antiseptic or product with an antimicrobial effect for two weeks before adjusting treatment and ceasing treatments when signs and symptoms resolve, unless there is a high risk of re-infection, in which case treatment may be continued.

Introduction

Antimicrobial agents are a cornerstone intervention for managing clinically identified infection alongside optimisation of the host, therapeutic cleansing, debridement and exudate management.

Antiseptics and systemic antibiotics are the most commonly used antimicrobial agents in wound care. Antiseptics, of which there are many types **[Figure 10]**, are biocides that are toxic to bacteria but specifically prepared for application directly to living tissue to treat local infection or prevent advancing colonisation and biofilm formation/recurrence. They are applied directly to the wound bed and/or periwound skin, in the form of solutions, wound dressings and/or gels.[327, 328]

Systemic antibiotics are drugs that are toxic to bacteria. They are administered into the body (usually orally or intravenously) so the medication can be spread through the vascular system to reach infection around the body.[329] While antifungals, antiparasitics, and antivirals are indicated in specific wound aetiologies, their use in wound care is much less common.

A comprehensive table of antiseptic products is provided in **Appendix A: Profiles of commonly used products for wound infection management and wound/skin cleansing**

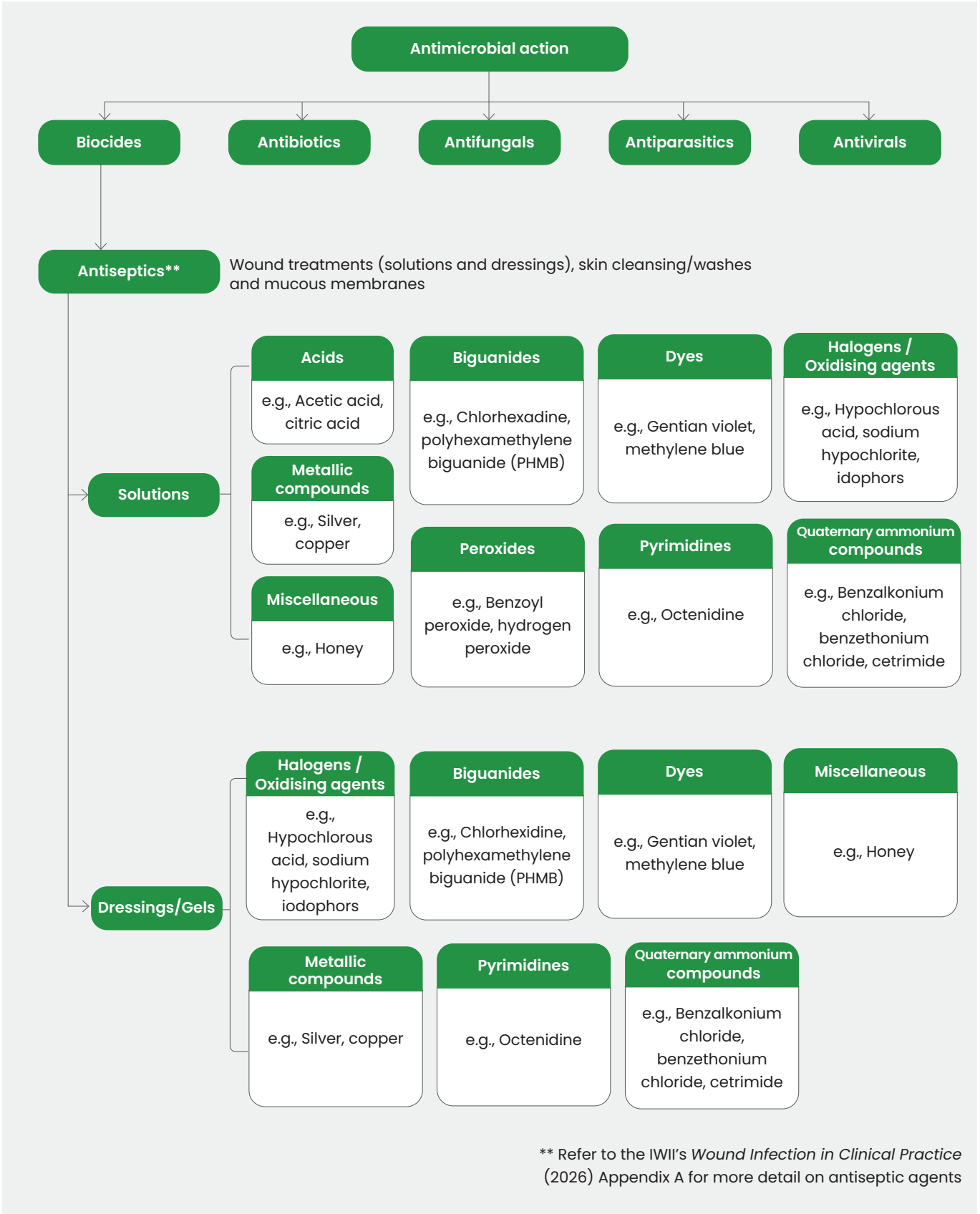


Figure 10. Classification of antimicrobial agents[6,7] (adapted from Idensohn et al. 2026[8] with permission)

Use of antiseptics

Most wounds do not require the use of antiseptics, but they are indicated for:[141, 155, 330]

- Individuals with a higher risk of invasive infection (e.g., those who are immunocompromised due to comorbidities) and
- Wounds suspected or confirmed to have high or increasing microbial burden [refer to **Figure 5: IWII -WIC**].

Recommendation 10

Use an antiseptic as part of a comprehensive wound infection management plan when wound infection is confirmed or suspected. Consider using an antiseptic in individuals at high risk for invasive wound infection based on a risk assessment.

(Underpinning evidence: Level 1 evidence[330-337])

Various systematic reviews[330-337] have compiled the evidence on antiseptic use in wound management. Individual clinical studies provide evidence that various antiseptics have a role in reducing infection, reducing signs and symptoms of local wound infection, promoting complete wound healing or improving the type of tissue in a wound bed. Some other benefits are also reported, including low risk of adverse events and high levels of satisfaction from clinicians and individuals with wounds.[338, 339] Some evidence also indicates that early use of antiseptics can suppress the development of biofilm.[340, 341]

However, the available research has methodological limitations and confounding factors that reduce its generalisability.[141, 155] These include failure to confirm the presence of wound infection or microbial contamination at baseline, limited reporting of the individual's clinical status, and variation in concurrent wound care regimens. There is also a lack of comparative effectiveness trials and clinical studies using standardised methodologies and outcome measures, limiting meaningful comparison between antiseptic agents and treatment approaches. Much of the research on the efficacy of antiseptics has explored their effects *in vitro* or in animal wound models.[342] Microorganisms may behave differently under laboratory conditions than within the complex wound environment, particularly when organised within biofilms.[242] In addition, the concentrations, exposure times, application methods and testing conditions used in laboratory research may differ substantially from the way antiseptics are used clinically.[289, 343, 344] Consequently, antimicrobial activity demonstrated *in vitro* should not be assumed to translate directly into improved clinical outcomes, and caution is required when extrapolating findings from laboratory and preclinical research to wound care practice.

Selection of antiseptics

Ensuring the appropriate selection and use of antimicrobial agents is important for achieving desired outcomes for the individual and their wound, minimising adverse effects and supporting antimicrobial stewardship.[8, 51, 345] Antiseptic selection should be guided by a comprehensive assessment of the individual and the wound, including risk factors for wound infection and delayed healing, the wound presentation (e.g., signs and symptoms of infection, microbial findings where clinically indicated), and the goals of care, such as reducing microbial burden, managing problematic signs and symptoms, or supporting wound healing.

Several factors should be considered when selecting an antiseptic. These include:

- Antimicrobial spectrum and resistance considerations, including activity against microorganisms likely to be clinically relevant.
- Properties of the active ingredient and formulation, including antimicrobial effectiveness, concentration, method and rate of release, duration of activity, and required contact time.
- Cytotoxicity, pH and tissue compatibility, particularly where repeated or prolonged exposure may affect viable cells involved in wound healing.
- The individual's clinical circumstances, including allergies or sensitivities, pain, wound location, comorbidities, and ability to tolerate the intervention.

- Goals of care, with ongoing review to determine whether antimicrobial treatment remains clinically indicated.
- Practical considerations, including availability, cost, required resources, clinician familiarity, and organisation/wound care service policies.

Recommendation 11

Select an antiseptic based on:

- **Antimicrobial spectrum and resistance,**
- **Properties of the active ingredient,**
- **Cytotoxicity, pH and allergenicity,**
- **Goals of care and other individual factors, and**
- **Resources, availability and organisation/wound care service policies.**

(Underpinning evidence: Level 1 evidence[331-336])

Antimicrobial resistance

All antimicrobial agents should be used judiciously and in conjunction with therapeutic cleansing, debridement, exudate management and other appropriate wound management strategies. Antiseptic therapy should be commenced only when clinically indicated, used for the shortest appropriate duration, and regularly reviewed to determine whether ongoing antimicrobial treatment is required. Where the clinical indication has resolved or the treatment goals have been achieved, antiseptic therapy should be discontinued or stepped down to non-antimicrobial wound management.

Antiseptics have broad-spectrum effects on microbes, depending on the active ingredient, formulation and concentration of the preparation. Most antiseptics act through multiple antimicrobial mechanisms and therefore have a low propensity for the development of microbial resistance.[328] Appropriate use of antiseptics therefore has the potential to assist in controlling microbial burden in wounds while reducing unnecessary exposure to systemic or topical antibiotics and supporting efforts to minimise AMR.[51, 345] However, this does not remove the need for antimicrobial stewardship. Unnecessary, prolonged or indiscriminate use of antiseptic products should be avoided. When selecting and reviewing antimicrobial therapy, consider organisation/wound service policies, formularies and clinical pathways developed within an antimicrobial stewardship program (AMSP). Chapter 2 provides further guidance on the responsible prescribing and use of antimicrobial agents.

Cytotoxicity

Antiseptics are not completely selective for microbial cells and may also adversely affect mammalian cells involved in wound healing. Cytotoxicity is influenced by the active ingredient, formulation and concentration, as well as the duration and frequency of exposure.[346] If the toxicity of an antiseptic to mammalian cells is clinically significant, it has the potential to impair wound healing. However, findings from *in vitro* cytotoxicity studies should be interpreted cautiously because laboratory exposure conditions may not reflect concentrations, contact times and interactions with wound fluid and tissue encountered in clinical practice.

Some older antiseptic agents have historically demonstrated substantial cytotoxicity when used at higher concentrations or under specific exposure conditions. Contemporary antiseptics therefore increasingly use agents, concentrations and formulations intended to achieve an appropriate balance between antimicrobial activity and tissue compatibility.[345, 347]
One example is iodine, which can be delivered as cadexomer iodine in which iodine is incorporated within a starch-based matrix and released progressively as the dressing absorbs exudate. Alternatively, iodine can be delivered as povidone-iodine (PVP-I), in which iodine is complexed with povidone and is not slow-released. Controlled availability or release of the active antiseptic may reduce tissue exposure while maintaining antimicrobial activity. The potential for cytotoxicity should therefore be considered in relation to the specific antiseptic, formulation, concentration, method of application and exposure time. The lowest effective

concentration and appropriate duration of exposure should be used in accordance with the product formulation, clinical indication and available evidence.

In resource-limited settings, access to contemporary wound antiseptic formulations may be restricted. Where alternative antiseptics with more favourable tissue-compatibility profiles are unavailable, selection should be based on the best locally available option and consideration of the balance between the potential harm associated with uncontrolled wound infection and the potential cytotoxic effects of treatment. Where agents with recognised concentration-dependent cytotoxicity are used, appropriate dilution, minimum effective exposure and regular review of the wound response are particularly important.

Therapeutic index/biocompatibility index

Biocompatibility data describe how well an antiseptic performs against microorganisms relative to its potential to damage mammalian cells or tissues. The balance between achieving toxicity to microbes while minimising harm to mammalian cells has been measured and reported as therapeutic index[346] or biocompatibility index.[348] These are ratios that quantify the balance between an antiseptic's *in vitro* antimicrobial efficacy and its cytotoxicity to mammalian cells **[Figure 11]**.[348]

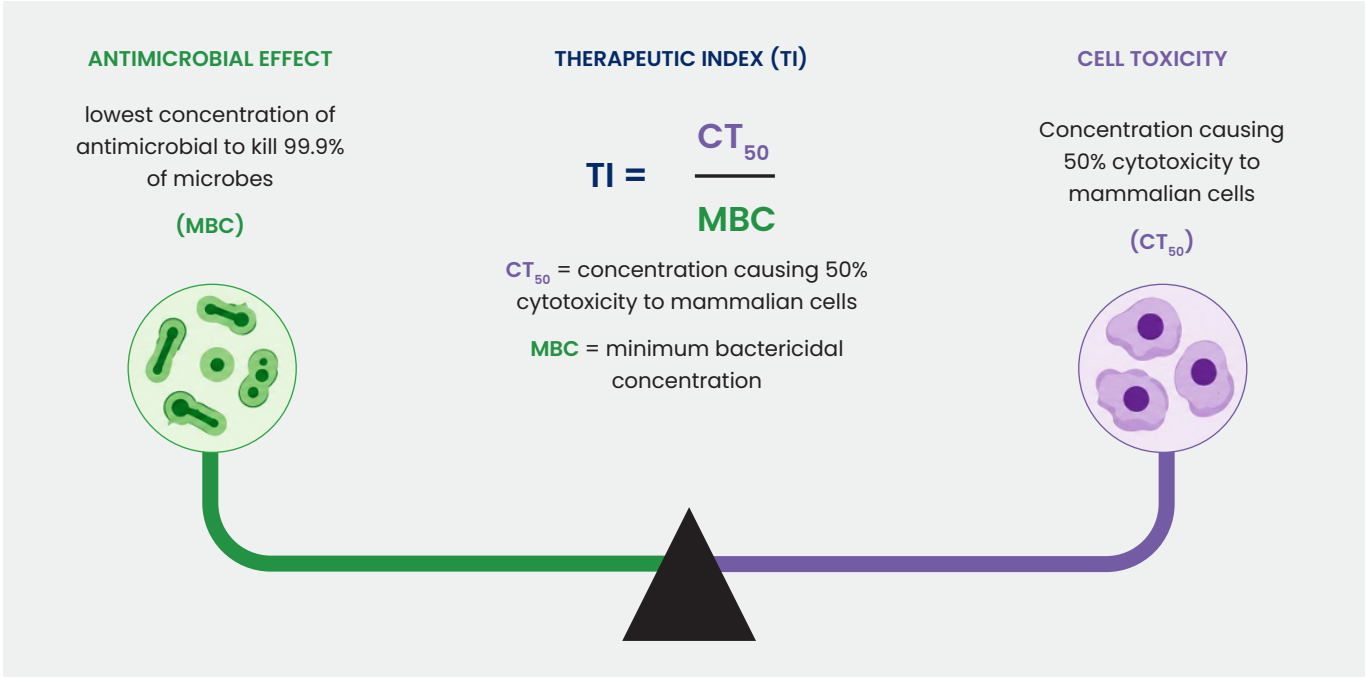


Figure 11. Therapeutic index[349, 350] and biocompatibility index[348] as calculated in laboratory studies

Therapeutic or biocompatibility indices may provide useful supplementary information when selecting an antiseptic. When making judgements regarding antiseptic products, clinicians are encouraged to inquire about any index testing results conducted by manufacturers, and to clarify the concentration of solutions used in testing. **Table 18** summarises therapeutic indices and biocompatibility indices of antiseptics reported in several studies. Higher index values indicate greater separation between bactericidal concentration and cytotoxic concentration, which means that as the index increases, the destruction of mammalian cells decreases.[333, 349, 350] The lower the index, the higher the toxicity to both microbes and mammalian cells.

Table 18: Reported therapeutic and biocompatibility indices for commonly used antiseptics*

Antiseptic	Therapeutic (TI) and biocompatibility indices (BI)# for tested organisms			
	S. aureus	MRSA	*E. coli*	*P. aeruginosa*
Benzalkonium chloride	BI: 0.79[348]		BI: 0.63[348]	
Chlorhexidine 20%	BI: 0.98[348]		BI: 0.83[348]	
Hypochlorous acid^ (experimental preparations of ≈0.000029–0.000066%)	TI: ≈30[349]		TI: ≈70[349]	TI: ≈60[349]
Hypochlorous acid 0.005%/sodium hypochlorite 0.005% mixture	TI: 9.58[350]	TI: 9.58[350]		TI: 19.18[350]
Octenidine 0.1%	BI: 2.11[348]		BI: 1.73[348]	
PHMB 0.1% with betaine	TI: 2.23[350]	TI: 1.78[350]		TI: 5.95[350]
PHMB 0.1% with poloxamer	TI: 3.26[350]	TI: 3.26[350]		TI: 6.51[350]
Povidone iodine 10%	BI: 0.68[348] TI: 0.95[350]	TI: 0.95[350]	BI: 0.68[348] TI: 0.95[350]	TI: 0.95[350]
Sodium hypochlorite (experimental preparations of ≈0.000074 to 0.000372%)^	TI: ≈10.7[349]		TI: ≈13.4[349]	TI: ≈53.7[349]
Silver sulfadiazine			BI: <0.006[348]	

* Therapeutic index and biocompatibility index values are dependent on the methods, organisms, mammalian cell types, antiseptic concentrations, exposure times and other variables used in testing. Values from different studies are not necessarily directly comparable. Values should be interpreted within the context of the methods used in each study rather than as an absolute ranking of antiseptic products.
# A value greater than 1 generally indicates that most microbes have been destroyed while not destroying more than 50% of mammalian cells.[348] No validated cut-off thresholds have been established.
^ Concentration and TI values extrapolated from the data available in the publication.

pH

The stratum corneum of the skin has a slightly acidic pH of 4 to 5.5, although this may vary according to factors such as age and anatomical location. The pH plays a part in the skin's barrier formation and antimicrobial defence functions. The pH of a wound, which is usually alkaline in chronic/hard-to-heal wounds, influences multiple healing processes **[Table 19]**[218, 219] The pH fluctuates throughout the stages of wound healing,[351] but the alkaline status of the wound bed is generally maintained until re-epithelialisation, after which the pH returns to the usual slightly acidic state of the stratum corneum.[352]

Bacterial survival and proliferation can occur within the alkaline environment of a wound.[219, 220, 352] Bacteria can rapidly adapt to the pH of their surrounding environment and can also modify that environment (e.g., through their metabolic processes) to a pH favourable to their proliferation.[357] This ability to modify pH may impact the performance of antimicrobials.[218, 219]

Table 19: Impact on wound healing of acidic and alkaline wound environments[218-220, 353-356]	
Acidic pH	**Alkaline pH**
• Positive effect on proliferation of fibroblasts, DNA cell synthesis, tissue oxygenation, collagen formation, angiogenesis and macrophage activity[218-220, 353, 354] • Keratinocyte differentiation[220, 355] • More rapid wound healing[220]	• Immunological response triggered by increased bacterial growth[220, 354, 356] • Increased proteolytic activity via increased matrix metalloproteases (MMPs) and decreased tissue inhibitors of MMPs (TIMPs)[220, 354, 356] • Fibroblast inhibition[220,354, 356] • Decreases tissue oxygenation[220, 354, 356] • Slower or stalled wound healing[220]

Antiseptics differ in their pH, with some being acidic and others being alkaline.[155] Those with a slightly acidic pH create an environment more hostile to microbes,[352] and can be strategically selected to optimise the pH of the wound environment. However, there is currently little research on practical considerations, for example the duration of time the pH adjustment is maintained in the wound (e.g., how quickly microbes might re-modify the environment). Serial pH measurement may help to guide antiseptic selection and frequency of use (see Chapter 6).

Goals of care

Goals of care are established through a holistic assessment of the individual and their wound. Selection of an antiseptic should consider whether the goal is to prevent wound infection in high-risk individuals, address increasing microbial burden in wounds, or provide palliative management. Consideration should also be given to the individual's experience of pain or discomfort that can be associated with application of some antiseptics.[155]

Available formats

Antiseptics are prepared in a range of formats for direct application to a wound, including liquids, gels, pastes and antiseptic-containing wound dressings. Some formulations also contain surfactants, which can assist therapeutic cleansing by reducing surface tension and facilitating the loosening and dispersion of biological debris and components of the wound biofilm.[262] The clinical and antimicrobial properties of an antiseptic formulation may be influenced by its vehicle, concentration, release characteristics and contact time.

The regulatory classification of antiseptic products varies between jurisdictions and according to their intended purpose and principal mode of action. Depending on these factors, an antiseptic-containing product may be regulated as a medicine/drug, medical device or combination product. Regulatory classification also influences the claims manufacturers can make. For example, an antiseptic incorporated into a wound dressing may be authorised primarily to control microbial contamination within the dressing or provide an antimicrobial barrier, rather than to claim treatment of wound infection.[358, 359] Consequently, regulatory claims should not necessarily be interpreted as representing the full range of biological effects demonstrated in experimental studies.

Wound dressings containing antiseptics are available in a range of formats, including foams, gels, island dressings and film dressings. Depending on their design, the antiseptic may remain predominantly associated with the dressing, where it acts on microorganisms and their by-products taken up with wound exudate, or it may be released into the wound bed. Release may occur relatively rapidly or be sustained over several days.[360] When deciding on antimicrobial dressing selection, clinicians should consider the properties of the active ingredient (e.g., effectiveness against microbes, release into the wound, activity duration, potential for cytotoxicity, etc.) as well as the wound dressing's ability to manage moisture (e.g., exudate management through absorption or moisture donation, depending on the amount of exudate). **Appendix A** summarises antiseptic wound dressing options.

Topical antibiotics and antifungal agents

Topical antibiotics (e.g., bacitracin, neomycin, polymyxin B, mupirocin and fusidic acid) should be avoided in routine chronic wound management unless there is a specific clinical indication. Although topical antibiotics have historically been used for the prevention and treatment of wound infection, evidence supporting their routine use is limited, and inappropriate or prolonged exposure may contribute to antimicrobial resistance. Many are available over-the-counter, and this has encouraged their inappropriate use.[361] By preferentially inhibiting susceptible organisms, antibiotic exposure can exert selective pressure favouring the persistence or emergence of resistant organisms [see Chapter 2]. In addition, some topical antibiotics, particularly neomycin, fusidic acid and bacitracin, are recognised causes of allergic contact dermatitis.[362] This may be particularly relevant in individuals with chronic/hard-to-heal wounds or chronic venous stasis, eczema or a history of work in sectors with high antibiotic exposure, and therefore increased risk of sensitisation (e.g., healthcare, the pharmaceutical industry, and agriculture).[362]

An exception may be topical metronidazole when used specifically for management of malodour associated with malignant or fungating wounds. Clinical studies have demonstrated reductions in wound odour following application of topical metronidazole gel, with potential associated improvements in comfort and quality of life.[363] A thin layer of gel may be applied according to the prescribed regimen, with frequency guided by the formulation, wound characteristics, exudate volume and frequency of dressing change.

Research has indicated that in at-risk individuals fungi may contribute to delayed wound healing and the formation of polymicrobial biofilms more than previously acknowledged.[32, 223] Fungal cultures, stains and molecular diagnostic methods may assist in determining the need for systemic antifungal therapy for individuals with ongoing clinical signs and symptoms of wound infection and/or delayed wound healing despite appropriate care.[32] Consultation with an infectious diseases specialist should be considered in these presentations.

For individuals with covert signs and symptoms of wound infection in which fungi are shown to be present, selection of a topical antiseptic with demonstrated efficacy against *Candida* biofilm (e.g., hypochlorous acid) may be considered.[364] Additionally, fungal infections of the periwound skin should be identified and treated with antifungal creams, ointment, or solutions (e.g., azoles or allylamines).[365]

Systemic antibiotic use in wound infection

Wound infection is a significant contributor to delayed wound healing. As described in the IWII-WIC [see **Figure 5: IWII-WIC**], if wound infection is not successfully addressed in the early stages, its progression to spreading and systemic stages has the potential to be life threatening.[206, 366]

Recommendation 12

Commence systemic antibiotic therapy urgently when there are signs and symptoms of systemic or spreading wound infection. Perform a risk assessment and use clinical judgement to determine whether commencement of systemic antibiotic therapy is appropriate for treating signs and symptoms of overt local wound infection.

(Underpinning evidence: Level 1 evidence[366–368])

There is agreement across clinical guidelines and systematic reviews that systemic antibiotic therapy should be commenced urgently in the presence of signs and symptoms of spreading or systemic infection or sepsis.[366–369] In the presence of signs and symptoms of overt local wound infection, systemic antibiotics should be commenced judiciously, with consideration to the individual's history and risk factors, type and presentation of the wound and response to antiseptic treatment.

Table 20: Prudent use of antibiotic treatment to reduce resistance[1, 37, 59]

- Use antibiotics only when necessary
- Optimise pharmacokinetics/ pharmacodynamics
- Select an agent with a narrow spectrum
- Reserve broad-spectrum agents for more resistant bacteria
- Avoid long term use for prophylaxis (except in the case of hard-to-heal infections associated with an implanted device in a poor candidate for surgical removal)
- Develop and adhere to local policies and procedures (e.g., guidelines, formulary, restrictions, etc.) that are evidence-informed and regularly evaluated
- Monitor trends in microbial sensitivity
- Implement antibiotic cycling

While systemic antibiotics are highly efficacious in managing infection and are a necessary and life-saving intervention in many clinical situations, there is global concern regarding the rapid rise in antimicrobial resistance that must always be considered.[368] Prudent decision-making is required in determining when to commence systemic antibiotics, and in maintaining prescribing patterns that are consistent with antimicrobial stewardship **[Table 20]**.

Evidence shows that there is no specific antibiotic class that is more effective than another.[366, 368] The effectiveness of systemic antibiotic therapy is influenced by:

- Characteristics of the medication: consider the ability of the medication to penetrate the site of the infection, dosing of the antibiotic to best achieve concentrations that target the pathogen, spectrum of the drug (how targeted it is to the pathogen), route, duration of use and any concurrent treatment.
- Characteristics of the pathogen: consider the susceptibility of organisms to the selected drug and potential resistance or development of biofilm.
- Characteristics of the individual/host: consider immunocompromised status, metabolism and the specific infective environment (e.g., pH levels at the site of infection), site of the wound and drug allergies.

Selection of therapy should start with empiric treatment, then be adjusted based on culture and sensitivity results, the individual's response to empiric treatment[370, 371] and the World Health Organization advice on first and second-choice antibiotics that are recommended with consideration to antimicrobial resistance.[368]

Empiric treatment is started when the clinical diagnosis of infection is made and is based on clinician judgement regarding probable infecting organisms. This judgement should be informed by the individual's history, specifically whether this is a first-time infection with first-time antibiotic treatment versus recurrent infection with a history of multiple antibiotic courses[370] **[Table 21]**. Consideration should be given to the possible presence of anaerobic microbiota, which have been associated with poorer wound healing outcomes and are often involved in hard-to-heal or recurrent wounds.[372] Risk of resistant organisms should be assessed **[Table 22]**. As soon as culture and sensitivity results are available the antibiotic regimen should be reviewed based on sensitivity and pathogenicity of microbes, the individual's response to empiric treatment and the wound status. As a spreading infection worsens, oral therapy may not be sufficient and hospitalisation and parenteral antibiotics may be required. Consultation with an infectious diseases specialist for worsening/spreading infections is recommended.

Monitoring and adjusting antimicrobial treatments

Good AMS requires ongoing monitoring of the individual and their wound to evaluate treatment efficacy and reduce use when appropriate. Antimicrobial treatments are not encouraged for longer-term use unless absolutely necessary, based on the individual's response to therapy. It is also important to promptly identify wounds in which signs and symptoms of infection are progressing to enable rapid escalation of treatment.[233] **Figure 12** provides an overview of monitoring and evaluation of antimicrobial treatment.

Table 21: Oral antibiotic coverage for local wound infection to early spreading infections*,ß [366, 368, 369, 371, 373-375]

	Organisms	Antibiotic choices
First time infections	Gram-positive • Methicillin-susceptible *Staphylococcus aureus (MSSA)* • *Streptococcus species*	Beta-lactams • Cephalexin • Amoxicillin/clavulanate • Dicloxacillin (in the absence of above options) For those with beta-lactam allergy • Doxycycline (may not reliably cover *Strep*) • Clindamycin • Trimethoprim-sulfamethoxazole (may not reliably cover *Strep*)
	Gram-positive • Methicillin-resistant *Staphylococcus aureus (MRSA)* • High risk for MRSA	• Doxycycline • Trimethoprim-sulfamethoxazole • Linezolid • Tedizolid
Recurrent infections/ previous antibiotics	Gram-positive (as above)	
	Gram-negative • *Pseudomonas aeruginosa* • *Escherichia coli* • *Proteus mirabilis* • *Acinetobacter species* • *Klebsiella pneumoniae, etc.*	Treatment is variable; culture-directed treatment is essential and may include: • Amoxicillin/clavulanate • Levofloxacin, moxifloxacin • Trimethoprim-sulfamethoxazole
	Anaerobes#	• Amoxicillin/clavulanate • Clindamycin • Metronidazole (usually in combination with antibiotic to cover aerobes if present)

* Table does not apply to surgical site infection (SSI) because antibiotic selection for SSI is based on type of surgery, which influences microbial exposure.
ß As a spreading infection worsens, oral therapy may not be sufficient and hospitalisation and parenteral antibiotics may be required. Consultation with an infectious diseases specialist for worsening/spreading infections is recommended.
Usually found in deep wounds and sinus tracts, check with laboratory regarding need for special collection methods

Table 22: Risk factors for MRSA infection [376-378]

Healthcare-associated risk factors	Community-associated risk factors	Host-associated risk factors
• Previous MRSA exposure or infection within 12 months • Prior hospitalisation within 12 months • Prior antibiotic use within 90 days • Chronic kidney disease and/or hemodialysis • Presence of invasive devices • Prolonged hospitalisation with exposure to broad-spectrum antibiotics • Colonisation with MRSA at time of admission	• Crowding (e.g., homeless shelters, military barracks, prisons, gyms, day-care centres) • Frequent skin-to-skin contact (e.g., contact sports, household contacts) • Compromised skin (e.g., wounds, rashes) • Contaminated surfaces (e.g., shared towels or equipment, public transportation)	• HIV/AIDS • Morbid obesity • Previous skin infection • MRSA colonisation • Diabetes with obesity • Intravenous drug use • Poor general hygiene

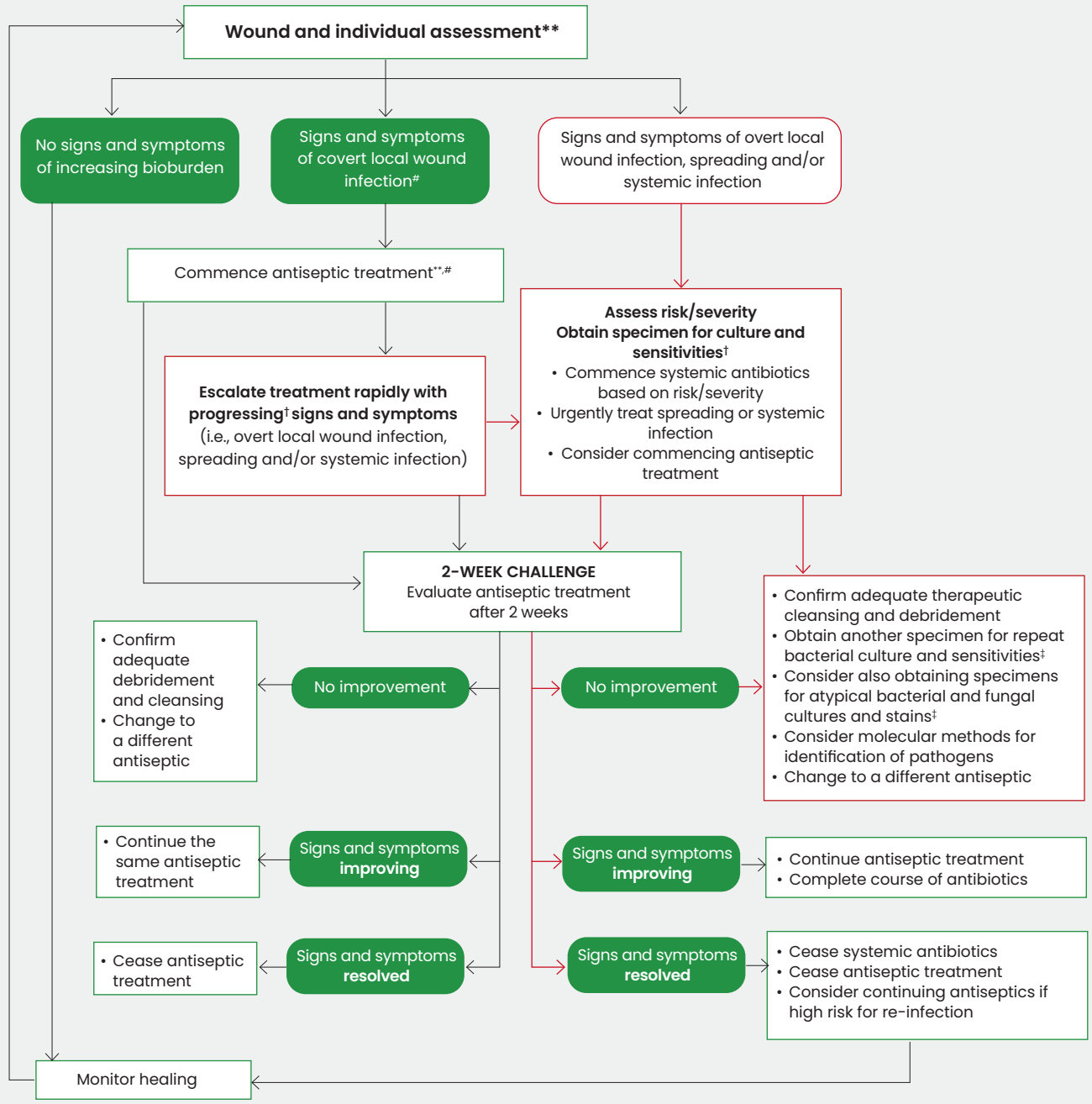
Oral antibiotics are usually prescribed for 5–10 days.[379] The course should be as short as clinically appropriate. However, the individual and the wound should be closely monitored and re-evaluated in the absence of a rapid response. Antimicrobial susceptibilities should be reassessed, atypical pathogens considered and adjustments may be made to therapeutic wound cleansing and debridement regimens, or the selection of antiseptics.[233] It should be noted that in the case of diagnosed osteomyelitis, antibiotic therapy has traditionally been administered intravenously for up to 6–8 weeks. In many cases, this may not be necessary, and an appropriately selected oral antibiotic could be used following only brief (or even no) parenteral therapy and given for no more than 6 weeks.[380-382]

Duration of antiseptic use in wounds exhibiting signs and symptoms of covert local wound infection should be individualised and based on regular wound assessment.[233] Expert consensus suggests that a two-week challenge is an appropriate guide for evaluating the efficacy of the selected antiseptic in treating wound infection. This allows sufficient time for the agent to exert observable activity to inform an evaluation of the management plan.[74, 141, 383] After two weeks of treatment, a decision can generally be made regarding the efficacy of the management strategy, although up to four weeks of antiseptic treatment may be required to attain results.[233] As noted in **Figure 12**, antiseptics should be ceased in the absence of signs and symptoms of wound infection, unless the individual is at high risk of re-infection.[115] In wounds that show no signs of improvement, a re-evaluation of the management strategy is required, and changing to an alternate antiseptic is suggested.[141, 383, 384]

Microbial-binding dressings with an antimicrobial effect

Dialkylcarbamoyl chloride (DACC)-coated dressings are non-medicated wound dressings with a physical microbial-binding effect. DACC is a hydrophobic fatty acid derivative applied to dressing fibres. DACC-coated dressings physically bind microorganisms through hydrophobic interactions. Bacteria and fungi with hydrophobic cell surfaces, as well as endotoxins containing the hydrophobic lipid A component, adhere to the dressing,[385] and are physically removed with the wound dressing. Experimental work has demonstrated physical adhesion of *S. aureus* to DACC-coated material, supporting this physical mode of action.[386, 387]

Several observational studies and pilot RCTs have demonstrated improved wound healing[388-390] and control of microbial burden[388, 391-393] with DACC-coated dressings, particularly in chronic/hard-to-heal wounds. However, there are few high-quality controlled trials and the extent to which microbes are cleared from a wound requires more research.[392] DACC technology does not rely on chemical antimicrobial activity; therefore, there is low/no risk of AMR. More research is required on the effectiveness of DACC-coated dressings in managing wound infection; however, a recent consensus document underpinned by a systematic review[8] suggested DACC-coated dressings could be used as an adjunct to comprehensive wound management that includes therapeutic cleansing, debridement and exudate management.



** The IWII's *Wound Infection in Clinical Practice* (2026) provides comprehensive guidance on holistic assessment of the wound and the individual, diagnostic techniques for wound infection, topical and systemic antimicrobial treatment strategies, including guidance in different clinical contexts.

The IWII's Wound Infection Continuum presents signs and symptoms of wound infection and broad guidance on commencing treatment.

† Progressing signs and symptoms require urgent review and commencement of systemic antibiotic treatment.

‡ The IWII's *Wound Infection in Clinical Practice* (2026) provides guidance on specimen collection techniques. Check with your local laboratory regarding appropriate collection and transfer of specimens for atypical bacteria and fungi to ensure survival of microbes.

Figure 12. Decision tree for monitoring and adjusting systemic antibiotic and antiseptic regimens

Chapter 11
Biophysical modalities

2026 updates and key points

- Biophysical modalities are increasingly being explored as complementary approaches to wound infection management as a response to the rise in antimicrobial resistance.
- Negative pressure wound therapy shows clinical benefit for treating wound infection in acute and chronic/hard-to-heal wounds. Its use is incorporated into standard wound care pathways in many clinical contexts.

Introduction

Biophysical modalities are increasingly being explored as complementary approaches to conventional wound infection management, particularly in response to growing concerns regarding antimicrobial resistance (AMR) and the limitations of systemic antibiotic therapy.

Many therapeutic biophysical modalities have been used to support wound healing and tissue repair; however, robust evidence demonstrating their effectiveness specifically for wound infection outcomes remains limited for most modalities. Negative pressure wound therapy (NPWT) represents a notable exception, with substantial evidence supporting its role in promoting faster healing, reducing bacterial burden and supporting infection control in selected wounds.

Therapeutic biophysical modalities

Biophysical modalities are non-pharmacological therapies that utilise the effects of physical forces or forms of energy to support wound healing and manage wound infection. These therapies act by modifying the wound environment, reducing microbial burden and stimulating tissue repair processes and local perfusion, without relying on systemic or local antimicrobial agents. Biophysical modalities represent an emerging group of adjunctive therapies that can complement conventional wound infection management by targeting both microbial burden and impaired healing processes through non-pharmacological mechanisms. An overview of these modalities and the evidence base for their use is provided in **Table 23**.

Negative pressure wound therapy

NPWT is a widely established non-pharmacological modality that applies controlled sub-atmospheric pressure to the wound environment through a sealed dressing system. NPWT operates through both mechanical and physiological mechanisms. At the macroscopic level, negative pressure induces macrodeformation, which contracts the wound edges, reduces wound size, and eliminates dead space where microorganisms can proliferate.[394] At the microscopic level, microdeformation at the wound–dressing interface stimulates lymphatic drainage, cellular proliferation and angiogenesis, while the mechanical forces also improve tissue perfusion, which is critical for immune function and the eradication of aerobic microorganisms.[395] Enhanced wound bed perfusion ensures better delivery of nutrients and systemic antimicrobial treatment to the wound bed.[396] Most importantly, NPWT facilitates continuous removal from the wound of exudate containing microorganisms, inflammatory mediators (e.g., interleukins) and proteolytic enzymes.[397, 398] The sealed dressing system also creates a controlled environment that minimises external contamination, reducing the risk of secondary infection.[399] This containment is particularly valuable in hospital settings, where cross-contamination poses a significant risk.

NPWT with instillation (NPWTi) is an enhanced form of NPWT. This technique combines negative pressure with periodic instillation of topical solutions such as saline or antiseptics. NPWTi enhances wound cleansing, disrupts biofilms, and improves bacterial clearance compared to standard NPWT alone.[400, 401] With respect to wound infection outcomes, a systematic review of 13 studies showed that wounds with various aetiologies treated with NPWTi were 4.4 times more likely to achieve a reduction in bacterial counts than those receiving standard care.[400] A systematic review of seven studies showed surgical site infection (SSI) rates were lower (5.6% versus 10%) for wounds treated with NPWTi.[401] Meta-analyses and clinical studies in diabetes-related foot ulcers,[402] surgical and traumatic wounds[403-406] have consistently demonstrated the benefits of NPWT for reducing infection rates, shortening healing time, and decreasing the need for repeated debridement.

Table 23: Overview of therapeutic biophysical modalities

Intervention	Mechanism of action	Clinical considerations
Negative pressure wound therapy (NPWT)	Sub-atmospheric pressure leads to: • Wound contraction • Dead space elimination • Stimulation of lymphatic drainage • Cell proliferation • Continuous removal of exudate	• **Strong clinical evidence** for improved healing in diabetes-related foot ulcers and surgical/traumatic wounds • NPWTi adds periodic instillation of antiseptics to disrupt biofilm, cleanse the wound and improves biofilm clearance • Reduced need for repeated debridement • Sealed system reduces external contamination • Species-dependent effects on bacterial counts • Can potentially cause Gram-positive species overgrowth • Device cost • Need for trained use • Possible pain or tissue damage if misapplied
Bioelectric (electroceutical) dressings	• Ag/Zn microcells activated by exudate create continuous microcurrent and electrochemical redox gradients that disrupt cell membranes, inhibit quorum sensing and promote regenerative signalling	• **Limited clinical evidence** • Broad-spectrum effect • Multi-targeted action lowers resistance risk • Potential for silver/zinc-related local toxicity or sensitivity • Variable product formulations
Photo-biomodulation (LEDs/lasers)	• Light absorbed by chromophores leads to a pathway that modulates signalling for repair • Specific wavelengths (e.g., blue 400–470 nm) produce reactive oxygen species that directly disrupt microbes and biofilms.	• **Limited clinical evidence** on infection-specific endpoints • Adjunct for difficult wounds • Heterogeneity in regimens leads to inconsistent results in clinical studies • Non invasive • Wavelength- and dose-dependent effects • Antimicrobial effects (especially blue light) • Regenerative effects • Risk of tissue heating with improper use
Cold plasma (NTAPP)	• Partially ionised gas produces RONS, leading to oxidative damage to cell membranes and DNA, disruption in biofilms, and favourable effects on host cells, metabolism and wound healing	• **Emerging clinical evidence** • Strong *in vitro* evidence for reduced bioburden • Heterogeneous devices and regimens • Rapid, broad-spectrum antimicrobial activity including multidrug-resistant organisms • Effective against biofilms • Minimal systemic toxicity • Device cost and availability
Therapeutic ultrasound (low frequency ultrasound debridement)	• Contact mode: cavitation and acoustic streaming lead to mechanical disruption of devitalised tissue and biofilm, bacterial detachment, enhanced debris removal • Non contact mode: acoustic streaming via mist leads to immunomodulation, reduced inflammatory cytokines, angiogenesis	• **Limited clinical evidence** • Used as adjunct to debridement with antimicrobial therapy • Variable efficacy by device and regimen • Bacterial load reduction • Biofilm disruption • Potentiates antibiotics • Promotes wound bed preparation • Useful for resistant biofilms • Requires multiple treatments and trained operators • Enhances antibiotic penetration into biofilm

While NPWT is highly effective, its impact on bacterial counts is variable and may be species-dependent.[407] While NPWT may suppress the proliferation of certain Gram-negative bacilli, particularly non-fermentative strains such as Pseudomonas species, the changed wound microbiome can promote growth of Gram-positive cocci instead.[407] This may explain the variance in effectiveness in reducing SSI observed across different surgery types in a review of eight studies.[408] As with all treatments, selection of NPWT should be individualised and with consideration of the type of surgery or wound, the wound characteristics, the individual's clinical context and care goals, and the response to other infection treatment interventions.[408]

Bioelectrical dressings

Bioelectric dressings are electroceutical therapies designed to modulate the wound microenvironment through low-level electrical stimulation.[409] These dressings incorporate a matrix of silver (Ag) and zinc (Zn) microcell batteries that are activated when they encounter wound exudate.[410, 411] When activated, a continuous microcurrent is generated across the wound surface, providing antimicrobial properties and supporting tissue repair.[412] The therapeutic effects arise from electrochemical reactions that establish localised electrical fields and redox gradients. These processes interfere with bacterial physiology by disrupting cell membranes, altering transmembrane potentials, and impairing electron transport systems essential for microbial metabolism.[413] They also inhibit quorum sensing, reducing the ability of bacteria to communicate, thereby limiting biofilm formation.[414] These multi-targeted mechanisms may reduce the likelihood of resistance development. However, antimicrobial effectiveness has primarily been demonstrated *ex vivo*,[410, 415] with only a small number of studies demonstrating clinical efficacy in burns[416] and chronic wounds.[417-419]

Photobiomodulation therapy

Photobiomodulation therapy (PBM) utilises non-ionising light sources including light-emitting diodes (LEDs) and lasers to eradicate bacteria. The biological effects of PBM are mediated through the absorption of light by endogenous chromophores. This initiates photochemical reactions that lead to increased production of adenosine triphosphate (ATP), reactive oxygen species (ROS), and nitric oxide (NO), which subsequently modulate the intracellular signalling pathways and cellular function of microorganisms.[420] These effects are highly wavelength-dependent (range of 400–1100 nm).[421] PBM has demonstrated broad-spectrum activity against bacteria and fungi, as well as disruption of biofilm formation.[420] Despite strong preclinical evidence supporting both antimicrobial and regenerative effects, clinical translation demonstrating reduction or eradication of wound infection remains limited as most studies have focused on wound healing outcomes.[422]

Cold plasma therapy

Cold plasma therapy, also known as non-thermal atmospheric pressure plasma (NTAPP), is an emerging modality that utilises partially ionised gas to produce reactive oxygen and nitrogen species (RONS), which are mediators for cellular signalling.[423] Cold plasma devices typically generate a mix of RONS, charged particles, and ultraviolet photons, which contribute to antimicrobial and regenerative effects.[424] The primary mechanism involves the interaction of these reactive species with microbial cells, leading to disruption of cell membranes, ultimately resulting in microbial inactivation.[425] Plasma also damages bacterial DNA through RONS that alter genetic material and promote DNA-protein crosslinks, hindering repair and replication of microorganisms.[426] Therefore, cold plasma exerts its effects through multi-target oxidative pathways, reducing the likelihood of antimicrobial resistance development. Additionally, cold plasma can disrupt the extracellular polymeric substance matrix of biofilms, enhancing susceptibility to host immune responses and adjunctive therapies.[425] Cold plasma has demonstrated broad-spectrum antimicrobial activity; however, most of the research is limited to *in vitro* studies.[427] Clinical evidence demonstrating reductions in microbial load remains limited.[427]

Therapeutic ultrasound

Ultrasound debridement employs low-frequency ultrasound waves, typically in the range of 20–60 kHz, to facilitate wound cleansing and healing.[428] Unlike conventional debridement methods, ultrasound offers both mechanical and biological benefits, making it particularly valuable in the management of chronic wounds complicated by infection. The therapy can be delivered through contact devices, which transmit acoustic energy to the wound bed, or non-contact devices, which transmit energy through a mist. Each modality operates via distinct mechanisms and produces different clinical outcomes.

The fundamental principle of ultrasonic debridement using low-frequency ultrasound lies in the generation of cavitation and acoustic streaming. Cavitation refers to the formation and oscillation of microbubbles within the wound. These bubbles collapse and release shear forces that mechanically disrupt devitalised tissue and biofilm structures, exposing bacteria to antimicrobial agents and immune cells, thereby reducing microbial persistence.[429] Acoustic streaming complements this process by enhancing fluid movement at the wound interface, facilitating debris removal and preparing the wound bed for healing.

Evidence highlights that ultrasonic debridement exerts direct antimicrobial effects when delivered via contact devices. Studies have demonstrated reductions in bacterial load, biofilms[430, 431] and visible slough[432] in chronic/hard-to-heal wounds. Ultrasonic debridement has been shown to enhance antibiotic penetration into biofilms, increasing bacterial susceptibility to treatment.[433] This synergistic effect is relevant in the era of rising AMR, as ultrasound can potentiate the action of antibiotics against pathogens. Experimental and *in vitro* studies further reinforce these findings.[434, 435]

In contrast, non-contact devices primarily exert indirect immunomodulatory effects, reducing inflammatory cytokines and modulating the wound environment rather than directly lowering bacterial burden.[436] However, there is a lack of clinical evidence exploring its use in reducing wound infection.[437]

Chapter 12
Wound infection in resource-limited settings

2026 updates and key points

- In settings with limited resources, focus on preventing wound infection.
- Advocate for the availability of affordable but effective wound dressings and devices through the national government's essential drug and dressings lists.
- Provide education and mentorship to local traditional practitioners to ensure they practice safe wound treatment.
- Engage local traditional practitioners in prevention, first aid, and wound management systems and governance, and focus on integration of local cultures and customs.
- Provide skin hygiene and other wound prevention education to primary carers.
- Research is still required on the efficacy of plant-derived therapies.

Introduction

The World Health Organization (WHO) endorses the World Bank definition for low, low to middle, upper-middle- and high-income countries, which are classified according to their annual gross national income per capita. In 2025 that equated to:[438]

- Low-income (in US dollars) economies: $1,145 or less.
- Lower-middle-income economies: $1,146 to $4,515.
- Upper-middle-income economies: $4,516 to $14,005.
- High-income economies: More than $14,005.

Globally, 1.58 billion people (19% of the world's population) live across 54 low-income countries and 762 million people live in extreme poverty on under $3 USD per day. Eighteen of the world's twenty poorest countries are in Africa.[438] Many of those affected by desperate poverty confront ethnic and sectarian strife, war, natural disasters, pandemics and a dearth of health services.

Although resource-limited settings are often associated with developing countries, pockets of resource limitation exist worldwide. Resource-limited settings can also occur in high-income countries due to population inequities or barriers to healthcare access, including severe financial constraints, homelessness and geographic isolation. It is estimated that over 70% of these areas are rural or remote settings.[439] A resource-limited setting in healthcare may be characterised by constraints in one or more of the following:

- Financial resources,
- Trained health care personnel,
- Patient to nurse ratios,
- Hospital capacities and crowding,
- Equipment or supplies,
- Medications,
- Transportation,
- Patient and family education and support, and
- Reliable electricity or water systems.

What is the burden of wounds in resource-limited settings?

Resource-limited settings bear a disproportionately high burden of wounds and wound-related morbidity. Burns and traumatic injuries are important causes of acute wounds, with burns frequently associated with domestic cooking practices and disproportionately affecting children.[440, 441] The WHO estimates that approximately 180,000 deaths occur annually due to burns, with the majority occurring in low- and middle-income countries.[442] Neglected tropical diseases, including lymphatic filariasis, [443, 444] Buruli ulcer,[445] Hansen's disease[446] and podoconiosis,[447] may also cause chronic skin breakdown, ulceration and disability.

Diabetes-related foot disease represents a growing global challenge. More than 830 million people worldwide are living with diabetes,[448] and the lifetime risk of developing a diabetes-related foot ulcer has been estimated to be as high as 34%.[449] Outcomes are particularly concerning in resource-limited settings, where access to preventive care, multidisciplinary wound services, diagnostics, antimicrobial therapy and surgical intervention may be constrained, contributing to higher risks of infection and amputation.[449] Antimicrobial resistance, including multidrug-resistant organisms and emerging pathogens, presents an additional challenge to wound management.[450, 451]

Recognising these intersecting challenges, the WHO has strengthened its focus on skin-related

neglected tropical diseases through the Skin Neglected Tropical Diseases framework and associated initiatives addressing skin health, wounds and rehabilitation.[452] These initiatives can promote greater international attention, coordinated research, workforce development and resource allocation for populations experiencing a high burden of wounds and skin disease.

What is best practice in resource-limited settings?

Guideline documents are often developed with well-resourced settings foremost in mind. Dissemination and implementation of clinical recommendations can be challenging in resource-limited settings due to the barriers outlined. Lack of training may result in inadequate or contradictory knowledge of evidence-based practice and an environment that does not support the implementation or sustainability of best practice recommendations. In resource-limited settings, implementation of guideline evidence will require adaptation to account for the accessibility of resources, local cultural norms and availability of ongoing support to maintain best practice delivery. Culturally sensitive practice enablers and ongoing mentorship are initiatives that can facilitate sustainability.[453]

What are the wound infection challenges in resource-limited settings?

As highlighted throughout this document, identification and diagnosis of wound infection are primarily clinical judgements informed by comprehensive, holistic assessment. In resource-limited settings, additional challenges may influence both assessment and management, including:

- Access to clean, safe water for cleansing the wound and surrounding skin. Where safe water is unavailable, locally feasible methods of water disinfection may be required. **Table 24** provides guidance on disinfection using household bleach, which is generally inexpensive and widely available.[454]
- Implementing appropriate aseptic technique, including access to methods for decontaminating or sterilising reusable instruments when single-use equipment is unavailable.
- Maintenance of skin hygiene and care to preserve skin barrier function.
- Safe adaptation and use of locally available materials for wound care.[455, 456]
- Consideration of traditional wound-healing practices, including evaluation of available evidence regarding their safety and effectiveness and adaptation of practices where appropriate.[456, 457]
- Active involvement of individuals with, or at risk of, wounds and their families, carers and other support networks in prevention, assessment and ongoing wound care.

Table 24: Procedure for disinfecting water[454]

Volume of water	Amount of 6% bleach to add#	Amount of 8.25% bleach to add
1 quart (1 litre)	2 drops	2 drops
1 gallon (3.8 litres)	8 drops	6 drops
2 gallons (7.6 litres)	16 drops (1/4 tsp or 1.25 ml)	12 drops (1/8 tsp or 0.62 ml)
4 gallons (15.1 litres)	1/3 tsp (1.7 ml)	¼ tsp (1.25 ml)
8 gallons (30.2 litres)	2/3 tsp (3.4 ml)	½ tsp (2.5 ml)

Procedure

1. Use a clean container.
2. If cloudy, allow to settle and filter through a clean cloth or paper towel.
3. Add bleach based on the following table using a clean dropper.
4. Stir and allow to stand for 30 minutes.
5. Should have a faint bleach odour. If not, repeat once.

#Note: Bleach comes in two strengths 6% and 8.25%. Check the strength carefully.

Plant-derived therapies

Plant-derived compounds [Table 25] have long been used in traditional medicine for their antimicrobial properties and have gained renewed attention as potential adjuncts in modern wound infection management. Plant-derived therapies are primarily used in resource-limited settings and traditional or Indigenous healing practices, where they remain culturally important.[458] Plant-derived compounds might target multiple pathways involved in microbial survival and persistence including:[459-466]

- Disrupting the cell membranes within microbes, thereby increasing their permeability,
- Inhibiting enzyme/protein activity within microbes,
- Interfering with quorum sensing, thereby limiting the ability of microbes to form biofilms, and
- Generating oxidative stress.

While some of these mechanisms have been demonstrated in bench science and animal wound models, the current evidence base is predominantly preclinical for most plant-derived therapies. Research on the clinical effectiveness, standardisation, safety and adjunctive potential of plant-derived compounds in managing biofilm-associated infections and antimicrobial-resistant strains is required.

As part of holistic management, ask the individual and their carer about their cultural norms and use of traditional (or home-based) remedies. Include the individual's support network and cultural leader, discuss the evidence, and undertake a risk-based assessment of cultural practices.

Table 25: Overview of claimed evidence for plant-derived compounds for wound infection

Plant/ compound (Bioactive components)	Reported antimicrobial mechanisms	Reported impact
Turmeric (Curcumin)	• Disrupts bacterial cell membranes, causing leakage of intracellular contents[459] • Inhibits microbial survival[459] • Anti-inflammatory effect through suppression of pro-inflammatory cytokines[459]	• Reduced malodour, exudate and pain in human case studies[459] • Reduced erythema in rat models[467]
Cinnamon (Cinnamaldehyde, eugenol)	• Disruption to microbial metabolism[460] • Inhibits quorum sensing and biofilm formation[460] • Reduces microbial colonisation[460]	Not reported
Garlic (Allicin)	• Inhibits essential bacterial enzymes[461] • Interferes with quorum sensing and biofilm formation[462]	• Decreased bacterial count in animal models[461]
Neem (Azadirachtin, nimbidin, flavonoids, limonoids)	• Ruptures bacterial cell walls, leaking contents[463] • Anti-inflammatory effect through suppression of pro-inflammatory cytokines[463]	Not reported
Oregano oil (Carvacrol, thymol)	• Increases membrane permeability[464]	Not reported
Papaya (paw paw) (Papain)	• Commercial papain extract-based agents are banned by the (USA) Food and Drug Administration due to anaphylaxis risk[468] • Potential antimicrobial effect[468] • Enzymatic debridement[468]	• Reduction of wound bed slough demonstrated in RCTs[468]
Tea tree oil (Terpinen-4-ol and other terpenes)	• Eradicates bacteria[466] • Anti-inflammatory activity[466]	• Eradication of MRSA shown in an RCT[466]
Thyme (Thymol, carvacrol)	• Bacteriostatic properties[465] • Biofilm inhibition properties[465]	Not reported

Chapter 13
Wound infection in neonates, infants and children

2026 updates and key points

- Neonates, infants and children are particularly vulnerable to wound infection due to their immature skin, developing immune systems and physiological vulnerability. Premature neonates are at significantly increased risk of wound infection because of the combined effects of immature skin barrier function, underdeveloped innate and adaptive immunity, frequent exposure to invasive procedures, and prolonged hospitalisation.
- Risk stratification for wound infection should be based on weight and age-related assessment of the above factors, as well as knowledge of the most common wounds seen in paediatric age groups.
- Monitoring for age-related signs and symptoms of wound infection facilitates early identification of wound infection and prompt treatment escalation.
- Effective paediatric wound care requires careful adaptation of therapeutic cleansing and debridement techniques, selection of local antiseptic treatments and wound dressings, diligent infection prevention practices and antimicrobial stewardship.
- Optimised wound care for neonates, infants and children requires meticulous infection prevention, a family-centred approach, knowledge of age-related factors that influence infection and its management, pain management, psychological support and family engagement.

Introduction

Paediatric wound infection should not be conceptualised as adult wound infection occurring in a smaller body. Developmental differences in skin structure, immune function, microbiome maturation, communication abilities and dependence on caregivers influence wound infection risk, clinical presentation and treatment throughout childhood. Premature neonates, neonates, infants, children and adolescents have unique age-related characteristics that need to be addressed in the context of wound infection. It is important to recognise that neonates and infants (up to 1 year of age) have particular vulnerability to skin injury and wound infection due to their:[469-473]

- Physiologically immature skin,
- Developing immune system, and
- Evolving microbiome interactions.

Premature infants are particularly susceptible because the skin barrier and immune functions are less developed at birth.[470] The rapid environmental change after birth further influences skin barrier maturation, fluid regulation and microbial colonisation.[469, 470] Both the skin barrier and immune system are immature in very early life stages. Thin epidermis and stratum corneum are responsible for reduced mechanical protection. Transepidermal water loss (TEWL) is increased and the susceptibility to skin breakdown/injuries and microbial invasion is greater. Additionally, the intrinsic immunological immaturity of premature infants significantly increases their susceptibility to wound infection. Reduced neutrophil function and impaired chemotaxis, decreased complement activity, impaired production of antimicrobial peptides, reduced adaptive immune responses and a limited immunological memory all compromise the host's ability to appropriately respond to pathogens, contributing to an increased risk of wound infection.[474] As survival of infants born at less than 24 weeks' gestation increases, there is growing recognition of the need for specialised skin care strategies to prevent iatrogenic injury and infection in neonatal care settings.[469, 472]

An association has been established between early-life skin injury, infection and antimicrobial exposure and later-life immune-mediated or atopic conditions, although causality, mechanisms and wound-specific pathways are still uncertain. For example, it is asserted that skin infections within a child's first two years can predispose to psoriasis, asthma and allergies.[471, 475, 476] This highlights the significance for the individual child of reducing wound infection risk.

These innate characteristics mean that effective paediatric wound infection management requires adapted approaches to prevention, local wound care, antimicrobial stewardship (AMS), pain management and psychosocial support that address the child's context. These should be delivered in a manner appropriate to the individual's age, development (including skin maturity and immune status), and family/carer context.

Age-related risk stratification approach to paediatric wound infection

Recommendation 13

Use an age-related approach to identify and mitigate risk for wound infection, to identify and assess wound infection and to select appropriate wound infection treatments in neonates, infants and children.

(Underpinning evidence Level 1 evidence,[477] Level 3 evidence,[478] Level 4 evidence[470, 479-483] and Level 5 evidence[469, 472, 476, 484-487])

An age-related risk stratification approach to assessing and mitigating wound infection risk, early identification and assessment of wound infection and selecting appropriate treatments is presented in **Table 26**. The conceptual approach is based on an original synthesis of:

- a systematic review on paediatric skin care practices to prevent infection,[477]
- a study reporting prevalence of injuries/wounds in paediatric age groups,[478] and
- reviews of descriptive studies[470, 479-483] and expert opinion[469, 472, 476, 484-487] on neonatal and paediatric skin barrier characteristics, the microbiome, wound infection signs, antiseptic safety and pain.

In paediatric care, using an age-related stratification approach to assess wound infection risk, identify signs of wound infection and plan appropriate prevention and treatment is important because risks, clinical presentation and safety of wound treatments vary substantially across age/developmental stages.[469, 470, 480, 481] A stratified approach supports earlier recognition of infection, safer selection of wound infection management strategies (e.g., cleansing solutions and antiseptics) and appropriate escalation.[470]

An age-stratified approach considers factors relating to:

- The individual child (e.g., age, gestational maturity, communication ability, pain expression, caregiver dependence),
- Maturity of physiological infection defences (e.g., skin barrier integrity, immune status, overall clinical condition, comorbidities), and
- The wound (wound aetiology, exposure to medical and other devices).

Age-related wound infection risk in neonates, infants and children

The impact of skin development and characteristics on risk of infection

Neonatal skin differs from adult skin in both structure and function. In preterm infants, the stratum corneum is incompletely developed; extremely premature infants may have only a few layers of corneocytes.[470, 472, 480] This results in increased permeability to water, irritants and microorganisms.[469, 470, 472, 480, 484] Consequently, neonatal skin contributes substantially to insensible water loss, thermoregulatory instability and infection risk. In extremely preterm neonates, even minimal epidermal disruption may increase transepidermal water loss (TEWL), expose immature tissue to irritants and microorganisms, and create a portal of entry for invasive infection.[472, 484] The dermal–epidermal junction is structurally weak in premature infants, increasing susceptibility to mechanical trauma from pressure, shear and friction.[477, 484]

Table 26: Age-related risk stratification approach to wound infection[469, 470, 472, 476-487]

RISK FACTORS			RISK LEVEL	INFECTION ASSESSMENT	INTERVENTIONS
Age group	**Skin, immune and developmental considerations**	**Common wounds/ risk contexts**	**Infection risk profile**	**Subtle or age-specific warning signs of wound infection**	**Key priorities in preventing and treating wound infection**
Extremely preterm neonates (22–24 weeks' gestation)	• Markedly immature epidermal barrier • Very thin/incomplete stratum corneum • Increased TEWL • Impaired thermoregulation • Developing innate immune responses • High vulnerability to chemical and mechanical injury	• Medical adhesive-related skin injury (MARSI) • Device-related injuries or pressure injuries (DRPI) • Extravasation injuries • Surgical wounds • Umbilical and line-associated skin breakdown	Very high risk. Minor skin disruption may rapidly become clinically significant due to: • Barrier immaturity • High permeability • Limited immune reserve • Frequent exposure to invasive devices	• Temperature instability • Apnoea or bradycardia • Increased oxygen requirement • Feeding intolerance • Lethargy • Irritability • Skin fragility or tissue breakdown around devices • Subtle local wound erythema or colour change, increased exudate, delayed epithelialisation	• Prevent iatrogenic injury • Minimise adhesive use • Use atraumatic dressings and silicone-based products where appropriate • Maintain humidity and thermal stability • Use antiseptics with extreme caution • Monitor closely for systemic deterioration • Involve neonatal specialists early
Preterm and term neonates	• Skin barrier still maturing • Skin pH and microbiome evolving • Immune responses remain immature • High body surface area-to-weight ratio increases risk of systemic absorption from topical agents	• Umbilical stump complications • Surgical wounds • Device-related injuries or DRPI • Extravasation • Traumatic birth-related wounds • MARSI • Pressure injuries in NICU settings	High risk, especially when exposed to: • Invasive devices • Prolonged hospitalisation • Broad-spectrum antibiotics • Compromised skin integrity	• Temperature instability • Poor feeding • Lethargy • Irritability • Local wound warmth, swelling, malodour, increasing exudate, wound edge deterioration and periwound colour change rather than obvious redness	• Support skin barrier maturation • Perform structured skin assessment • Minimise adhesive use • Use atraumatic silicone-based products where appropriate • Avoid unnecessary antiseptics • Use gentle cleansing • Protect periwound skin • Consider taking samples for culture and sensitivities when infection is progressing, systemic or not responding to treatment
Infants < 1 year	• Skin remains relatively thin and sensitive • Microbiome and immune system continue to develop • Limited verbal expression of pain • High dependency on carers for wound observation and care	• Burns and scalds • Surgical wounds • Ostomy-related wounds • Trauma • Diaper-associated dermatitis • MARSI	Moderate to high risk depending on: • Comorbidities • Nutrition • Device exposure • Wound aetiology • Care setting	• Crying during handling • Sleep disturbance • Reduced feeding • Increased clinginess • Irritability • Reduced movement of affected area • New or increasing wound pain • Delayed healing, odour, increasing exudate	• Avoid routine antimicrobial prophylaxis • Engage caregivers as diagnostic partners • Use age-appropriate pain assessment techniques • Minimise adhesive use • Use atraumatic silicone-based products • Select atraumatic dressings • Review antiseptic use frequently • Educate family on red flags and when and how to seek urgent help • Antimicrobial safety considerations include age, weight, wound size and body surface area

Table 26: Age-related risk stratification approach to wound infection[469, 470, 472, 476-487] (continued)

RISK FACTORS → RISK LEVEL → INFECTION ASSESSMENT → INTERVENTIONS

Age group	Skin, immune and developmental considerations	Common wounds/ risk contexts	Infection risk profile	Subtle or age-specific warning signs of wound infection	Key priorities in preventing and treating wound infection
Toddlers and preschool children	• Developing immune competence • Active mobility increases trauma risk • Limited ability to describe symptoms accurately • Fear and distress may interfere with assessment and dressing changes	• Traumatic wounds • Burns • Surgical wounds • Bites • Skin tears • Peristomal skin complications • Wounds associated with eczema or skin inflammation	Variable risk, increasing with: • Wounds exposed to contaminants • Delayed presentation • Poor hygiene • Foreign bodies • Burns • Immunosuppression or inadequate wound cleansing	• Fever • Behavioural changes • Refusal to walk or use affected area, guarding • Crying during dressing change • Irritability • Increasing wound pain • Spreading local warmth, oedema, malodour, purulent exudate	• Engage caregivers as diagnostic partners • Assess tetanus status where relevant • Prioritise pain and distress control • Use play, distraction and caregiver presence • Cleanse gently but effectively • Escalate with signs and symptoms of spreading or systemic infection.
School-aged children	• More mature skin barrier and immune responses • More reliable symptom reporting • Psychosocial impact of visible wounds, odour and dressings may become important	• Trauma • Sports injuries • Burns • Surgical wounds • Infected abrasions • Wounds/PIs in those with disability or chronic disease	Moderate risk overall, higher in children with: • Diabetes • Vascular compromise • Immunosuppression • Malnutrition • Neurological impairment • Complex care needs	• Fever • Reluctance to participate in usual activities • Anxiety about wound care • New or increasing pain • Wound enlargement, increased exudate, odour, delayed healing, spreading erythema or colour change.	• Combine clinical assessment with child-reported symptoms • Promote age-appropriate education • Support self-care participation • Assess psychosocial impact • Use antimicrobial therapy only when clinically indicated
Adolescents	• Skin physiology approaches adult patterns • Wound outcomes strongly influenced by adherence, body image, autonomy, risk-taking behaviours and psychosocial factors	• Sports injuries • Surgical wounds • Burns • Pilonidal disease • Acne-related wounds • Wounds/PIs in those with disability, chronic disease or self-care challenges	Variable risk that may increase with: • Immunosuppression • Diabetes • Delayed help-seeking • Smoking/vaping • Obesity • Mental health concerns • Non-adherence • Social barriers	• Concealment of symptoms and delayed reporting • Reduced participation in school, sport or social activities • Avoidance of dressing changes • Increased pain • Wound deterioration, malodour, exudate	• Respect autonomy and confidentiality • Use shared decision-making • Address barriers to adherence • Involve family appropriately while supporting adolescent independence • Provide clear education about AMS • Encourage cessation of risky lifestyle factors • Consider psychosocial and body image concerns
Medically complex or immune compromised children of any age	• Age-specific factors as listed above • Altered immunity • Impaired healing, • Malnutrition • Repeated hospitalisation • Invasive devices • Antibiotic exposure • Complex care pathways	• Oncology-related wounds • Surgical wounds • PIs and DRPIs • Stoma-related skin breakdown • Epidermolysis bullosa • Wounds related to neurological impairment or immobility	High to very high risk: • Classical signs of infection may be subtle • Deterioration may be rapid • Resistant organisms and recurrent infection are more likely	• Non-specific deterioration • Reduced interaction • Fatigue • Fever, or absence of fever despite infection • Increased pain • Delayed healing despite appropriate care, change in exudate or odour • Caregiver concern	• Maintain a low threshold for reassessment • Involve multidisciplinary teams and continuity across care settings • Consider early input from microbiology and infectious diseases experts • Optimise nutrition, perfusion and pressure relief • Practice AMS

Environmental factors such as humidity, temperature regulation and care practices also influence skin barrier development and injury risk.[469, 470, 472, 479] Medical adhesives, medical devices and repeated handling, which are ubiquitous in the care of preterm neonates, are not only sources of mechanical injury but also modifiable infection risk factors.[469, 472, 484, 488] In this cohort, prevention of skin injury equates to prevention of infection. Despite this, up to 80% of infants born between 22 and 24 weeks' gestation experience skin or tissue injuries.[469, 489]

As the skin develops, so too does the skin microbiome. The skin microbiome plays an important role in immune regulation and microbial balance. Disruption of microbial homeostasis may contribute to infection and impaired healing.[470, 476] Skin pH, which plays a critical role in maintaining barrier integrity and regulating microbial colonisation, changes with skin maturation. Healthy skin is slightly acidic (pH 4.5–5.5), which supports antimicrobial defence and epidermal enzyme activity. However, immature neonatal skin has a neutral pH, gradually acidifying during the first weeks of life as lipid production, enzyme activity and microbial colonisation develop.[469, 470, 484] In premature infants this acidification process may be delayed due to immature epidermal structures and metabolic pathways.[470, 484]

When the skin barrier is disrupted, the wound environment becomes more alkaline (often pH 6.5–8.5) (see Chapters 6 and 10). Currently there are no paediatric-specific pH thresholds for predicting wound infection or biofilm-associated delayed healing.[469]

Paediatric wound aetiologies and infection risk profiles

Paediatric wound infection risk is strongly influenced by wound aetiology. Commonly seen wounds aetiologies are outlined in **Table 26**. Several factors influence the prevalence patterns seen across the age/developmental stages. In neonates and infants, the most commonly seen wounds are related to interventional care, for example device-related pressure injuries (DRPI), medical adhesive-related skin injury (MARSI), extravasation injuries and surgical wounds.[485] As children grow and become mobile, wound prevalence patterns reflect an increased risk of accident-associated wounds (e.g., trauma, burns and bites), and those associated with chronic disease, disability and dermatological conditions.[486] Burns and contaminated traumatic wounds are especially relevant in resource-limited settings[487] where delayed presentation, limited access to clean water, restricted access to microbiology and reduced availability of appropriate antimicrobials contribute to increased infection risk.

Wound infection prevention and control

Preventing skin injury and therefore the risk of infection is a primary objective in neonatal care. Clinical interventions aim to prevent wound infection in neonates, infants and children by **[Table 27]**:

- Promoting skin integrity,
- Minimising trauma to maturing skin from pressure, shear, friction and moisture,[485] and
- Reducing the risk of infection should the skin barrier become compromised.

Age-related wound infection identification, assessment and diagnosis

Recognising wound infection can be challenging in the youngest ages (very preterm, preterm and neonates) and in infants and non-verbal children. Indicators of local wound infection[27] may present subtly, be difficult to interpret or be completely absent. Systemic or behavioural deterioration may precede obvious wound changes. Therefore, application of the IWII Wound Infection Continuum [see **Figure 5: IWII-WIC**] in neonates and children requires age-specific interpretation. Assessment for wound infection in neonates, infants and non-verbal children should include:

- Signs and symptoms of local wound infection (i.e., delayed healing, erythema, warmth, swelling, increasing exudate, malodour, new/increasing pain),[141] and
- Systemic or behavioural indicators of wound infection (e.g., temperature instability, feeding intolerance, apnoea, bradycardia, lethargy, irritability, altered sleep, reduced interaction, reduced movement of the affected area and carer reports of concern).[483, 487]

Table 27: Key strategies to reduce wound infection risk in neonatal and paediatric care

Promote awareness[481]
- Education for clinicians and parents/carers
- Routine "skin rounds" in paediatric care settings

Promote skin integrity and function[469, 470, 472, 476, 477, 479, 484, 485, 490]
- Maintaining environmental humidity
- Maintaining moisture balance by treating maceration or excessive dryness
- Address skin hygiene, particularly with nappy/diaper changes

Prevent skin injury[469, 472, 481, 482, 485, 486]
- Regular skin inspection should include checking underneath devices
- Earliest possible removal of medical devices
- Regular device repositioning
- Rotate between different devices when possible (e.g., change between nasal prongs and mask)
- Secure devices using strategies that reduce pressure and shear
- Use silicone-based adhesives and atraumatic dressings
- Use a suitable interface between devices and the skin
- Use adhesive removal products

Prevent introduction of infection[470, 472]
- Routine comprehensive skin assessment
- Meticulous hand hygiene
- Aseptic technique during wound procedures

Diagnosis of wound infection remains primarily clinical. It should be based on the overall pattern of wound signs, systemic indicators, host vulnerability, wound aetiology, device exposure, immune status and clinical trajectory. A positive wound culture alone should not be interpreted as wound infection, because open wounds may be colonised without causing a clinically relevant host response or delayed healing.[141]

Microbiology results should always be interpreted alongside clinical findings.[141] In neonates, suspected sepsis or systemic deterioration should prompt urgent assessment and escalation according to local neonatal or paediatric sepsis care and referral pathways.

Wound infection treatment in neonates, infants and children

Local wound care: therapeutic cleansing and debridement

Optimal wound management, including therapeutic cleansing, debridement and exudate management can reduce microbial burden and limit the need for systemic antibiotic therapy.[469] Therapeutic wound cleansing in neonates, infants and children should be effective and non-traumatic. In fragile neonatal skin, cleansing should minimise friction, avoid unnecessary disruption of viable tissue and protect the periwound and surrounding skin. The choice of a cleansing solution should consider:[469, 470, 480]
- Wound aetiology,
- Age-stratified wound infection risk,
- Skin maturity,
- Wound size,
- Exposed structures,
- pH,
- Cytotoxicity,
- Systemic absorption risk, and
- Local policy.

Cleansing should be adapted to the clinical objective, with routine wound assessment, removal of debris, preparation for dressing application, preparation before microbiological sampling, or management of suspected infection. In suspected infection, therapeutic cleansing should support visualisation of the wound bed, reduce surface contaminants and

prepare the wound for appropriate dressing selection or further intervention. Warming the solution may be more soothing than using a cold solution, reducing procedural distress.[485]

The decision to debride the wound bed should balance infection control, tissue preservation, procedural pain and the overall goals of care.[141, 383] Debridement in neonates and children should be individualised and undertaken only after assessment of the:[485]

- Wound (e.g., aetiology, tissue viability, depth, local perfusion, exposed structures),
- Child's context (e.g., clinical stability, pain and bleeding risk), and
- Infection risk.

Conservative approaches, including autolytic or gentle mechanical debridement,[485, 491] are generally preferred when clinically appropriate, particularly in fragile neonatal skin. Sharp or surgical debridement may be required in selected cases, particularly where devitalised tissue, abscess, necrosis, foreign material or spreading infection is present.[383] These procedures should be performed only by trained and competent clinicians and with appropriate analgesia, sedation or anaesthetic support when needed.[383]

Wound dressings should protect fragile tissue, maintain moisture balance and minimise trauma during removal.[484, 486] In neonates, infants and children, consideration should be given to preventing the child from removing the dressing (e.g., use a medical wrap, cotton mittens, elastic stockinette tubing, etc.). Older children and adolescents should be educated about the role of the wound dressing in protection and infection prevention.

Procedural pain management

Pain associated with wound care procedures is common in neonates, infants and children.[481] Untreated pain may lead to psychological instability and potential long-term alterations in pain perception.[481, 492] Developmentally appropriate pain assessment tools should be used (e.g., the Neonatal Infant Pain Scale [NIPS], Premature Infant Pain Profile [PIPP], Face, Legs, Activity, Cry, Consolability Scale [FLACC], Faces Pain Scale-Revised [FPS-R]).[485] Pain management strategies include pharmacological and non-pharmacological approaches[485] (e.g., distraction, swaddling, breastfeeding, sucrose administration and parental presence during procedures).

Antiseptic safety in neonates and children

Antiseptics in solution or antimicrobial-dressing form should not be used routinely in the absence of clinical indication.[486] Not only is AMS critical in wound care due to the increasing threat of antimicrobial resistance, but in neonates, infants and children, inappropriate antibiotic use may disrupt the developing microbiome and contribute to resistant organisms.[476] The clinical decision to use antiseptics should balance antimicrobial efficacy against the risk of cytotoxicity, chemical injury, systemic absorption and disruption of skin barrier maturation.[469, 470, 480, 493] This is particularly important in extremely preterm and very-low-birth-weight infants because of their immature stratum corneum, high body surface area-to-weight ratio and increased skin permeability.[469, 480]

However, antiseptics and antimicrobial dressings may have a role in the presence of clinical signs of local wound infection, concern about biofilm-associated delayed healing or high infection risk.[472] Their use should be clinically justified and reviewed regularly. Consideration should be given to the following factors when selecting a local antiseptic (solution or wound dressing):[472, 480]

- Gestational and postnatal age,
- Weight,
- Wound size and exposed surface area,
- Exposure time required for optimal efficacy of the product,
- Need for removal after application,
- Potential adverse effects associated with absorption of chemicals (e.g., iodine),
- Local AMS guidelines (see Chapter 2), and
- Availability of safer treatment alternatives.

Silver dressings should not be used as routine prophylaxis in infants. Some guidance[494] indicates that silver dressings can be used safely for a limited duration of up to 2 weeks in

infants. However, for infants less than one year of age, silver dressings should only be used where their benefits clearly outweigh alternative treatments. Nanocrystalline silver dressings with slow release may deliver steady antimicrobial effects and reduce the risk of adverse effects compared to traditional silver delivery methods.[494]

Holistic approach to family-centred education and safety-netting

Family caregivers are essential partners in paediatric wound infection prevention and early recognition. Carer-reported changes should be considered clinically meaningful, particularly in neonates, infants and non-verbal children. Parents/carers are often the first to detect early changes in feeding, sleep disturbances, pain/comfort or irritability, mobility/activity, behaviour, crying patterns, fever or temperature instability, wound signs and symptoms or interaction before these changes are obvious during clinical assessment. These observations may facilitate earlier clinical assessment and intervention.

Families should receive clear safety-netting advice, education and empowerment to actively participate in the early recognition and monitoring of wound infection. Family education should include:

- Observing changes in the wound and periwound skin, including the development of erythema, maceration, oedema, tissue deterioration or delayed healing.
- Recognising signs of local wound infection, such as increased exudate, malodour, pain, warmth, friable granulation tissue or wound enlargement.
- Monitoring systemic signs of infection, including fever, increasing pain, reduced feeding, lethargy, irritability, or other age-specific indicators of clinical deterioration.
- Using the prescribed dressings correctly to manage exudate, maintain an optimal wound healing environment, and support local infection control.
- Promptly seeking professional reassessment when multiple clinical changes occur simultaneously or when there is evidence of wound deterioration, treatment failure or progression towards local or systemic infection.

Wounds and wound care procedures can have significant psychological impacts on children and their families. Repeated painful procedures may cause anxiety, distress and behavioural changes. Children may also experience paediatric medical traumatic stress associated with hospitalisation and painful medical interventions.[481] Parents/carers frequently experience emotional distress related to the child's illness or injury, which can influence the child's emotional response to treatment. Family-centred care, promotion of self-care/independence for older children, and parental involvement in treatment decisions can help reduce anxiety and improve coping.[486]

Chapter 14
Future initiatives: IWII strategic priorities for wound infection

Introduction

Wound infection remains one of the most significant challenges in chronic/hard-to-heal wound care, contributing to delayed healing, increased burden and mortality for individuals with wounds, higher healthcare costs, and escalating antimicrobial resistance. Over the next decade, advances in diagnostics, digital health, biomaterials, and regenerative therapies will create new opportunities to improve prevention, detection, and management. At the same time, global pressures—including population ageing, chronic disease, climate change, healthcare inequities, and antimicrobial resistance—will require approaches that are evidence-based, patient-centred, and adaptable across diverse healthcare settings. This section summarises key priorities and future directions identified by the International Wound Infection Institute (IWII) as being required to advance wound infection science and practice into the next decade.

Key drivers of change

- Rising prevalence of ageing, diabetes mellitus, vascular disease, frailty, and multimorbidity.
- Escalating antimicrobial resistance (AMR).
- Climate change, natural disasters, migration, conflict, and humanitarian crises.
- Rapid digital transformation, including telehealth, wearable sensors, artificial intelligence (AI), and remote monitoring.
- Increasing pressure to deliver equitable care across both high- and low-resource settings.

Strategic priorities

1. Transform diagnosis and assessment

Future diagnostics should combine:

- Clinical assessment,
- Molecular diagnostics,
- Imaging technologies (fluorescence, hyperspectral imaging, thermography), and
- Host and wound biomarkers.

The goal is earlier, more accurate infection detection while supporting antimicrobial stewardship.

2. Advance understanding of slough and biofilm

A major research priority is developing internationally agreed classifications of:

- Inflammatory slough,
- Infective slough, and
- Biofilm-associated slough.

Better phenotyping could improve decisions regarding therapeutic cleansing, debridement, antimicrobial use, and healing expectations.

3. The socio-microbial environment: New and future perspectives

Wound infection should be viewed as a dynamic interaction between four interconnected domains:

- Microbial ecology and biofilm,
- Host immunity and tissue perfusion,
- Local wound environment, and
- Social determinants and healthcare access.

Infection develops when these factors converge to create conditions that impair healing and increase susceptibility to tissue damage. Future wound infection science must therefore move beyond simply identifying the presence of microorganisms and adopt a more holistic understanding of infection risk. Future diagnostic and treatment models should integrate microbial and host phenotyping while recognising the influence of wound characteristics, health literacy, socioeconomic factors, and access to care. This broader socio-microbial perspective has the potential to improve risk prediction, guide targeted interventions, and enhance healing outcomes.

4. Harness AI and digital health responsibly

AI has potential to support:

- Wound assessment and measurement,
- Risk prediction,
- Clinical decision support,
- Antimicrobial stewardship, and
- Education and workforce development.

However, implementation must ensure transparency, validation, explainability, bias mitigation, and human oversight.

5. Accelerate innovation in biomaterials and regenerative therapies

Promising developments include:

- Smart dressings with sensing and responsive drug delivery,
- Biomaterials targeting biofilm and infection,
- Microneedle and bio-printed therapies,
- Microbiome-based therapies, and
- Bacteriophage therapy.

Clinical adoption should be guided by robust evidence demonstrating improved healing, safety, patient outcomes, and cost-effectiveness.

6. Promote global equity, sustainability and climate resilience

Future guidance should address:

- Climate-related impacts on wound infection,
- Disaster and humanitarian settings,
- Vulnerable populations,
- Low-cost diagnostic and treatment pathways,
- Sustainable approaches that reduce carbon emissions and plastic use, and
- Adaptation of recommendations to local resources and contexts.

Innovation must be globally accessible, not restricted to highly resourced environments.

Research priorities

Key research areas include:

1. Multimodal infection diagnostics.
2. Affordable diagnostics for low-resource settings.
3. Microbiome signatures and infection prediction.
4. Biofilm-focused clinical trials.
5. Validation of smart dressings and biomaterials.
6. Ethical AI frameworks.
7. Slough phenotype research.
8. Climate–infection interactions.
9. Digital education effectiveness.
10. Regenerative therapies integrated with infection control.

Conclusion

The future of wound infection care must be earlier, smarter, more precise, more responsible, and more equitable. Progress will depend on the integration of multimodal diagnostics, improved understanding of slough and biofilm, responsible use of artificial intelligence, advances in biomaterials and regenerative therapies, and stronger alignment with antimicrobial stewardship. Future research and innovation must extend beyond technological development to demonstrate meaningful improvements in healing outcomes, patient experience, cost-effectiveness, and health equity. As global challenges such as antimicrobial resistance, climate change, population ageing, and healthcare disparities continue to evolve, wound infection science must remain patient-centred, evidence-based, and adaptable across diverse healthcare settings and resource environments.

Chapter 15
Glossary

Acute wound: *(2016 IWII consensus definition)* A wound with an aetiology that occurs suddenly (e.g., trauma or planned surgery), either with or without intention, but then heals in a timely manner.

Adjunct: Interventions that are used in addition to the standard/usual primary interventions for wound care. Adjunct therapies enhance the impact of primary wound care interventions.

Antibiotic: A natural or synthetic medicine that has the capacity to destroy or inhibit bacterial growth.[141]

Antibiotic cycling: The planned rotation of antibiotic classes used within a health service to reduce the risk of microbial resistance. Health services that practice antibiotic cycling switch between similar types of antibiotics (e.g., switching between different aminoglycosides or ß-lactams) on a pre-defined block time schedule (e.g. every few weeks to months).

Antimicrobials/antimicrobial agent: An all-encompassing term for any natural, semi-synthetic, or synthetic substance that kills or inhibits the growth or activity of microorganisms. It includes antibiotics, antivirals, antiparasitics, antifungals and antiseptics. It also includes disinfectants, which are used for killing or inhibiting microorganism growth on environmental surfaces (i.e., not applied to wounds).[2]

Antimicrobial resistance: *(2022 IWII consensus definition)* The ability of a microorganism to grow in the presence of an antimicrobial agent at concentrations that would normally inhibit or kill susceptible organisms. Antimicrobial resistance occurs when microorganisms change over time in ways that render the antimicrobials ineffective.[141, 495]

Antimicrobial spectrum: The specific range of microorganisms (like bacteria, fungi, viruses, or parasites) that a particular antimicrobial drug can inhibit or destroy. It is the primary way clinicians classify antimicrobial agents, guiding everything from initial empiric therapy to targeted therapies.

Antimicrobial stewardship: The supervised and organised use of antimicrobials in order to decrease the spread of infections that are caused by multidrug-resistant organisms and to improve clinical outcomes by encouraging appropriate and optimised use of antimicrobials.[40]

Antimicrobial tolerance: *(2022 IWII consensus definition)* The ability of a microbial population to survive transient exposure to an antimicrobial agent without a corresponding increase in the minimum inhibitory concentration.[136]

Antiseptic: *(2022 IWII consensus definition)* A topically administered agent with broad-spectrum activity that inhibits multiplication of, or sometimes kills, microorganisms. Depending upon its concentration, an antiseptic may have a toxic effect on human cells. Development of resistance to antiseptics is uncommon.[136]

Asepsis: A state of being free from infectious (pathogenic) agents.[264]

Aseptic technique framework: A practice framework to prevent microorganism cross-infection when performing a wound dressing procedure.[264] The two accepted standards of aseptic technique are: sterile/surgical aseptic technique and clean/standard aseptic technique.[141, 274]

Bioburden: See microbial burden.

Biofilm: *(2022 IWII consensus definition)* Aggregate of microorganisms that have unique characteristics and enhanced tolerance to treatment and the host defences. Wound biofilms are associated with impaired wound healing and signs and symptoms of chronic inflammation.[136]

Cellulitis: An acute, diffuse and spreading infection of the skin and subcutaneous tissues that occurs when bacteria (commonly *S. aureus* or beta-haemolytic streptococci[496]) and/or their products have invaded surrounding tissues causing acute inflammation and erythema.[497] When noted on periwound skin, cellulitis requires culture and sensitivities of the involved wound, and management with systemic antibiotics.[496]

Chronic/hard-to-heal wound: *(2016 IWII consensus definition)* A wound that makes slow progression through the healing phases or displays delayed, interrupted or stalled healing. Inhibited healing may be due to intrinsic and extrinsic factors that impact on the person, their wound and their healing environment.[135] When a wound fails to heal within an expected timeframe or within 4–6 weeks it is usually considered to be chronic/hard-to-heal.

Clinical competency: A holistic term that refers to overall capacity or ability to perform care in a way that successfully supports the achievement of clinical goals. It involves obtaining, building on and applying knowledge, values, attitudes and skills to achieve desired outcomes and innovation in nursing practice.[498]

Clinical reasoning: Refers to the process of health professionals thinking and acting when they perform assessment, diagnosis, and treatment in clinical situations, taking into account the individual's specific context, preferences and care goals.

Colonisation: *(2022 IWII consensus definition)* Refers to a stage in which microorganisms present within the wound are generally irreversibly attached and presumed to be undergoing limited proliferation. No significant host reaction is evoked and no delay in wound healing clinically observed.[136]

Contamination: *(2022 IWII consensus definition)* Refers to a stage of microbial adhesion in which microorganisms within the wound are presumed to not be proliferating (i.e., the microbial

burden is in a dormant or lag phase of growth). No significant host reaction is evoked and no delay in wound healing clinically observed.[136]

Cytotoxic: Refers to a substance that has a toxic effect on an important cellular function. In the context of wounds, cytotoxicity generally refers to the potential adverse effect of destroying cells that are involved in tissue healing, including keratinocytes, fibroblasts, macrophages and neutrophils that may be a risk associated with applying substances to the wound.[499] CT_{50} refers to the concentration of an antimicrobial agent that causes 50% cytotoxicity to mammalian cells.[349, 350]

Debridement: *(2025 IWII consensus definition)* The removal of devitalised (non-viable) tissue from or adjacent to a wound. Debridement also removes foreign matter, exudate and microorganisms from the wound bed and promotes a stimulatory environment.

Delayed wound healing: Wound healing that progresses at a slower rate than expected. Chronic wounds without infection can be expected to show signs of healing within two weeks.[482]

De novo mutation: The process through which new genes evolve from DNA sequences that were ancestrally non-genic.[500]

Devitalised (non-viable) tissue: Dead tissue presenting as necrotic tissue or slough.[482, 501]

Drug formulary: A drug formulary is a list of pharmacological treatments (in the context of this document, antimicrobial agents) that are recommended or approved within a health service for management of specific conditions (in the context of antimicrobials, for treating specific pathogens). A drug formulary usually includes preferred therapies, routes, dosage and therapy duration.

Empiric treatment: selection of a medication based on clinical judgement and experience.

Erythrocyte sedimentation rate (ESR): A blood test that provides a non-specific indicator of inflammation activity in the body.[502]

Erythema: Superficial reddening of the skin.[482]

Eschar: Necrotic, devitalised tissue that appears black or brown, can be loose or firmly adherent and hard or soft, and may appear leathery.[482]

Exudate: *(2022 IWII consensus definition)* Fluid that is released from tissue and/or capillaries in response to injury, inflammation and/or microbial burden. It is mainly comprised of serum, fibrin, proteins and white blood cells.[136]

Fibrinous wound base/surface: *(2022 IWII consensus definition)* A metabolic by-product of healing occurring as a layer that is loosely adherent to the wound bed. It is composed of serum and matrix proteins that may be white, yellow, tan, brown or green, and has a fibrous or gelatinous texture and appearance.[136]

Foreign body: Presence in the wound of non-natural bodies that may be a result of the wounding process (e.g., gravel, dirt or glass) or might arise from wound treatment (e.g., sutures, staples, orthopaedic implants or drains).

Friable granulation tissue (unhealthy granulation): *(2022 IWII consensus definition)* Fragile tissue that bleeds easily.[136]

Fungi: Single-celled or complex multicellular organisms categorised in the biological kingdom Fungi. This includes many ubiquitous organisms, a small number of which can be pathogenic in humans. Examples of fungi include yeasts, moulds and mildew.

Granulation tissue: The pink/red, moist, shiny tissue that glistens and is composed of new blood vessels, connective tissue, fibroblasts, and inflammatory cells that fills an open wound when it begins to heal. It typically appears deep pink or red with an irregular, granular surface.[482]

Hard-to-heal wound: See chronic wound.

Hypergranulation: *(2022 IWII consensus definition)* An increase in the proliferation of granulation tissue such that the tissue progresses above or over the wound edge and inhibits epithelialisation. It presents as raised, soft/spongy, shiny, friable, red tissue.[136]

Hyperkeratotic tissue: Thick, scaly outer layer of skin displaying red/grey/brown patches of dry, scaly, cracked and/or fissured skin.[262]

Induration: Hardening of the skin and soft tissue around a wound due to inflammation that may be associated with secondary infection.[482]

Inert: An inert solution is one that is biologically inactive.

Infection: Occurs when the quantity or pathogenicity of microorganisms in a wound become imbalanced such that the host response is overwhelmed and wound healing becomes impaired.[106] Transition from non-infected to infected is a gradual process determined by the quantity and virulence of microbial burden and the individual's immune response.[141] See the *IWII Wound Infection Continuum* for more detailed information.

Irrigation: A therapeutic wound cleansing technique that involves flushing a wound with a stream of cleansing or antiseptic solution to remove non-viable tissue and other debris.

Key site: A concept in aseptic technique that defines breaches to the skin integrity through which pathogens could enter the body (e.g. a wound or a puncture site).[264]

Key part: A concept in aseptic technique that defines parts of equipment that make direct contact with the individual or with other equipment during an aseptic procedure such as a WDP (e.g. a wound dressing or forceps tips).[264]

Limb hygiene: *(2025 IWII consensus definition)* The cleansing and drying of the affected limb to achieve and maintain skin integrity.

Local infection: *(2022 IWII consensus definition)* Local infection refers to the presence and proliferation of microorganisms within the wound that evoke a response from the host that often includes delayed wound healing. Local infection is contained within the wound and the immediate periwound region (less than 2 cm). Local infection often presents as subtle (covert) signs that may develop into the classic (overt) signs of infection.[136]

Lymphangitis: Inflammation of lymph vessels, seen as streaking, linear erythema running proximally from a site of infection toward lymph nodes. Presentation reflects inflammation of the underlying superficial lymphatic system. Most often associated with acute bacterial infections including *S. aureus* and *S. pyogenes*, usually requiring management with systemic antibiotics.[503]

Maceration: *(2022 IWII consensus definition)* Maceration refers to wrinkled, soggy and/or soft periwound skin occurring due to exposure to moisture. Macerated periwound skin usually presents as white/pale and is at increased risk of breakdown.[136] In dark skin tones maceration can appear as shiny, grey, purple, or darker discolouration.

Macrodeformation: Large-scale tissue contraction, can be observed when NPWT vacuum is applied to the wound.

Microbial burden: *(2022 IWII consensus definition)* Microbial burden is the number of microorganisms in a wound, the pathogenicity of which is influenced by the microorganisms present (i.e., the species/strain), their growth and their potential virulence mechanisms.[136]

Microdeformation: Small-scale or microscopic changes to the tissue or cell shape or structure.

Microorganism: An organism that is microscopic in size (i.e., too small to see with the naked eye) including bacteria, fungi, yeasts, archaea and parasites. Although viruses are not considered to be living organisms, they are often included when using the general term microorganism.

Minimum bactericidal concentration (MBC): The lowest concentration of an antimicrobial agent to kill 99.9% of the test strain of an organism in controlled *in vitro* conditions.[349, 350]

Minimum inhibitory concentration (MIC): The lowest concentration of an antibacterial agent that completely prevents visible growth of the test strain of an organism in controlled in vitro conditions.[504]

Necrotic tissue/necrosis: Dead (devitalised) tissue that is dark in colour and comprised of dead tissue cells. Necrotic tissue acts as a barrier to healing by preventing complete tissue repair and promoting microbial colonisation. It is usually managed with debridement, but only after a comprehensive assessment of the individual and their wound.[482]

Osteomyelitis: Infection of the bone that occurs through infection of the bloodstream or from a wound that allows bacteria to directly reach bone.[482]

Periwound: (*2025 IWII consensus definition)* The skin and tissue immediately adjacent to the wound edge extending out 4 cm and/or including any skin and tissue under the wound dressing.

pH: pH stands for potential of hydrogen. It is a numeric scale used to measure how acidic or basic (alkaline) a liquid or solution is. The pH scale ranges from 0 to 14, with 7 being neutral, greater than 7 being more alkaline and less than 7 being more acidic. The skin has a natural pH of around 4.0 to 5.5.

Phagocytosis: A cellular process by which certain living cells ingest and destroy other large cells or particles. Phagocytosis is a critical first-line component of the host's defence, with phagocytes (e.g., neutrophils and macrophages) detecting and binding to the cell surface of invading microorganisms in order to eradicate them. The process of phagocytosis also initiates other host immune responses, including the release of proinflammatory cytokines.[505]

Planktonic bacteria: Unicellular bacteria growing in a free-living environment, meaning they are not part of a structured community or biofilm.[506]

Pocketing: *(2022 IWII consensus definition)* Pocketing occurs when granulation tissue does not grow in a uniform manner across the entire wound base, leading to a dead space that can potentially harbour microorganisms.[136]

Potable water: Water that is of a quality suitable for drinking, cooking and bathing. Unless the water supply is known to be safe for consumption, it should be considered non-potable. Tank water, pool water and dam water may or may not be of potable quality.[507]

Procalcitonin: An amino-acid protein biomarker that is associated with the host's immune response to infection. Procalcitonin-guided protocols use monitoring of procalcitonin in serum or plasma samples to guide the initiation and de-escalation of antibiotic use based on the host response.[47]

Prophylaxis: The use of one or more measures to prevent the development of a specific disease.[508] In the context of wound infection, prophylactic interventions can include antiseptic use and debridement. Prophylactic antibiotics are sometimes used to prevent surgical site infection; however, antimicrobial stewardship should guide prescribing to prevent overuse. For most procedures, antibiotic prophylaxis is not recommended. Appropriate indications include pre-surgical infection, high risk of post-surgical infection (e.g. contaminated surgery) or when consequences of infection are high (e.g., cardiac valve surgery).[509]

Pyrexia: Abnormal elevation of the core body temperature (above 38.3°C), usually occurring due to the host's inflammatory response to infection.[510, 511]

Physicochemical environment: The local physical and chemical environment. In the context of wounds, this refers to wound bed characteristics (e.g., pH, temperature, moisture, and oxygen levels). These conditions are known to influence microbes and the ability of the wound to heal.[512]

Psychometric properties: A term that encompasses the reliability and validity of measurement scales, referring to the adequacy and accuracy of the measurement processes.[513]

Sepsis: Sepsis is systemic, excessive and inappropriate inflammation most commonly associated with bacterial infection with multi-organ dysfunction, characterised by a range of signs and symptoms, arising from an overwhelming host response to bacterial, fungal or viral infection.[514] Sepsis occurs on a wide spectrum, with the most severe being septic

shock and imminent risk of death. Presentation of sepsis varies and can be influenced by age, comorbidities and time since onset.[515] Signs and symptoms can include excessive pain, confusion or disorientation, shortness of breath, shivering, high fever, high heart rate, and clamminess, often with local signs such as necrotising soft tissue.[515]

Sequestration: The act of isolating or separating (usually) wound exudate and holding it in a separate space (usually within a wound dressing structure) away from the wound bed.

Slough: (2022 IWII consensus definition) Slough is non-viable tissue of varying colour (e.g., cream, yellow, greyish or tan) that may be loose or firmly attached, slimy, stringy, or fibrinous.[136]

Spreading infection: Refers to microorganisms arising from a wound that spread into adjacent or regional tissues, evoking a response in the host in the structures in the anatomical area beyond the periwound region. Signs and symptoms of spreading infection include diffuse, acute inflammation and infection of skin or subcutaneous tissues.[141]

Stratum corneum: The outermost layer of the epidermis.

Support network: People with personal connection to the individual with a wound and who are involved in their care. This might include significant others, family members, neighbours, colleagues and other people who are providing support (e.g., advocacy, care planning, direct care or other levels of support) to the individual.

Surfactant: *(2022 IWII consensus definition)* A hydrophobic/lipophilic agent that reduces the surface tension between liquid and debris, slough and/or biofilm in a wound. The reduction in surface tension better disperses the liquid, improving the cleansing effect.[136]

Systemic infection: *(2022 IWII consensus definition)* Refers to microorganisms arising from the wound that spread throughout the body via the vascular or lymphatic systems, evoking a host response that affects the body as a whole. Signs of systemic infection include a systemic inflammatory response, sepsis and organ dysfunction.[136]

Therapeutic index: A ratio that quantifies the balance between an antiseptic's *in vitro* antimicrobial efficacy against common organisms and its cytotoxicity to mammalian cells, including cells specifically involved in wound healing (e.g., keratinocytes, fibroblasts, and/or neutrophils).[349, 350]

Therapeutic wound cleansing: *(2025 IWII consensus definition)* Active removal of surface contaminants, loose debris, non-attached non-viable tissue, microorganisms and/or remnants of previous dressings.

Therapeutic skin cleansing: Skin hygiene performed to remove debris, scales, exudate, microorganisms and excessive sweat and lipids from the wider area of skin, particularly when it has been covered by securing bandages or compression bandages/stockings/wraps.[516]

Therapeutic cleansing zones: The IWII defines the therapeutic cleansing zones as the wound bed and wound edge (zone 1), the periwound that extends up to 4 cm beyond the wound edge (zone 2) and the surrounding skin that extends to 20 cm from the wound edge (zone 3).[155]

Therapeutic relationship: A professional, person-centred relationship based on trust, respect, effective communication and partnership, in which the health professional supports the individual's physical, emotional, social and spiritual needs and promotes shared decision-making and wellbeing.[517]

Tunnelling: Tunnels are usually narrow channels or tracts that extend deep into the surrounding subcutaneous tissue or muscle, creating pockets or passages in the underlying tissue, which can complicate healing and create a nidus for bacterial growth.

Undermining: An area of tissue destruction extending under intact skin along the periphery of a wound. It can be distinguished from a sinus tract in that it involves a significant portion of wound edge.[482]

Wound culture: A sample of tissue or fluid taken from the wound bed for laboratory testing. In the laboratory the sample is placed in a substance that promotes growth of organisms and the type and quantity of organisms that grow is assessed.[109, 518]

Wound dressing procedure: The process of preparing the wound for healing and protection of the wound with a wound dressing (i.e., the process referred to as changing a wound dressing). The procedure, which can be performed with differing considerations to asepsis, includes distinct steps and phases.[282, 342] These steps include therapeutic cleansing and debridement, assessment and application of a new wound dressing. Additional activities may also be performed during the wound dressing procedure, including (but not limited to) applying topical agents to stimulate wound healing or performing a wound swab for microscopy, culture and sensitivities.[282]

Chapter 16
Methodology

The recommendations and clinical guidance are underpinned by the best available evidence. The topics of interest for this consensus document were determined through the IWII experts' critical review of the previous (2022) edition and a scoping of emerging issues in wound infection. The IWII experts participated in a group meeting in January 2026 to reach agreement on the document focus and topics for consensus voting.

Identifying and classifying the best evidence

The literature builds on that identified in previous editions. A systematic search was undertaken to identify research relevant to the inquiry topics. The search strategy used MeSH terms and EBSCO terms that were adapted for other databases. Controlled vocabulary searches developed by the IWII experts through successive editions of the document covered the following concepts:

- wound infection, wound, wound care, chronic wound, surgical wound
- wound cleansing, cleaning, cleanse, wound irrigation, asepsis, cleansing, shower, technique, therapeutic cleansing, cleansing solution
- diagnosis, microscopy, imaging, investigations, point of care
- signs and symptoms, clinical assessment, assessment tools
- antimicrobial resistance, antimicrobial stewardship, clinical governance
- biofilm, microbial mechanisms, microbiota
- antimicrobials, antimicrobial, topical agent, antiseptic, surfactant
- therapeutic relationship, alliance, holistic, partnership, person-centred
- resource-limited, natural therapy
- non-pharmaceutical, non-medicated, adjunct, biophysical, negative pressure, light therapy, ultrasound.

Searches were conducted in December 2025 in the following databases: Medline, PubMed, Embase, the Cochrane Library and Google Scholar. Google searches and targeted searches of wound-focused websites were undertaken to identify relevant consensus documents and guidelines. Additional publications recommended by the IWII experts were also included. The search was limited to reports in English since 2022. Identified evidence was screened based on title/abstract for relevance to the inquiry topics. Evidence was classified based on study design/type of article using the Joanna Briggs Institute (JBI) Levels of Evidence for Effectiveness **[Figure 28]**, and this ranking was used to identify the level of underpinning evidence for the recommendations. The JBI level reflects study design and should not be interpreted as a rating of certainty, methodological quality or strength of recommendation.

Consensus process

The IWII also undertook a formal consensus process with a goal of attaining agreement on updates to the IWII-WIC, clinical indicators associated with wound biofilm and the use of pH for wound monitoring. The consensus process was undertaken using the RAND/UCLA Appropriateness Method, a Delphi method for reaching formal agreement on the interpretation of science.[519] The consensus process extended previous work undertaken by the IWII to develop its conceptual frameworks, and used the same previously published process.[135, 136] Participants in the current consensus process included the IWII's Expert Working Group, as listed under the acknowledgements.

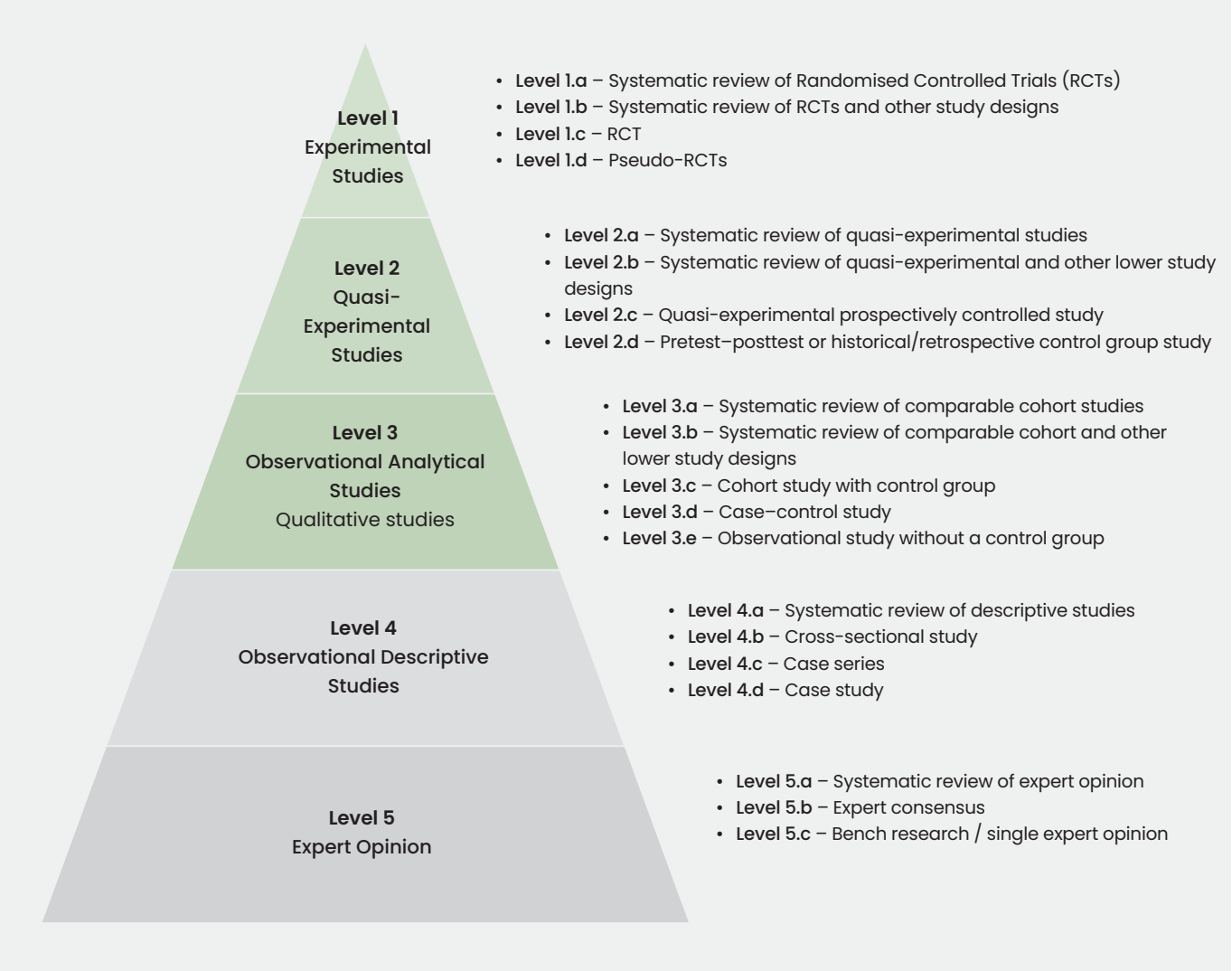


Figure 28. JBI Levels of Evidence for Effectiveness

Appendix A: Profiles of commonly used products for wound infection management and wound/skin cleansing

Antiseptic/ cleanser *	Properties	Antiseptic type	Examples of available formats	pH	Cytotoxicity, therapeutic/ biocompatibility index** and safety profile#	Mode of action
* This is not an exhaustive list of antimicrobial agents. There are multiple different preparations available for most products. Data is indicative only, always read the product information. ** Therapeutic index and biocompatibility index values are dependent on the methods, organisms, mammalian cell types, antiseptic concentrations, exposure times and other variables used in testing. Values from different studies are not necessarily directly comparable. Values should be interpreted within the context of the methods used in each study rather than as an absolute ranking of antiseptic products. A value greater than 1 generally indicates that most microbes have been destroyed while not destroying more than 50% of mammalian cells.[348] No validated cutoff thresholds have been established.[333, 348] # Always review the manufacturer's information regarding safe product use. Refer to Chapter 10 for more information						
Antiseptic solutions and dressings						
Acetic Acid	Antimicrobial	Acids	Solution: 1%–5% concentration	2.4	• Cytotoxicity to human cells is reported at concentrations as low as 0.25%[520] • Allergic reaction is rare[521] • May cause stinging/ pain on application	• Passively diffuses into bacterial cells, resulting in anion accumulation and osmotic alterations that impair metabolic processes.[522]
Cadexomer iodine	Antimicrobial	Halogens/ oxidising agents	Powder, paste, beads, impregnated tulle		• Dose dependent cytotoxic effect on keratinocytes and fibroblasts[523] • Contraindicated in children <12 years, iodine sensitivity, thyroid or renal disorders and extensive burns[523]	• Slow release of iodine over 72 hours
Chlorhexidine	Antimicrobial	Biguanides	Solution: concentration varies depending on intended use	5.5–7	• Cytotoxicity reported[333, 524] • Reported to damage granulating tissue[525] • Hypersensitivity reported[526, 527] • Mean biocompatibility index at 20% concentration approximately 0.80 to 0.98 depending on pathogen, and may differ with different concentrations[348]	• Binds to bacterial cell wall, interfering with the metabolic capacity of the cell, interferes with the cell membrane integrity causing leakage of cellular material from the bacteria[528] • Tolerance and resistance have been reported in gram-negative and gram-positive bacterial species[527, 528]
Citric acid	Antimicrobial (Used in other preparations to adjust pH)	Acids	Solution: 3% concentration	3–6	• Concentration and pH dependent cytotoxic effect	• Disrupts the bacterial cell membrane and lowers the pH, slowing bacterial growth[529] • Alters bacterial metabolic activity[529]
Copper oxide	Antimicrobial	Metallic compounds	Polymer dressings, foam dressing		• No cytotoxicity reported[530]	• Sustained continuous release of copper into wound bed[530] • In anaerobic conditions, copper interacts with microbial proteins, disrupting their structure and enabling penetration into the cell[530]

Appendix A: Profiles of commonly used products for wound infection management and wound/skin cleansing

Antiseptic/ cleanser *	Properties	Antiseptic type	Examples of available formats	pH	Cytotoxicity and therapeutic/ biocompatibility index** and safety profile#	Mode of action
Antiseptic solutions and dressings						
Gentian violet/ methylene blue (GVMB)	Antimicrobial	Dyes	Polyvinyl alcohol (PVA) and polyurethane foam dressings		• No cytotoxicity reported	• Manages exudate through wicking action • Acting within the dressing, alters the extracellular surface of bacterial membranes with respect to their oxidative metabolism function, creating a hypoxic environment[531, 532]
Glucose oxidase combined with lactoperoxidase, and guaiacol	Antimicrobial	Other: Enzymatic	Alginate gels	5.7	• No cytotoxicity reported	• Inhibitory effect on biofilm[533] • Combined action of autolytic debridement, exudate absorption, and a patented enzyme system that destroys bacteria[534] • Able to penetrate bacterial cell walls and cause non-specific oxidation of membranes and damage to DNA[533]
Honey (medical-grade)	Antimicrobial	Other: Osmotic solution	Liquid form, paste, hydrocolloids, alginates, tulle	3.2–4.5	• Select products that have been gamma irradiated[535]	• Antimicrobial effect relates to production of hydrogen peroxide by an enzyme within honey[536] • Promotes autolytic debridement.[148, 537]
Hypochlorous acid (HOCl) (pure)	Antimicrobial Hypotonic	Halogens/ oxidising agents	Solution: 0.03% concentration, gel 0.05% concentration	3.5–5.5	• Minimal cytotoxicity reported[347, 538-540] • pH can drift if the HOCl solution is impure or degraded[538, 541] • With experimental preparations of ≈0.000029–0.000066% therapeutic index ranged from ≈30-70[349]	• Passively diffuses into bacterial cells, leading to anion accumulation and osmotic alterations that impair metabolic processes[542] • Oxidizes the surfaces of bacterial cells to disrupt membrane function and softens tissue, aiding its removal during cleansing and debridement[283, 543, 544] • Has an anti-inflammatory effect through reducing activity of histamines, MMPs, mast cell and cytokine activity[543, 545]
HOCl combined with sodium hypochlorite (NaOCl)	Antimicrobial	Halogens/ oxidising agents	Solution with various concentrations	7.5–10.5	• Minimal cytotoxicity reported[540] • Mean therapeutic index at HOCl 0.005%/ NaOCl 0.005% ranged from 9.58 to 19.18[350]	• A low concentration of a salt dissolved in water through which electrical current is passed through to create charged ions that react with microbes[546] • Free radicals and ions react to denature bacterial cell walls, disrupting their structure[547] • Concentrations and pH vary[548] • Has anti-inflammatory effects[549]

Antiseptic/ cleanser *	Properties	Antiseptic type	Examples of available formats	pH	Cytotoxicity and therapeutic/ biocompatibility index** and safety profile#	Mode of action
Antiseptic solutions and dressings						
Octenidine dihydrochloride	• Antimicrobial • Surfactant • Cationic	Pyrimidines	Solution: 0.1% concentration, gels, combination with hyaluronic acid, absorbent cellulose	1.6–12.2	• Allergic reaction rare[521] • Avoid use on exposed tendon • Minimal cytotoxicity reported[347] • Mean biocompatibility index at 0.1% concentration approximately 1.73 to 2.11 depending on pathogen.[348]	• Disrupts outer cell membrane and binds to bacteria leading to cell death • Has anti-inflammatory effects[383]
Poly-hexamethylene biguanide (PHMB) with betaine	• Surfactant (betaine) • Antimicrobial (PHMB)	Biguanides	Solution: 0.1% concentration, gels, impregnated gauze, gauze combined with BEC, non-adherent dressing	6–8	• Minimal cytotoxicity is reported[333] • Potential for allergic reaction is low[550] • Mean therapeutic indices range from 1.78 to 5.95 depending on pathogen, and may differ based on concentration[350]	• Polyhexanide increases bacterial membrane permeability and disrupts adenosine triphosphate (ATP) production,[551, 552] interferes with bacterial production of homoserine and interferes with quorum sensing ability[553] • Betaine reduces the adherence quality of microbes, reducing the force required to remove bacteria and debris[552, 554]
PHMB with poloxamer	Antimicrobial	Biguanides	Solutions, foam	3.2–4.5	• Minimal cytotoxicity is reported[555] • Potential for allergic reaction is low[550] • Mean therapeutic indices range from 3.26 to 6.51 depending on pathogen, and may differ based on concentration[350]	• Polyhexanide increases bacterial membrane permeability and disrupts adenosine triphosphate (ATP) production[551, 552] interferes with bacterial production of homoserine and interferes with quorum sensing ability[553]
Povidone-iodine (PVP-I)	Antimicrobial	Halogens/ oxidising agents	Solution: 10% concentration, ointments, creams, nonadherent dressing	4	• Dose dependent cytotoxic effect on cells[347, 523] • Contraindicated in neonates, iodine sensitivity, thyroid or renal disorders and very large wounds[360, 523, 556] • Mean therapeutic indices at 10% concentration ranged from 0.68 to 0.95 depending on pathogen, and may differ with different concentrations[348, 350]	• Oxidises and subsequently destabilises bacterial cell membranes leading to cytosolic enzyme deactivation and cell death[551] • Has anti-inflammatory effects[383]
Polyethylene glycol (PEG) povidone-iodine (PEG-PVP-I)	Antimicrobial	Halogens/ oxidising agents	Tulle	3.2–4.5	• Does not cause significant cytotoxicity • Contraindicated in neonates, iodine sensitivity, thyroid or renal disorders and very large wounds[360, 523, 556]	• Sustained release of iodine at 1% from dressing into the wound.[557] • In contact with moisture, iodine rapidly penetrates microbial cell walls and destroys essential proteins, enzymes, and nucleic acids, which kills a broad spectrum of bacteria, fungi, and viruses[557]

Appendix A: Profiles of commonly used products for wound infection management and wound/skin cleansing

Antiseptic/ cleanser *	Properties	Antiseptic type	Examples of available formats	pH	Cytotoxicity and therapeutic/ biocompatibility index** and safety profile#	Mode of action
Antiseptic solutions and dressings						
Silver sulfadiazine	Antimicrobial	Metallic compounds	Foam, creams		• Dose and time dependent cytotoxic effects on human fibroblasts, keratinocytes and endothelial cells551 may delay epithelialisation[556] • Biocompatibility index < 0.006[348]	• Continuous release of silver [558] • Absorbs bacteria • Eradicates mature biofilm[558]
Silver: oxides, nitrates, phosphate, sulphates and chlorides (metal and nano-crystalline)	Antimicrobial	Metallic compounds	Alginates, foams, polyurethanes, powders, gels, silicone dressings, superabsorbent dressings and others		• Silver has dose-dependent toxicity[558, 559] • No or mild concentration-dependent cytotoxic effect on fibroblasts[558, 560, 561]	• Inhibits biofilm formations[562] • Products differ according to silver composition and amounts • Broad spectrum effects against gram-negative and gram-positive bacteria[563] • Physically interact with a bacterial cell membrane, causing an increase in membrane permeability[563] • Penetrate bacterial cells, interacting with cellular structures [563]
Silver combined with benzethonium chloride (BEC) and ethylenediam-inetetraacetic acid (EDTA)	• Antimicrobial • Surfactant	• Metallic compounds • Quaternary ammonium compounds	Wound dressing: 1.2% ionic silver enhanced with EDTA[564] and BEC[565-567]		• Silver has dose-dependent toxicity[558, 559] • No or mild concentration-dependent cytotoxic effect on fibroblasts[558, 560, 561]	• Prevents biofilm formation,[565, 566] EDTA is a metal-chelating agent that encourages biofilm matrix disruption in this preparation[568] • Accumulates on bacterial cell walls, enters the cell, damaging DNA and inhibiting vital proteins[568] • BEC reduces the biofilm surface tension[568]
Sodium hypo-chlorite (NaOCl)	Antimicrobial	Halogens/ oxidising agents	Solution: 0.0125%–0.5% concentration	9–12	• Dose dependent cytotoxic effect on cells,[569] concentration below 0.025% is suggested[545]	• Free radicals react with and oxidise nitrogen- and sulphur-containing groups on the surface of bacterial cells to disrupt membrane function.[544]
Wound dressings						
Dialkyl-carbamoyl-chloride (DACC)	Antimicrobial effect from physical microbial-binding action	N/A	Tulle, foam, gel, pads		• No cytotoxicity demonstrated in studies[570]	• Capacity to bind with bacteria and biofilms[52] • Bacteria bind to the dressing fibres and are removed with dressing change[52, 393, 571] • Antimicrobial effect is achieved by physical binding[52, 393, 572]
Gelling fibre dressings	Exudate management with sequestration, immobilisation and retention	N/A	Carboxy-methylcellulose, PVA dressings		• No cytotoxicity reported	• Absorbs and traps exudate and microbes • Supports autolytic debridement and wound bed preparation

Antiseptic/ cleanser *	Properties	Antiseptic type	Examples of available formats	pH	Cytotoxicity and therapeutic/ biocompatibility index** and safety profile#	Mode of action
Wound dressings						
Moisture-managing wound dressings	Exudate management with sequestration, immobilisation and retention	N/A	Hydrogels, hydrofibres, hydrocolloids, hydro conductive, Ringer's solution–activated polyacrylate dressing		• No cytotoxicity reported	• Absorbs and traps exudate and microbes • Supports autolytic debridement and wound bed preparation
Negatively charged fibre desloughing dressings	Electrostatic attraction, retention and adherence	N/A	Dressing with polyacrylate fibres with a silver-containing lipido-colloid		• No cytotoxicity reported	• Liquifies slough (solid or gelatinous) allowing for ease of cleansing and debridement.
Superabsorbent dressings	Exudate management with sequestration, immobilisation and retention	N/A	Dressings, silicone SAP dressings		• No cytotoxicity reported	• Removes exudate and suspended bacteria • Very high absorption prevents exudate pooling
Wound and skin cleansers						
Normal saline (NaCl)	Isotonic	N/A	Solution: 0.9% concentration	5.5	• No cytotoxicity reported	• Exact mechanism of normal saline is not known.
pH balanced skin cleanser	Surfactant	N/A	Solutions	4.5-5.5	• No cytotoxicity reported	• May stimulate autolytic debridement and reduce inflammation by influencing protein activity[554] • Reduces the adherence quality of microbes, reducing the force required to remove bacteria and debris[552, 554]
Surfactant wound cleansers (e.g., Poloxamer 407, undecylena-midopropyl betaine and macrogolum)	Surfactant	None unless combined	Commonly combined with antimicrobial/ antimicrobially-preserved agents	5-7.5	• Low cytotoxicity to fibroblasts and keratinocytes *in vitro*[554] but may be affected when combined with antimicrobials	• Removes bacteria without damage to healing wound tissues[554]
Water (sterile)	Hypotonic	N/A	Solution	5.5-7	• No cytotoxicity demonstrated in studies[570]	• Effect achieved through mechanical detachment of contaminants[573]
Water (drinkable/ potable)	Hypotonic	N/A	Solution	6.5-8.5	• No cytotoxicity demonstrated in studies[570] • Not sterile • Safe alternative when sterile solutions not available or feasible (e.g., low resource or community settings)[574] • If no potable water access, use water after boiling and cooling[575] • When using potable tap water, run to remove contaminants before using water[576]	• Effect achieved through mechanical detachment of contaminants[573]

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This International Wound Infection Institute (IWII) consensus document is dedicated to two special founding members. The contributions of **Dr David Keast** and **Dr Greg Schultz** to the global wound community and to the International Wound Infection Institute have been immense.

David and Greg are deeply missed but never forgotten. They are remembered with great affection, love and respect by their IWII colleagues, and their infinite wisdom will continue to underpin wound infection management across the world.

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