TIME*‡ - Principles of Wound Bed Preparation

Clinical observations	Proposed pathophysiology	WBP clinical actions	Effect of WBP actions	Clinical outcomes
Tissue non-viable or deficient	Defective matrix and cell debris impair healing	Debridement (episodic or continuous) · autolytic, sharp surgical, enzymatic, mechanical or biological · biological agents	Restoration of wound base and functional extra-cellular matrix proteins	Viable wound base
Infection or inflammation	High bacterial counts or prolonged inflammation ↑ inflammatory cytokines ↑ protease activity ↓ growth factor activity	· remove infected foci topical/systemic · antimicrobials · anti-inflammatories · protease inhibition	Low bacterial counts or controlled inflammation: ↓ inflammatory cytokines ↓ protease activity ↑ growth factor activity	Bacterial balance and reduced inflammation
Moisture imbalance	Desiccation slows epithelial cell migration. Excessive fluid causes maceration of wound margin	Apply moisture balancing dressings. Compression, negative pressure or other methods of removing fluid	Restored epithelial cell migration, desiccation avoided oedema, excessive fluid controlled, maceration avoided	Moisture balance
Edge of wound - non advancing or undermined	Non migrating keratinocytes. Non responsive wound cells and abnormalities in extracellular matrix or abnormal protease activity	Re-assess cause or consider corrective therapies · debridement · skin grafts · biological agents · adjunctive therapies	Migrating keratinocytes and responsive wound cells. Restoration of appropriate protease profile	Advancing edge of wound

*Courtesy of International Advisory Board on Wound Bed Preparation2003
Adapted from table 6 - ‡Schultz GS, Sibbald RG, Falanga V et al, Wound Rep Reg (2003);11:1-28
Wound Bed Preparation and TIME are clinical concepts supported by Smith & Nephew Medical Ltd.

Wound Bed Preparation is the management of the wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures

Tissue - Remove non-viable or deficient tissue - may be episodic or continuous

Dry necrosis

After debridement

Necrotic Tissue
Sharp surgical debridement
if adequate arterial supply -
alternatively autolytic methods

Slough

After debridement

Sloughy Tissue
Autolytic, enzymatic, mechanical or biological debridement

Infection or inflammation - diagnose and treat infection or inflammatory diseases

Infection

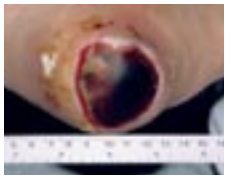
Pyoderma gangrenosum

Vasculitis

Infection - Diagnosis of infection can be difficult. The interpretation of swab results needs to be done with care and always consider clinical features. Management of infection may require intravenous or oral therapy. Consider the use of modern topical anti-microbials. If infection is not resolving after 2 weeks of therapy, consider referral or seek advice.

Inflammation - Consider inflammatory diseases in ulcers that have unusual presentations/appearances and are not responding to first line treatment. Confirmation of diagnosis may require specific blood tests, biopsy or if in doubt, consider referral

Moisture imbalance - correct dessication and avoid maceration



Dessicated wound

Moisture balance achieved

Intervention
Rehydrate/debride (Revisit T)

Wet Venous Ulcer

Healthy venous ulcer

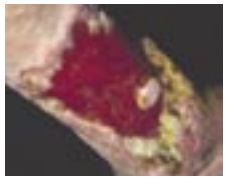
Address Cause
Control oedema by appropriate means. Use moisture balance dressings e.g. foams, alginates, hydrofibres

Edge - consider surgical intervention or advanced therapies if edge is not advancing and T, I & M have been addressed

Undermining edge

Healthy edge

Intervention
Surgical debridement



Non-migrating edge

Migrating edge

Intervention
Reassessment. Consider biological agents, advanced therapies or skin grafting.

Selection and interventions will be based on clinician's knowledge, skills, resources, patient choice and cost-effectiveness.