WOUNDS-UK XN-SQNOOM

APPLIED WOUND MANAGEMENT SUPPLEMENT

3

Sponsored by:



Editorial. David Gray

- 4 Wound Bed Preparation And its Implication for Practice: An Educationalist's Viewpoint. V Jones
- 9 The Wound Healing Continuum. Gray, White, Cooper and Kingsley
- 13 The Wound Infection Continuum. Kingsley, White and Gray
- **19 The Wound Exudate Continuum.** *Gray and White*
- 22 Common Wound Types and their principles of Management. Cooper, Russell and Stringfellow

31 Applied Wound Management. Gray, White Cooper and Kinglsey

The key to progressive wound healing



MAKING SENSE. MAKING PROGRESS.

hnson Johnson ADVANCED WOUND CARE division of ETHICON



Applied Wound Management: A New Conceptual Framework in Wound Management

elcome to this Applied Wound Management [AWM] supplement which contains a series of articles which present a new conceptual framework. The AWM framework has been designed to support clinical decision making and clinical audit. Software to support AWM will be available across the UK as the year progresses.

In the first article Vanessa Jones sets the scene by reviewing Wound Bed Preparation [WBP] from an educationalists point of view. It is clear that WBP has focussed the practioners attention on a more systematic approach to wound management than was perhaps the case previously. WBP has utilised existing concepts and presented them in a systematic manner. It is from within the new paradigm of WBP that the Applied Wound Management framework has emerged, a framework which further develops the concept of a systematic approach to wound management.

Applied Wound Management utilises three wound continuums, Healing, Infection and Exudate. These three continuums are each explored in individual articles within the supplement. By utilising these continuums the practioner is able to arrive at a point where after recognition of the wound pathology a definition of wound type can be arrived at. This approach greatly facilitates a logical and systematic approach to management, encouraging the practioner to recognise the factors which may affect wound healing. By facilitating the defining of wound types the AWM framework also facilitates clinical audit and provides definitions which can be used in different centres across the UK, providing useful data. As data begins to be collected on a reasonable scale across the UK, so a pattern of wound management in the UK will emerge. This type of clinical audit data is vital if the wound management field is to progress in the UK. At present we are unable to state with any certainty the numbers and healing rates associated with the most common wound types in the UK, the emergence of the AWM software should see this situation change.

When a practioner uses the three continuums it is vital that they consider the underlying pathology of the wound. Cooper, Russell and Stringfellow provide an insight into the key management principles associated with common wound types. It is essential that the practioner takes account of these different issues before embarking upon a plan of treatment / management. Failure to recognise the need for specialist referral or appropriate investigation can lead to delayed healing or serious complications

Applied Wound Management is a decision making framework supported by three continuums. It can never be a substitute for clinical knowledge or expertise it is an adjunct to both decision making and audit. When used by a practioner correctly it should support a systematic and logical approach to wound management which in turn should lead to improved clinical outcomes for those individuals with wounds.

David Gray

Clinical Nurse Specialist Department of Tissue Viability Grampian Acute Health Services

> Publishers Wounds-UK, 8 Prospect Terrace, Aberdeen AB11 7TD 01224 582355, www.wounds-uk.com email: enquiries@wounds-uk.com.
> Design and Print The Studio Bleackheath Ltd. 72a Old Dover Rd. London SE3 8SY www.thestudio-london.com email: design@thestudio-london.com.

Wound Bed Preparation and it's Implication for Practice: An Educationalist's Viewpoint

Abstract

A growing understanding of normal and chronic wound healing has allowed identification of the defects that arise within chronic wounds and the way in which they act as barriers to healing. This has produced a new model of chronic wound healing that incorporates features common to all chronic wounds. The concept of wound bed preparation (WBP) uses this model to identify and treat barriers to healing so that a wound can heal endogenously or respond positively to bioactive therapies. In this way WBP directs clinicians to treat the wound with an active and systematic approach rather than simply dressing it. Adoption of this concept raises a number of educational issues. Knowledge of wound biology and microbiology are required to understand the need for and the effect of appropriate therapies. This leads to a need for a change in practitioner attitudes where common features of different chronic wounds are considered for treatment and to a subsequent aquisition of decision making skills necessitated by the complexity of treatment choices available.

> ver the last 20 years technological advances linked to our rapidly increasing knowledge of the healing process have led to the introduction of a number of new therapies that may be used for treatment of chronic wounds . Examples include growth factors (Jaschke, 1999) or tissue engineered dermal replacements (Roberts, 2002) that are designed to stimulate the healing process and pharmacologic agents (Fray, 2003) that inhibit factors delaying healing. Experience of somewhat disappointing results with growth factor therapy suggests that no single modality can act as a panacea for chronic wounds (Robson, 1999). This is most likely a consequence of the complexity of the healing process and also because of the adverse environment within a chronic wound (Trengrove, 1999). It has become increasingly apparent that the wound has to be in an appropriate state for these treatments to achieve their full potential (Nagai, 2002).

> The recognition that no matter how advanced a therapy the wound still requires optimal care and preparation has led to the development of wound bed preparation (WBP) as a standardised system of wound care. In this way it is hoped that success obtained in clinical trials of advanced therapies

performed at wound care centres may be transferred to more general treatment within the clinical community with maximum benefit for the patient (Falanga, 2000). Development of the WBP concept and encouragement of its adoption by the wound healing community has required the development of a model of the chronic wound which classifies it's cell biology and the barriers to healing that need to be eliminated to prepare the wound for healing. This allows wound status to be categorised and appropriate treatment defined.

DO WE NEED A NEW MODEL OF WOUND HEALING?

Conventionally chronic wound treatment has been considered in the context of the known physiology of acute wounds. These follow a predictable healing sequence leading from injury to wound closure and scar formation (Cherry, 2001). The intervening phases of inflammation, granulation tissue formation and re-epithelialisation are tightly controlled with regulated cell activation, proliferation and finally down regulation as healing is completed in healthy individuals. For those patients with comorbidities (Mulder, 1998), possibly exacerbated by ageing, delayed healing and wound chronicity can become problematic. Even when underlying pathologies such as peripheral vascular disease are treated with conventional therapy a proportion of wounds fail to heal (Franks, 1995).

The chronic wound differs markedly from the acute wound in that the biological processes involved are disordered and positive healing events occur on a slower timescale than the acute wound. By comparison to the acute wound the chronic wound is characterised by exhibition of a number of determinants that contribute towards non-healing:

- High levels of proteases
- Bacterial colonisation / infection
- Chronic inflammation
- Disordered growth factor profiles
- Defective granulation tissue
- Inhibited re-epithelialisation

Vanessa Jones Senior Lecturer Wound Healing Research Unit Cardiff Medicentre Heath Park Cardiff CF14 4UJ UK Previously, wound care professionals have considered chronic wounds as falling into particular classes of wound - pressure ulcer, venous leg ulcer or diabetic foot ulcer. These are defined by their aetiology and managed with conventional treatments (for instance, Dealey, 1994). There are however, many features shared by these wounds. They are all a consequence of failure of dermal and epidermal repair and share common biological characteristics such as elevated protease levels (Yager, 1999). Thus, when the different underlying pathologies have been treated there remains a commonality that can be drawn into a unifying chronic wound model with cell biological processes that differ significantly from the acute wound. A unified model of the chronic wound can then be used to provide a rational and systematic approach to prepare a wound for stimulation of healing -Wound Bed Preparation.

WOUND BED PREPARATION

"Wound bed preparation is defined as the global management of wounds to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures" (Falanga, 2002).

For the experienced wound care clinician the overall steps required for WBP are obvious (Falanga, 2000) in that wounds need to be free from infection and necrosis, have a well vascularised wound bed with no fibrinous material or scarring and good exudate control. However before considering treating the wound it is essential to identify and rectify if possible the cause of tissue damage and those extrinsic factors which impair healing.

The chronic wound will not initiate healing until the correctly balanced biomolecular environment is established by WBP to remove local barriers to healing. The first step is removal of eschar by debridement. Necrotic tissue inhibits healing by stimulating chronic inflammation, acting as a focus for infection and forms a mechanical barrier to keratinocyte migration thus preventing reepthelialisation. In acute wounds autolytic debridement is achieved by enzymatic digestion of eschar as part of the normal healing process. Paradoxically, despite high levels of proteases in chronic wounds it is necessary to use surgical, mechanical or enzymatic methods to achieve debridement. Removal of necrotic tissue eliminates one of the factors promoting infection although therapeutic intervention may be required to decrease the bacterial burden of the wound tissue.

Bacteria contribute to delayed healing directly by production of virulence factors or by stimulating chronic inflammation resulting in the biochemical imbalance characterising chronic wounds. It is not necessary to totally eliminate wound bacteria as wounds are capable of healing whilst still producing positive bacterial cultures. The objective of WBP is to achieve numbers and species of bacteria, ie. an appropriate balance (Robson, 1999; Trengrove, 1996) that will allow healing to proceed.

Exudate management is another cornerstone of WBP for chronic wounds. Acute wound exudate is stimulatory for healing whilst that from chronic wounds will inhibit proliferation of fibroblasts, endothelial cells and keratinocytes (Brantigan, 1996) and excessive exudate production requires management to achieve an optimally moist wound environment for healing to proceed. Conversely if the wound bed is too dry an appropriate moisture balancing dressing may be selected to allow rehydration of the wound bed.

Our model of the chronic wound based on an understanding of the cellular and molecular events involved in generation and maintenance of wound chronicity now allows us to understand how a particular intervention will modulate biological events in the wound (Schultz, 2003) (Table 1). In this way the concept of WBP integrates clinical practice and experience with scientific knowledge to provide a clinical framework and knowledge base for education and further research. This framework based on assessment and treatment of the barriers to healing has been given the acronym of TIME by division (Sibbald, 2003) into the four areas of:

- T Tissue
- I Infection
- M Moisture balance
- E -- Epidermal advancement

IS IT A PARADIGM SHIFT?

The Oxford concise dictionary defines a paradigm as "an example or pattern which is used to explain an underlying theory or methodology". Clearly WBP falls into such a definition because of the way it describes the chronic wound and explains how appropriate treatments may exert a beneficial effect. Such a description is important because for a paradigm to have value it has to be believed in and acted upon by its adherents – in this case

Wound bed preparation, chronic wound, education, treatment clinicians, scientists and patients. Such beliefs can be powerful but not always to the benefit of mankind. Historically the flat earth theory was a widely held belief. However because the fear of falling off the edge of the world inhibited distant exploration it could be thought to have exerted a negative impact on human development. Medical paradigms can also be held as a truth for long periods. The Roman physician Galen postulated that blood flowed outwards from the liver and carried nutrients to the periphery where "it was consumed". It took 1500 years before Harvey demonstrated experimentally in the 17th century that blood re-circulated throughout the body (Porter, 2002).

So it would seem that the concept WBP fits with the dictionary definition of a paradigm. Is it then of value and does it represent anything new? The need to debride a wound, treat infection and manage excess exudate are long standing modalities for good wound care (Dealey, 1994). The WBP concept has integrated knowledge related to these clinical practices with scientific knowledge of wound chronicity. Falanga (Falanga, 2000) believes that WBP represents a worldview on preparing a wound to heal certainly and it seems to have been bought into by a significant number of international Key Opinion Leaders who are authors on a recent article describing WBP as "a systematic approach to wound management" (Schultz, 2003). Is it new? The conceptual name WBP may be but actually it is a systematisation of practices that were already part of good clinical practice although possibly not practiced systematically. Wound Bed Preparation requires a systematic approach and should allow practitioners to maximise the benefit of the range of dressings available.

The process described by WBP may not be a fundamental change in the same way as a paradigm shift was originally defined (Kuhn, 1962). For this to be the case it would have to represent a scientific revolution that completely changes the world (of wound healing) as we know it. WBP may however represent a more subtle change in our way of thinking about chronic wounds. Instead of using dressings to manage a wound it directs thinking to active treatment of the wound. It is a means to bring the right interventions to the wound at the right time.

ADOPTION OF THE PARADIGM

Adoption of a systematic approach to WBP by practitioners is helped by the thought processes involved in following the TIME acronym and it is necessary to devise an educational strategy for its implementation. For this we can draw on previous experience with the introduction of other systematic concepts to patient care. The "nursing process" is a problem-solving framework designed to enable nurses to plan individual patient care. It originated in the US and was introduced to the UK by the then General Nursing Council in an attempt to redirect nursing away from task orientation towards an individualised treatment system with a rational basis. In a similar way to TIME it had 4 stages to assess the patient, plan care, provide intervention and then evaluate it. Many practitioners found difficulty with its introduction. Medical staff felt that it was simply about documentation making comments such as "I can't get any sense out of the nurses any more because all they are doing is writing things down". Clearly it's introduction did not have the correct approach in terms of developing a new systematic approach to nursing - it was just seen as more paperwork.

The nursing process required a change in knowledge, skills and attitudes for its successful implementation and the same challenges are inherent in adoption of TIME.

EDUCATIONAL CHALLENGES

Knowledge

As discussed earlier, although a knowledge of acute healing is required WBP is about specific issues related to the chronic wound that require to be considered across all wound types because of their common features. The problem of infection , the second component of TIME, is of major importance and knowledge of microbiology is required. Inappropriate use of antibiotics and avoidance of resistance generation demand that we deal with the presence of bacteria in wound tissue understanding that we do not necessarily need to produce a sterile wound environment. A wound may heal even in the presence of some bacteria if an appropriate balance is achieved. The trend in recently introduced wound therapies is towards ever more sophisticated modes of action and in order to identify the correct treatment the appropriate therapy has to be matched to the identified barrier to healing.

DECISION-MAKING

Decision making is a complex process that is compounded as the range of choice increases. It is assumed that appropriate decisions can be made because they are an integral part of modern existence and generally a certain latitude with respect to incorrect decisions can be tolerated. In the clinical setting a wrong decision may be catastrophic with respect to healing outcome and effect on patient quality of life. Development of critical thinking faculties are essential to decisionmaking although the existence of a framework such as TIME may be of assistance in achieving an appropriate end-point. However TIME introduces a large number of options. Based on assessing the clinical presentation of the wound and the possible clinical actions, for instance whether to debride, what sort of debridement, whether to treat with antibiotics, which antibiotics etc, one may have to choose from 48 possible actions. To compound this complexity there are further decisions to be made about particular interventions and products.

ATTITUDES

Courses on wound healing have focussed on dealing with different wound types whereas with WBP we shall be moving away from thinking of individual wound types to focus on chronic healing. The biochemists have demonstrated the similarities between defects in the different types of wound and the focus may need to shift away from the differential between the diabetic, pressure or leg ulcer towards common therapies. Thus chronic wound healing has to be considered as a single entity. Such thinking is driven by identification of the biological and molecular factors affecting healing and inherently this may generate more interest in wound healing from the medical profession.

CONCLUSION

The systematic approach defined by WBP requires an experienced and skilled practitioner with a good knowledge of the underlying physiological processes and a growing requirement for knowledge of wound assessment techniques. The theory supporting WBP is based on sound scientific principles developed by researchers who by definition take a reductionist approach to the subject under investigation. This concentration on the wound with a focus on its molecular balance appears to return to a medical model of care even though an acknowledgement is made that the wound is one component of the patient. Possibly a focus on the wound and its treatment is a return to task rather than process because it is simpler to concentrate on it in a reductionist way. It is possible that the thought processes of WBP are subtly moving along the reductionist trajectory.

Wound bed preparation, chronic wound, education, treatment

TABLE 1SOME BIOLOGICAL IMPLICATIONS OF WOUND BED PREPARATION

Wound appearance	Cell Biology-Cause	WBP	Cell Biology-Effect
Necrotic tissue	Accumulation of cell debris	Debridement	 Infection focus Chronic Inflammatory stimulus
Infection	High bacterial load	Antimicrobial	Bacterial balance
Chronic Inflammation		Protease inhibitors	Resolution of inflammation
	Growth factor activity	Bioactive therapies	Growth factor balance
	Maceration	Moisture management therapy	Moist wound environment
Exudate	Keratinocyte migration		

Adapted from Schultz, 2003

Wound healing knowledge has developed from models of acute wounds followed by investigation of individual chronic wound types to the current concept of WBP which defines the chronic wound environment arising from differing aetiologies. This is a developing paradigm but probably not a paradigm shift although its implementation generates a major educational challenge. It blends scientific and clinical knowledge in a way that is new to wound healing. This raises the possibility of a new specialism; possibly called "woundology" that will integrate wound assessment, wound physiology and treatment technologies. Any such specialism will have to take care that a focus on the wound bed will still be considered in the holistic context of the patient.

REFERENCES

Baharestani M (1999) The clinical relevance of debridement. In: baharestani M, Gottrup F, Holstein P, Vanscheidt W, eds. The clinical relevance of debridement, Springer-Verlag, Heidelberg, 23-80

Brantigan CO (1996) The history of the understanding of growth factors in wound healing. Wounds 8: 78-90

Cherry GW, Hughes MA, Leaper DJ, Ferguson MWJ (2001) Wound Healing. In, Morris PJ, Wood WC, eds. Oxford Text Book of Surgery, 2nd Edition, Oxford University Press, 129 – 59

Dealey C (1994) The care of wounds. Blackwell, Oxford: 83-121

Falanga V (2000) Classifications for wound bed preparation and stimulation of chronic wounds. Wound Rep Reg 8: 347-52

Falanga V. (2002) Wound bed preparation and the role of enzymes: a case for multiple actions of therapeutic agents. Wounds 14: 47-57

Franks PJ, Moffatt CJ, Connolly M, et al (1995) Factors associated with healing leg ulceration with high compression. Age Ageing 24: 407-10

Fray MJ, Dickinson RP, Huggins JP, Occleston NL (2003) A Potent, Selective Inhibitor of Matrix Metalloproteinase-3 for the Topical Treatment of Chronic Dermal Ulcers. J Med Chem 46: 3514-3525 Jaschke E, Zabernigg A, Gattringer C (1999) Recombinant human granulocyte-macrophage colony-stimulating factor applied locally in low doses enhances healing and prevents recurrence of chronic venous ulcers. Int J Dermatol 38: 380-6 Kuhn TS (1962) The structure of scientific revolutions. University of Chicago Press, Chicago

Mulder GD, Brazinsky BA, Harding KG, Agren MS (1998) Factors influencing wound healing. In: Leaper DJ, Harding KG . Wounds biology and measurement.Oxford University Press, 52-70

Nagai MK Embil JK (2002) Becaplermin: recombinant platelet derived growth factor, a new treatment for healing diabetic foot ulcers. Expert Opin Biol Ther 2: 211-8

Porter, R (2002) Blood and Guts, A short history of medicine. Penguin Books, London

Roberts, C. and J. Mansbridge (2002). The scientific basis and differentiating features of Dermagraft. Can J Plast surg 10 Suppl A: 6A-13A

Robson MC, Mustoe TA, Hunt TK (1998) The future of recombinant growth factors in wound healing. Am J Surg 176: 80S-82S

Robson MC, Mannari RJ, Smith PD, Payne WG (1999) Maintenance of wound bacterial balance. American Journal of Surgery 178: 399-402

Schultz GS, Sibbald RG, Falanga V, et al (2003) Wound bed preparation: a systematic approach to wound management. Wound Rep Reg 11(Suppl 1): S1-S28

Sibbald RG, Orsted H, Schultz GS, Coutts P, Keast D (2003) Preparing the wound bed 2003: focus on infection and inflammation. Ostomy Wound Manage 49: 23-51

Trengrove N, Stacey MC, McGechie DF, Stingemore NF, Mata S (1996). Qualitative bacteriology and leg ulcer healing. J Wound Care 5: 277-80

Trengove NJ, Stacey MC, MacAuley S, et al (1999) Analysis of the acute and chronic wound environments: the role of proteases and their inhibitors. Wound Rep Regen 7: 442-52

Yager DR, Nwomeh BC (1999) The proteolytic environment of chronic wounds. Wound Rep Reg 1999 7:433-41

The Wound Healing Continuum, An Aid To Clinical Decision Making And Clinical Audit

Ince wound management began to develop as a specialty there have been numerous attempts to categorise or grade different wounds. To date none of these methods have been able to cover all wounds healing by secondary intention. All of these systems have been focused on the same objective facilitating the management of the patient. This is true of all the systems whether designed for the novice or expert. Some of the classification and grading systems have used a basic colour coding approach (Cuzzell, 1988; Stotts, 1990; Krasner, 1995; Lorentzen et al, 1999). All of these systems have utilized colour as their basis, a spectrum of colours -black, yellow and red - to equate with necrotic, sloughy and granulation tissue.

It has been suggested that this colour system facilitates the phase of the healing a wound is in, and, as a consequence, broad guidelines on management. For example, a yellow 'sloughy' wound requires debridement (Krasner, 1995). It was recognized by the author that the limitations of this generalization were observed in two types of yellow wound - the sloughy and the infected/pusfilled. Red can also be too general a term if used without qualification; granulating, healthy wounds are red as are slough-free wounds colonized or infected with beta- haemolytic streptococci. In the case of wounds which are critically colonised the term red maybe used, however the presence of red tissue alone does not indicate that the wound is actively healing [White et al 2003]. One author reported 'moderate' inter-observer correlation using the three colour system, however its use seems to have fallen out of favour with many in the field of wound healing (Lorentzen, 1999). The deficiencies of the three-colour system have been identified by various authors and it has been described as 'an over-simplification' (Gray White and Cooper, 2001). Its failure to identify the presence of unhealthy yellow or unhealthy red tissue at a time when there is a growing awareness of the impact of wound bio burden on delayed healing was also recognized by [Gray White and Cooper].

Abstract

Attempts have been made in the past to use colour as part of the wound assessment process. These methods have been useful but have failed to recoanise the transient nature of the wound and where there maybe more than one colour present in the wound. The presence of unhealthy red tissue can also be ignored using traditional colour methods. The Wound Healing Continuum encourages users to think in terms of a progressions from the left of the continuum, black, rightwards to the end of the continuum, pink. It is recommended that users of this continuum recognise that the colour closest to the left of the continuum should be used to define the wound e.a. in a wound which contains black yellow and red tissue the wound could be categorised as a black wound or if there are approximately equal amounts of black and vellow tissue a black /vellow wound. The Wound Healina Continuum allows the categorisation of wound tissue based on clinical importance using colour as the clinical marker. This continuum is an effective audit tool and an aid to clinical decision making, it does not however replace sound clinical judgment.

WOUND HEALING CONTINUUM

Kingsley (2001) devised a wound infection continuum which sought to facilitate understanding of wound bio burden. This, too, is a framework for clinical practice. It relies on an understanding of some commonly-used terms and a spectrum which extends from the sterile to the infected wound. This continuum has been further clarified (White et al, 2002) and its most recent update is contained within a complementary article in this supplement (Kingsley, White and Gray 2004). The wound healing continuum (WHC) that has been developed [Gray, White and Cooper 2003] relies on the identification of the colours present in any given wound and applying the most clinically significant to the spectrum which extends from black to pink with intermediate gradations (Figure 1). This use of colour is an attempt to address the restrictions identified by previous authors of the three colour system. The overriding principle which dictates the most clinically significant colour is the need to address that component to permit wound healing. In a wound that contains any black eschar, the primary

David Gray Clinical Nurse Specialist Department of Tissue Viability Grampian Acute Health Services Aberdeen

Richard White Senior Research Fellow Department of Tissue Viability Grampian Acute Health Services Aberdeen

Pam Cooper Clinical Nurse Specialist Department of Tissue Viability Grampian Acute Health Services Aberdeen

Andrew Kingsley Clinical Nurse Specialist [Infection Control and Tissue Viability] North Devon District Hospital Barnstaple

Wound Healing Continuum

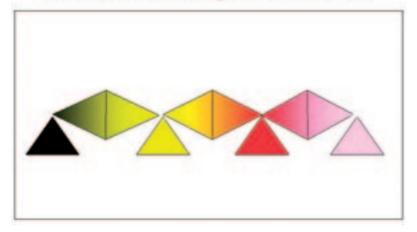


Figure 1. Wound Healing Continuum



Figure 2.



Figure 3.



Figure 4.

requirement is to debride before healing can proceed - without this intervention, there will be no healing. The WHC allows the majority of wounds that heal by secondary intention to be included. There will always be exceptions to any rule and the authors recognise that there are exceptions to the WHC, such the management of patients with peripheral vascular disease who develop as necrotic wounds. It maybe inappropriate to attempt to rehydrate such tissue. When the WHC is used in conjunction with the Wound Infection Continuum the concerns raised by Gray, White and Cooper 2001 regarding unhealthy tissue types are dealt with. The WHC allows the identification of red tissue in the wound bed and the WIC encourages the practitioner to consider if the red tissue can be considered as healthy or unhealthy.

USING THE WOUND HEALING CONTINUUM

Identify the colour that is furthest to the left of the continuum, for example if the wound contains yellow slough and red granulating tissue the wound would be defined as a yellow/red wound, a key objective of the wound management plan would be to remove the yellow tissue and promote growth of red granulating tissue. The management plan should focus on removal of the yellow sloughy tissue and promotion of the red granulation tissue. As this objective is achieved so the patient can progress along the wound healing continuum towards the right and pink /healing status. Not all wounds will naturally progress in this manner and when first assessed by a practitioner the wound may have been present for some time and not contain any black tissue. In general, it is most important to identify the colour that is furthest to the left of the continuum, then to implement management procedures to eradicate that colour, enabling the wound to progress steadily to the right. When used in this manner the WHC facilitates clinical decision making by encouraging the user to recognise the type of tissue which requires to be removed to facilitate progress along the WHC also facilitates continuum. The communication regarding the wounds status allowing terms such as a yellow red wound to replace descriptions such as sloughy. When used as a clinical audit tool the WHC definitions allow for easy recognition and classification thus facilitating clinical audits.

THE BLACK WOUND

Black has been termed the 'unhealthiest' colour and represents the beginning of the WHC (Stotts, 1990). Necrotic tissue which is dehydrated presents as a hard scab and has connotations of gangrene and of malignant melanoma. It should be recognised that a black covering on a wound may be very superficial and healthy tissue maybe found below such tissue. Heel pressure ulcers can present as a hard black necrotic area which may, on removal reveal a deep cavity filled with yellow slough or a granulating wound. When reviewing a pressure ulcer, it is also good practice to described it in terms of the local pressure ulcer grading tool (Russell, 2002). Debridement is required before the wound can heal(Torrance, 1983), however care should be exercised regarding the appropriateness of debriding a wound without first considering the patients overall condition . Following debridement the wound maybe re classified as a Yellow wound or a yellow red wound depending on the degree of slough present on the wound bed.

THE BLACK-YELLOW WOUND

Most wounds will not move directly from Black to Yellow as this process requires the re-hydration of eschar and the softening of the black tissue. This process is gradual as can be seen in Figure2 . Such a wound presenting with these two colours may be deteriorating to the left of the continuum, or improving as eschar is removed it is vital that this distinction between the two is made. This can best be done by identifying the condition of the wound previously, thus the use of the WHC can facilitate such communication

THE YELLOW WOUND

For any wound classified as a yellow wound should be assessed to exclude the possibility of pus and therefore localised or spreading infection. Once this has been considered and dismissed as a possible reason for the yellow colour, then the yellow can be attributed to slough (Tong, 1999) (Figure 3). Slough is usually yellow but may also be white; it serves as a medium for the growth of bacteria and must, therefore, be removed (Tong, 1999).

THE YELLOW-RED WOUND

The Yellow Red state is characterised by a wound with roughly 50% of each type of tissue present. Again care should be taken to assess whether the wound is moving in a positive manner to the right of the continuum or if it is deteriorating and moving to the left. Such a deterioration may indicate an increase in the wound bioburden. In this case the red component is probably due to granulation tissue, however, not all red is positive some may be attributable to colonization or infection with a haemolytic bacterium such as betahaemolytic Streptococcus species (groups A, B, C and G; Schraibman, 1990). These organisms are pathogens seen in leg ulcers and burns/plastic surgery. Red may also be attributable to the presence of frank blood, possibly from friable granulation tissue (an infection criterion), or trauma. Once these factors have been eliminated as causes, then granulation tissue remains as the most likely explanation.

THE RED WOUND

As stated by Gray, White and Cooper [2003] and in the previous section of this article not all red tissue can be classified as healthy. Only once all other potential causes of the red tissue are excluded is it reasonable to describe the wound as being filled with healthy granulation tissue (Figure 4). Care should be taken be recognise that red tissue may develop unhealthy characteristics such as critical colonization (Kingsley, 2001). Critical colonization of previously healthy granular tissue will result in it failing to heal and possible deterioration in the wound bed [Figure 5].

THE RED-PINK WOUND

By this point in the WHC the wound has almost finished the process of filling and covering the original defect in. Figure 6 shows a wound virtually covered the granulation tissue with fresh epithelium. The provision of a moist environment is likely to ensure the epithelialisation process can be completed.

THE PINK 'WOUND'

The original wound has ceased to exist as an open wound following the growth of new epithelium (Figure 7). However, this wound will continue to heal and remodel under the epithelium and may require protection until the tissues have consolidated. In darkly pigmented skins the colour of the epithelium will be tonally relevant to normal skin but like scar tissue in lighter skins will be different to surrounding undamaged epidermis.

MULTIPLE COLOUR WOUNDS

Some wounds will present with a variety of different coloured tissue. Figure 8 shows such an example with every colour from the WHC present. However by focusing management objectives on the removal of the tissue with the colour closest to the left hand side of the continuum – in this case black tissue – the wound can be moved along the continuum towards the right/pink. As seen in Figure 9 within 7 days the wound has moved to the yellow/ red category.

DISCUSSION

The authors have attempted to remedy the limitations of previous colour based systems and the resulting Wound Healing Continuum can also facilitate clinical decision making by highlighting the tissue most in need of removal. The framework allows the progress or deterioration of the wound to be charted using terms that are easily understood. This is an important feature as the WHC also lends itself to clinical audit. Clinical audits of the wound healing field in the UK are long overdue. There is a real need for those active in the field to undertake such audit projects so as to identify healing rates, prescribing patterns and to identify where resources might be best allocated.

CONCLUSIONS

In this paper the authors have sought to introduce the Wound Healing Continuum which when used in conjunction with Wound Infection and Wound Exudate Continuums can support practioners in identifying the optimum management for their patients. It is important that users recognise the need for a good working knowledge of wound healing and rely on their own clinical decision making skills and use the continuums as an aid.



Figure 5.



Figure 6.



Figure 7.



Figure 8.



Figure 9.

REFERENCES

Cuzzell JZ (1988) The new red, yellow, black color code. Am J Nurs 88(10): 1342–46

Gray.D, White R,J, Cooper P, The Wound Healing Continuum, In R.J. White [ed] The Silver Books Division, MA Healthcare Ltd, Dinton Wiltshire

Kingsley A (2001) A proactive approach to wound infection. Nurs Stand 11(15): 50–8

Kingsley A, White R, Gray D The Wound Infection Continuum : A Revised Perspective, Applied Wound Management Supplement, Vol 1 No 1 Wounds-UK

Krasner D (1995) Wound care: how to use the redyellow- black system. Am J Nurs 95(5): 44–7

Lorentzen HF, Holstein P, Gottrup F (1999) Interobservatorvariation ved rodt-gultsortsarbeskrivelsessystemet. Ugeskr Laeger 161(44): 6045–8

Russell L (2002) Pressure ulcer classification: the systems and the pitfalls.

Schraibman IG (1990) The significance of bhaemolytic streptococci in chronic leg ulcers.

Stotts NA (1990) Seeing red and yellow and black. The three-color concept of wound care. Nursing 20(2): 59–61

Tong A (1999) The identification and treatment of slough. J Wound Care 8(7): 338–9

Torrance C (1983) Pressure Sores. Aetiology,Treatment and Prevention. Croom Helm, London and Canberra

Villavicencio RT (1998) The history of blue pus.

White RJ (2002) The wound infection continuum. BrJ Nurs 11(22 Suppl): 7–9

White RJ, Cooper R, Kingsley A (2002) A topical issue: the use of antibacterials in wound pathogen control. In: White RJ, ed. Trends in Wound Care. Quay Books, Salisbury

The Wound Infection Continuum: a revised perspective.

Wound Infection Continuum



Figure 1. Wound Infection Continuum

WOUND BIOBURDEN – A FACTOR FOR CHRONICITY

any wounds healing by secondary intention become indolent causing extended periods of discomfort and inconvenience for the patient, rise in healthcare costs and increase in workload for staff. A common cause for this is the effect of the wound bioburden (Browne, Dow, Sibbald 2001). This indolence is due to either invasive infection, the quantity or mixture of microbes present, or, the effect of their toxins. The indiscriminate use of antibiotics in all open wounds would raise healthcare costs and contribute to the development and selection of multi-resistant micro-organisms. Therefore systemic antibiotics are reserved for proven cases of spreading wound infection. However diagnosis of infection is restricted to the use of clinical signs and symptoms with qualitative microbiology providing information for the checking of antibiotic prescriptions initiated on clinical infection recognition to the organisms and resistance patterns they possess. The use of microbiology (qualitative, semi-quantitative or quantitative) alone is flawed because results require interpretation based on the prevailing wisdom of the relative importance of bacteria and/or quantities of bacteria in any particular context on the body. As infection is a clinical diagnosis there remains large scope for either over or under-treatment dependent on the diagnostic skills of the clinician. Based on the results of audit it is seen that nurses (Kingsley and Winfield-Davies 2003) and other clinicians

Abstract

With increasing concerns regarding the inappropriate use of systemic antibiotics in all branches of medicine, those involved in wound care are duty bound to use alternative treatments to the best clinical effect. These alternatives include the wide variety of antimicrobial wound dressings. Despite much clinical research into the management of wound infection, we are still largely involved in treating infections rather than pro-actively treating to avoid infection. This latter objective may be described as the best approach in terms of reduced morbidity and costs, and for patient quality of life. How then may this be achieved? A framework is required for the early recognition of factors that might lead to infection. An awareness of increasing wound bioburden, of colonisation with specific pathogens, and, recognition of clinical signs and symptoms that herald incipient infection is essential for success. These factors are included in the evolving Wound Infection Continuum and the related treatment guidelines.

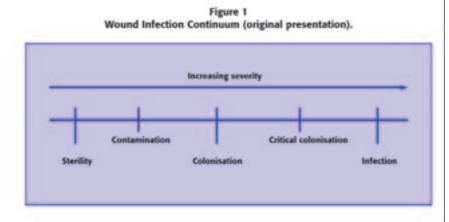
(Bamberg et al 2002) vary in their ability to diagnose. Wounds that do not exhibit the classical signs of infection may become indolent through the effects of bioburden, and though they might benefit from an antimicrobial strategy, either topical or systemic, often go untreated. To improve clinicians' ability to make a clinical diagnosis requires a consolidation of current wisdom surrounding microbiology and clinical signs and symptoms into an easily taught and understood conceptual package. As is often the case in the infection control and tissue viability arenas evidenced by the 'EPIC guidelines' (Pratt et al 2001), and by NICE guidelines (NICE 2001; NICE 2003), hard facts from high quality clinical research are often not available to guide practice decisions. It could be argued that this is true of wound infection with no clear consensus of what constitutes the clinical signs of infection, though work on this is underway (Cutting and White 2003, 2004) in an international Delphi study. Thus it is necessary to extrapolate the principles of microbiological growth, transmission and pathological potential developed in the laboratory to the clinical setting. The concept for building a bridge between microbiological theory and clinical practice is called the wound infection continuum. This continuum seeks in highly simplified form to marry-up and describe clinical states with the probable bacterial bioburden back-drop, enabling

Andrew Kingsley CNS Tissue Viability North Devon District Hospital Barnstaple EX31 4JB

Richard White Senior Research Fellow Department of Tissue Viability Grampian Acute Health Services Aberdeen

David Gray Clinical Nurse Specialist Department of Tissue Viability Grampian Acute Health Services Aberdeen

APPLIED WOUND MANAGEMENT SUPPLEMENT



the practitioner to interpret what is happening and encourage suitable action when it is needed. In particular it is possible using the wound infection continuum to suggest when it is appropriate to use the plethora of new antimicrobial therapies now available. This makes for good 'prescribing' in clinical and cost effectiveness terms.

In clinical practice, the main focus is on reducing high levels of organisms that are causing problems. Trying to maintain a low level through chemoprophylaxis is tempting for the benefits if it could be achieved without toxicity to healing cells, selection for bacterial resistance, or elevating costs. The issue of the quantity of micro-organisms has two major aspects, firstly the sheer weight of numbers present and secondly the number of different species. The so-called magic number of 105 (or 100,000) organisms per millilitre of exudate or gram of tissue as a threshold for infection works reasonably well in the closed surgical wound as these tend to behave predictably. The open wound can often tolerate much more without showing signs of deterioration, but the response is dependant on a number of factors, predominantly the adequacy of the host immune response, and potentiating factors such as foreign bodies that reduce the quantitative threshold of infection. Trengove et al (1996) have provided evidence to support the notion that the effect of multiple species, four or more, is to produce delay to healing. In general fewer species and less numbers are better for normal healing progress. Simplistically the outcome both positive and negative from wound organisms is a story of quantity and can be illustrated as the wound infection continuum.

WHAT IS THE WOUND INFECTION CONTINUUM?

The wound infection continuum is a conceptual representation of the varying levels of bioburden in the wound (Kingsley 2001; White 2003). This 'spectrum' is essentially based on the quantity of micro-organisms present but actual quantification is, as yet, not defined. Using the Wound Infection Continuum as a sliding scale against which to judge the level of wound bioburden can facilitate clinical decision making. Quantification may prove difficult as clinical outcomes rely on the ability of the host to mount an immune response to the presence of micro-organisms, and this will be different for each individual. Progression along the continuum in the direction of increasing clinical severity denotes increasing bioburden, becoming clinically relevant for chronicity only once the state of colonisation has been passed. The most controversial point on the continuum is that of critical colonisation, a state of delayed healing. This is now being scientifically rationalised for its existence from studies on the effect of anaerobes penetrating the wound bed and releasing agents that interfere with the normal cellular processes of healing (Stephens et al 2003).

WOUND INFECTION CONTINUUM

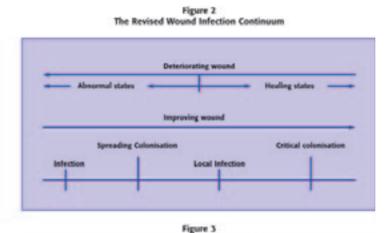
The quantity and diversity of microbes representing the states of colonisation, critical colonisation local and spreading infection are individual and dependent on the quality of the host immune response. Some wounds progress quickly from colonisation to infection via a clinically indistinct 'critical colonisation' state. Other wounds stop at this point and become indolent (Heggers et al 1992; White, Cooper, Kingsley 2002; Cutting and White 2003 and 2004). Critically colonised wounds will become increasingly 'chronic' or indolent because cellular cascades are disordered and biochemical imbalance arising from bacterial metabolism (Sorkin and Niederman 1998; Wall et al 2002). Once this is established, wounds in this state can prove resistant to adjustment with current therapy, as well as emerging novel therapies such as protease inhibitors, extracellular matrix components and topical growth factors. Thus, early recognition of disordered healing caused commonly either wholly or partly by microbes is vital to achieving good outcomes.

COLONISATION, CRITICAL COLONISATION LOCAL AND SPREADING INFECTION

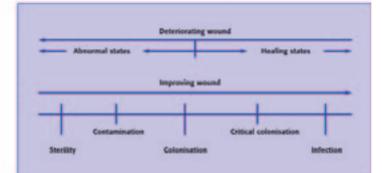
The continuum to date has been drawn to show increasing severity of clinical and microbiological states from left to right. However it can be reversed to link with other continuums such as the wound healing continuum (Gray et al 2003) to promote ease of assessment and documentation of progress in clinical practice. In the original infection continuum (Kingsley 2001), the states of sterility and contamination were included to reflect the presence of microbial growth from the outset of wounding. Sterility represents the absence of any organism in the wound and is a very unusual situation. It might exist at the moment of, and shortly after, thermal injury, though depending on the depth of the burn even this may not be sterile as organisms are present in the deep dermis in the hair follicles. It is unlikely that even in clean category surgery with good skin antisepsis that the surgical wound at the time of incision is completely devoid of microbes carried in from the skin. Absolute sterility and a swab sample result of 'no growth' are not synonymous, the latter just means nothing would grow to produce a visible colony in standard laboratory conditions. For the purpose of clinical practice understanding this state can be ignored. Similarly contamination, which means presence of organisms with no active growth and not accompanied by a visible host response, is of no relevance to clinical practice in the wound healing secondary intention. The normal bv microbiological state of a healing wound is that of colonisation which represents growth and death of organisms being kept in a healthy balance or below the healing disruption threshold by the immune system (Heinzelmann et al 2002). In the wound healing by secondary intention, this situation of microbial/host equilibrium is likely to be reached early on in the life of the wound and any original inflammation from the wounding event may have faded away leaving no obvious visible host response in the surrounding undamaged tissues. In contrast, infection represents the microbial population in ascendancy and overrunning the immune response mounted in the wound bed itself. This results in an additional inflammatory response, seen as fresh inflammation or an extension of the red border in the skin surrounding the wound. In some wounds a pre-existing inflammatory ring may be present' though at the time of infection only some of it may be painful to pressure - indicating a change to the host response and an exacerbation of microbial invasion. Inflammation can result from causes other than microbial invasion, such as eczema, contact dermatitis and contact with exudates. Whilst these wounds may be described as sore by the patient, they are often no more painful when pressed with a gloved finger - unlike infected wounds which almost always are more painful. The exception to this is in the insensate wound, such as foot ulcers with a sensory neuropathic aetiology in diabetics, alcoholics and leprosy cases, or various wounds in the spinally injured patient. In the event of the local immune response being inadequate, systemic responses may be seen. Some authors differentiate infection into 'local' and systemic infection (Sibbald et al 2000) which has been used to provide guidance on route of administration for systemic antibiotics. Dow et al (1999) and Schultz et al (2003) utilise, with other presentational factors, a ring of cellulitis <2cm to suggest antibiotic treatment via oral route with extensive cellulitis (by absence of further definition presumed to be >2cm) requiring intravenous therapy. Choice of route of antibiotic delivery is about the time taken to achieve a therapeutic level of antibiotics at the site, intravenous delivery naturally being faster than oral administration. Intravenous therapy is indicated in the more severe case where the consequences of systemic infection can be grave. It seems logical to relate the extent of the inflammation with the severity of infection and provide guidance for intervention, but to our knowledge the choice of the 2cm threshold and therapeutic response are not yet validated by published research. The use of the word 'systemic' for infection that has spread beyond the 2cm from the wound edge could also be unintentionally misleading. Some wounds have wider zones of peripheral redness but remain local in character, meaning that the inflammation zone does not continue to extend and the patient does not exhibit systemic infection signs produced by the consequences of bacteraemia notably fever, rigor, and positive blood culture. Therefore, for the purposes of re-considering the states to depict on the wound infection continuum, the terms 'local' and 'spreading' infection will be used denoted by the 2cm threshold as previously discussed in the literature. The remaining state lies between colonisation and infection and is called critical colonisation (Davis 1998), a state which has also been referred to as covert infection (Dow et al 1999; Dow 2001), and localized infection (Gardner et al 2001) or could equally be described as 'sub-clinical' infection. These terms are used to identify different states of microbiological activity.

The change from one state to another depends on

APPLIED WOUND MANAGEMENT SUPPLEMENT







many factors:

- the virulence of the initial inoculum of microorganisms,
- continuing additions to the species variation,
 quantity of bioburden acquired from exogenous sources (such as the air) and endogenous sources (i.e. from other body sites such as the bowel);
- potentiating factors in the wound such as haematoma.
- the host immune response (Cooper and Lawrence 1996a, 1996b).

Each successive stage from right to left on the continuum (see figure 2) involves an increase in the quantity of microbes, a new pathogen arrival, an increase in the quantity of virulent organisms, or, an increase in the virulence (Wilson 2002) of the collective species mixture through bacterial synergy (Kelly 1980; Bowler et al 2001). The situation may shift in favour of the micro-organisms if the host immune response is impaired or suddenly reduced (Heinzelmann et al 2002; Bowler 2002). In addition shift may result from the presence of potentiating factors such as the introduction of foreign bodies that reduce the necessary inoculum needed to produce a worsening microbiological environment. The revised continuum can be expanded to include the clinical signs and symptoms that define each stage, and provide guidance on anti-microbial interventions, see figure 3

CONCLUSIONS

The predominant aim of wound care is about the removal of negatives or barriers to healing. These negative influences include the cause of wounding (such as pressure or venous hypertension), necrotic debris, excess exudates, high bioburden, high blood sugar, contamination and excoriation from incontinence, pain, anxiety, excess proteases, nutritional deficits, and so on. We are moving into an era of therapy where positive factors that will speed healing beyond its normal maximal rate are available e.g. growth factors, extracellular matrix components, living skin equivalents and bioabsorbable collagen scaffolds. To ensure good clinical results and cost effectiveness with these techniques, the effects of high bioburden will continue to be a treatment objective. Treatment actions must conform to sound principles, in particular rapid debridement, systemic antibiotics for spreading infection (perhaps in tandem with selected topical antiseptics), and the use of certain topical antiseptics in safe formulations for locally infected and critically colonised wounds.

Good wound care outcomes rely on practitioners who know what to expect of a wound in terms of the stages, tissue types and timing of normal healing, observe the wound closely and interpret what they see correctly, and are empowered to initiate prompt treatment when things stop healing. Putting knowledge into practice will achieve early resolution of wounds that would otherwise all too easily become complex and stop healing.

REFERENCES

Bamberg R, Sullivan P, Conner-Kerr T. (2002) Diagnosis of wound infections: current culturing practices of US wound care professionals. Wounds. 14:9; 314 – 327

Bowler P G (2002). Wound pathophysiology, infection and therapeutic options. Ann Med 34; 419-27.

Bowler P, Duerden B, Armstrong, D. (2001). Wound microbiology and associated approaches to wound management. Clin Microbiol Reviews. 14: 2; 244 - 269

Browne A., Dow G, Sibbald R.G (2001). Infected wounds: definitions and controversies. In: V. Falanga (Ed.) Cutaneous Wound Healing. London: Martin Dunitz.

	Spreading infection	Local Infection	Critically Colonised	Colonised
Key local characteristic	>2cm redness with pain (unless insensate).	2cm or less redness with pain. Sudden necrosis on wound bod (red inflammatory zone may not be present).	Static (despite appropriate therapy). No cellulitis.	Expected progression towards healing. No cellulitis (but may be small degree of inflammation in early stage consistent with inflammator phase – generally not more painful to pressure than background wound pain).
Other local characteristic	Heat. Swelling.	Heat and swelling (can be difficult to identify in small red inflammatory zone).		
Additional local characteristics that may be present in addition to key ones	Extension to main wound at skin level. Bistering (fluid filled). New satellite wounds in red inflammatory zone. Increased wetness. Haemonhagic patching or spotting in surrounding skin. Purulent exudates*. Maceration if control of exudates is inadequate. Extensive necrotic and/or sloughing necrotic tissue.	Extension to main wound at skin level. Extension to wound at its base (pocketing). Increased wetness Purulent exudates*. Maceration if control of exudates is inadequate. Extensive necrotic and/or sloughing necrotic tissue. Discolouration of granulation tissue (darkening). Friable bleeding granulation tissue (possibly with very bright red tissue) Foul odour	Thick slough not responding to standard debridement techniques. Fast returning thick slough after sharp or maggot debridement. Wet wound. Purulent exudates. Maceration if control of exudates is inadequate. Blue/green exudate (Pseudomonas aeruginosa). Foul odout. Discolouration of granulation tissue (darkening). Friable bleeding granulation tissue (possibly with very bright red tissue).	Debride damaged tissue under standard therapeut approaches. Gently moist wound surfac Slough but light and mobile in consistency. Inflammation from initial wounding consistent with expectation for inflammatic phase of wound healing but fading away or gone is wound older. Granulation tissue of healthy red colour. Epithelial tissue with colo different from but relevan to normal skin tone. Reducing wound size in last 1 – 2 weeks.
Possible systemic features	Neutrophilla Rising C-reactive protein Fever. Rigors. Confusion (in the elderly). Bacteraemia. Tachycardia. Tachycardia. Tachypnoea. Lymphangiltis. Lymphadenitis.	Neutrophilia Rising C-reactive protein.	None	None
Suggested treatment	Systemic antibiotics – oral if red zone static and still localised even though >2cm – IV if red zone more than obvious local ring around wound or if actively spreading. Use local formulary. Consider topical antiseptic dressings – at this stage medicated dressings may not be cost or clinically effective, though it is clinically reasonable to use them in the diabetic foot ulcet, critically ischaemic wounds, burms and the severely immuno- compromised patient.	Systemic antibiotics – oral. Use local formulary. Topical antiseptic dressings – normally iodine or silver in formulation or combination suitable for wet wound. Locally infected wounds with a necrotic eschar will need a wetter formulation. Adjunctive measures – rapid debridement of necrotic tissue may be necessary consider relevant strategy e.g. sharp or surgical.	Topical antiseptic dressings – normally iodine or silver in formulation or combination suitable for wet wound. Slow release formulations are preferred. Medical grade Manuka honey may be considered especially to control foul odour. Use local formulary. Consider topical antiseptic irrigation – some authors suggest use of dilute vinegar to control Pseudomonas il blue/green exudate is present. Adjunctive measures – debridement of necrotic tissue may be necessary consider relevant strategy e.g. maggots. Use of anti- protease therapy may be valuable with antimicrobials.	Standard wound therapy and control of underlying aetiological factors (e.g. venous hypertension, forces of pressure) as loca guidelines. Topical antiseptic dressing – Normally no antimicrobials are necessary, however prophylaxis may be considered in vulnerable wound groups such as diabetic foot ulcer or vulnerable immuno- suppressed patients. It can also be considered if the patient has a recurrent history of infection in this wound.

Cooper R A, Lawrence J C (1996a). Microorganisms and wounds. J Wound Care 5:5; 233-36.

Cooper R A, Lawrence J C (1996b) The prevalence of bacteria and implications for infection control. J Wound Care 5:6; 291-95.

Cutting K, Harding K.G. (1994). Criteria for identifying wound infection. J Wound Care 3:4; 198 – 201.

Cutting K.F, White R.J. (2003) Developing criteria to identify wound infection – the next step. Poster displayed at Wounds UK Harrogate November 2003.

Cutting K, White R J (2004). Defined and refined: criteria for identifying wound infection revisited. Brit J Comm Nurs 9:3; (Suppl)S6-15.

Davis E. (1998) Education, microbiology and chronic wounds. J Wound Care 7:6; 272 – 274.

Dow G. (2001) Infection in chronic wounds. In: D. Krasner, Rodeheaver G., Sibbald, R.G. (eds.) Chronic Wound Care: A clinical source book for healthcare professionals (3rd Edn.) HMP Communications; Wayne PA.

Dow G, Browne A., Sibbald R.G. (1999) Infection in chronic wounds: Controversies in diagnosis and treatment. Ostomy Wound Manag. 45:8; 23 – 40.

Gardner S, Frantz R, Doebbeling B. (2001) The validity of the clinical signs and symptoms used to identify localized chronic wound infection. Wound Rep Regen 9:3; 178 – 186.

Gray D, White R.J, Cooper P. (2003) The wound healing continuum. In: R.J. White (ed.) The Silver Book. Quay Books Division, MA Healthcare Ltd. Dinton, Wiltshire UK.

Heggers J P, Haydon S, Ko F, et al (1992). Pseudomonas aeruginosa exotoxin A: Its role in retardation of wound healing. J Burn Care Rehab 13; 512-18.

Hienzelmann M, Scott M, Lan T (2002). Factors predisposing to bacterial invasion and infection. Am J Surg 183; 179-90.

Kelly M J (1980) Wound infection: a controlled clinical and experimental demonstration of synergy between aerobic and anaerobic bacteria. Ann Roy Coll Surg Engl 62:1; 52-59.

Kingsley A. (2001) A proactive approach to wound infection. Nurs Stand 15:30; 50 – 58

Kingsley A, Winfield-Davies S. (2003) Audit of wound swab sampling: why protocols could improve practice. Prof Nurse; 18: 6; 338 – 343 Laato, M., Niinikoski, J., Lundber, C., Gerdin, B. (1988) Inflammatory reaction and blood flow in experimental wounds inoculated with Staphylococcus aureus. Eur. Surg. Res. 20: 33 - 38

NICE (2001) Guidance on the use of debriding agents and specialist wound care clinics for difficult to heal surgical wounds.

www.nice.org.uk/pdf/woundcareguidance.pdf

NICE (2003) Pressure ulcer prevention: Pressure ulcer risk assessment and prevention, including the use of pressure-relieving devices (beds, mattresses and overlays) for primary and secondary care. www.nice.org.uk/pdf/CG7_PRD_NICEguideline.pdf

Pratt R, Pellowe C, Loveday H. et al (2001) The epic project: developing national evidence based guidelines for preventing healthcare associated infections – phase 1: guidelines for preventing hospital - acquired infection. J Hosp Infect. 47 (suppl) 1 – 82

Schulz G, Sibbald R.G, Falanga V. et al. (2003) Wound bed preparation: a systematic approach to wound management. Wound Rep Regen. 11: 1 - 28.

Sibbald R. G., Williamson D, Orsted H, et al (2000) Preparing the wound – Debridement, bacterial balance, and moisture balance. Ostomy Wound Manag. 46: 14 – 35.

Sorkin B C, Niederman R (1998) Short chain carboxylic acids decrease keratinocyte proliferation and increase apoptosis and necrosis. J Clin Periodontol 25:4; 311-15.

Stephens P, Wall I B, Wilson M J et al. (2003). Anaerobic cocci populating the deep tissues of chronic wounds impair cellular wound healing responses in vitro. Brit J Dermatol. 148:3; 456-66.

Trengove N, Stacey M, McGechie D, Mata S. (1996) Qualitative bacteriology and leg ulcer healing. J Wound Care. 5: 6; 277 – 280.

Wall I B, Davies C E, Hill K E et al. (2002). Potential role of anaerobic cocci in impaired human wound healing. Wound Repair Regen. 10:6; 346-53.

White R. J., The wound infection continuum. (2003) In: R.J.White (ed.) The Silver Book. Quay Books, Dinton Wiltshire.

White R J, Cooper R A, Kingsley A (2002) A topical issue: the use of antibacterials in wound pathogen control. In: White R J (Ed) Trends in Wound Care. Quay Books, Dinton Salisbury UK.

Wilson J W, Schurr M J, Le Blanc C L, et al (2002) Mechanisms of bacterial pathogenicity. Postgrad Med J 78; 216-24.

The Wound Exudate Continuum: An Aid to Wound Assessment

ound healing occurs in four overlapping phases: haemostasis and inflammation, granulation epithelialisation, and tissue remodelling [Davidson 1992]. Upon injury vasoconstriction occurs with the aim of reducing blood loss; haemostasis is achieved by the formation of a clot, which seals the wound. Following haemostasis, the inflammatory process begins during which wound exudate is produced by the tissues surrounding the wound. Normal, serous exudate is essential to the healing of the wound. However, wound exudate is not always 'normal' in terms of volume and/or consistency; it can present significant management challenges and be a sign of underlying problems relating to the wound bio burden

[Cutting and Harding 1994; Gilchrist 1999; Vowden and Vowden 2003, 2004; Cutting 2004].

Where a wound is healing without complication, exudate can be considered a normal feature. It is produced when blood vessels dilate, post haemostasis, as part of the inflammatory process. Endothelial cells enlarge to allow the flow of fluid from the blood vessels – extravasation of serous fluid. The presence of this fluid in the tissues surrounding the wound contributes to localised pain, heat and swelling associated with inflammation.

Normal wound exudate is mainly comprised of three components, serous fluid from the leaking blood vessels, debris from local damaged tissue, and growth factors or cytokines (Chen et al 1992; Cutting 2004).

Wound Exudate has a key role to play in the moist wound healing process as it provides not only moisture, but also elements which support the removal of dead tissue and the formation of new tissue. Factors such as the underlying condition of the patient, dressing selection and the pathology of the wound affect the production of exudate [White 2001]. Wound exudate has been shown to contain different elements at different stages of healing, acute wounds containing growth factors and chronic wounds containing tissue degrading enzymes [White 2001; Cutting 2003].

Abstract

Wound exudate is a key component of healing in the healthy wound. Traditionally, practitioners have considered exudate in terms of its volume alone. This approach fails to recognise the potential impact of wound exudate viscosity. Not only can the viscosity of wound exudate impact upon the absorptive performance of the wound dressing, but, can also provide a valuable insight into the underlying health of the wound.

In this article, the authors present the Wound Exudate Continuum, an assessment tool intended for use as part of the Applied Wound Management framework. Through using Wound Exudate Continuum, it becomes possible to assess both the viscosity and volume of the wound exudate in terms of the health of the wound, and, to rank the combination in terms of clinical significance. When utilised along side the Wound Healing and Wound Infection Continuums, a systematic approach to wound assessment can be achieved

Exudate can also present significant management challenges; for example, in the case of venous leg ulcers and pressure ulcers, protease enzymes which can, if present in large volumes and available to the surrounding skin, result in the development of excoriation and maceration [Cameron and Powell 1992]. Large volumes of exudate can result in the saturation of the wound bed and peri-wound area resulting in maceration of both [Cutting 1999; White and Cutting 2003]. Wound exudate can also increase the risk of infection where it soaks through a dressing allowing bacteria 'strike - through' the wound dressing. Wound exudate can facilitate healing if it is managed so as to promote healing and avoid damage to surrounding skin (Jones and Harding 2001; Bishop et al 2003).

WOUND EXUDATE AND THE HEALING PROCESS

Exudate will be found in all wounds healing by secondary intention at some point during their existence. The volume and viscosity of wound exudate produced by the wound will be influenced by the stage of healing the wound is at and the presence or absence of factors such as infection. Wounds healing by secondary intention without complication will gradually redcue their production of exudate as the healing process progresses David Gray Clinical Nurse Specialist Department of Tissue Viability Grampian Acute Health Services Aberdeen

Richard White Senior Research Fellow Department of Tissue Viability Grampian Acute Health Services Aberdeen

ASSESSING EXUDATE.

Traditionally exudate has been described in terms of its perceived e.g. as light / low, moderate, or heavy [Watret 1997]. This form of assessment is very subjective and difficult to quantify in the absence of significant investigation such as the weighing of dressing pre- and post use. Such assessments can be variable between practioners and confusing [Thomas 1997].

Vowden and Vowden [2003a, 2004] suggest that exudate volume should not be viewed in isolation but in conjunction with the viscosity. By considering both of these aspects, we can gain an insight into the underlying condition of the wound and of the patient.

We suggest that wound exudate volume and viscosity be assessed by:

- considering the exudate which is retained within the dressing,
- the number of dressing changes required in 48 hours, and,
- visual inspection of the wound.

This approach to assessment is complimentary to current management strategies such as the six 'Cs' as proposed by Vowden and Vowden (2003).

Wound Exudate Continuum

The wound exudate continuum is offered as an aid to quantifying the volume and viscosity of wound exudate. The gradings [see fig 1] are 'high, medium and low' for both of these features, allowing wound exudate to be categorised by a numerical score e.g. a wound of low volume and of medium viscosity would be in the Low/Med category and would score 4 (placing it in the low exudate [green] portion of the continuum). Any score in the green zone should be seen as advantageous to wound healing. Should a score of 6 be awarded, then this would place the wound in the amber zone. Wounds which are assessed as being in the amber zone require careful consideration as this score could indicate and improvement or deterioration in the wounds condition. If the previous recording had been in the green zone then the practioner should seek to identify why the wound has moved (deteriorated) into the amber zone. A change in score to red to amber maybe due to an alteration in the wound bio burden, indicating critical colonisation or the development of an infection. However, if the previous score had been in the red zone, an amber score would indicate an improvement in the condition of the wound. Any score in the red zone should be investigated urgently as this may indicate local or spreading infection, particularly if the previous score had not been in this zone.

Using the Wound Exudate Continuum

When reviewing the wound, the exudate on the dressing and present in the wound should be assessed as presented above. Any wound assessed as having both high viscosity and high volume of wound exudate would score a full ten points and be regarded as causing serious concern. It is likely that such a wound may indicate a spreading infection, sinus or fistula formation or some other cause for concern. Any wound scoring 6 points would be regarded as requiring regular review, it maybe that this finding is entirely consistent with the treatment applied e.g. the liquefying of wound slough, the wound may have previously been scored in the red zone and as such a score of 6 would indicate an improvement. Where a wound had previously been scored as 2-4 points and is observed to be in the amber zone [scoring 6 points] this could be an early sign of critical colonisation or of the development of a wound infection and should be acted upon. The overriding aim of the wound exudate continuum is to encourage a systematic approach to wound care and to support clinical decisions making.

DISCUSSION

Wound exudate is, under normal circumstances, an integral part of the wound healing process and it can have a positive impact on the wound. However, where the production of exudate is excessive and/or badly managed, there can be adverse consequences for the patient e.g. wound maceration and dressing leakage. Traditionally wound exudate has been viewed largely as a management issue for wound dressings. However, as identified by Vowden and Vowden [2003a], by considering wound exudate in terms of its viscosity and volume a valuable insight into the wound's underlying condition can be obtained. The Wound Exudate Continuum is designed to provide the practioner with a method of assessment based on estimation of viscosity and volume of exudate, which relates to the underlying condition of the wound. Its use is intended as part of a thorough assessment and to be used within the Applied Wound Management framework. The values attached to the different levels of exudate are designed to provide the practioner with an aide to assessment and are not designed to replace sound clinical decision making.

CONCLUSION

Applied Wound Management relies on the assessment of three different aspects of the wound using three continuums. The Healing Continuum and the Infection Continuum are concerned with the type of tissue in the wound bed and the wound bio burden respectively. Both are complemented by the addition of the Wound Exudate Continuum which encourages the practioner to consider the wound exudate in relation to the wounds overall condition. It should be recognised that a systematic assessment of wound exudate can positively inform the decision making process and the Wound Exudate Continuum is an adjunct to this. However it is vital practioners recognise that these methods are intended to act as aids to sound clinical decision making not as a substitute.

REFERENCES

Bishop S M, Walker M, Rogers A A, Chen W Y J 2003. Importance of Moisture balance at the wound-dressing interface. J Wound Care 12:4; 125-128.

Cameron J, Powell S 1992 Contact dermatitis: its importance in leg ulcer patients. Wound Management.2: 3; 12-13.

Chen WY, Rogers AA, Lydon MJ. 1992. Characterization of Biologic Properties of Wound Fluid collected during early stages of Wound Healing. Journal of Investigative Dermatology 99:5; 559-564.

Cutting KF 1999 The causes and prevention of maceration of the skin. J Wound Care 8:4; 200-202.

Cutting K F 2004. Wound exudate. In: Trends in Wound Care Vol 3, Ed R J White, Quay Books, Dinton Salisbury UK.

Cutting KF, Harding KG. 1994 Criteria for identifying wound infection. J Wound Care 3:4; 198-201.

Davidson JM 1992 Wound Repair. Pp 809-819.In: inflammation: Basic Principles and Clinical Correlates. 2nd.Edition. Eds. Gallin JI, Goldstein IM, Snyderman R. Raven Press. New York.

Field C, Kerstein M 1994 Overview of wound healing in a moist environment. American Journal of Surgery 167(Suppl 1a); 25-30.

Gilchrist B. Wound Infection. In: Wound Management: Theory and Practice Eds. M Miller and D Glover. NT Books London 1999.

Lamke LO, Nilsson GE, Reithner HL. 1997. The evaporative water loss from burns and water permeability of grafts and artificial membranes used in the treatment of burns. Burns. 3: 159-165.

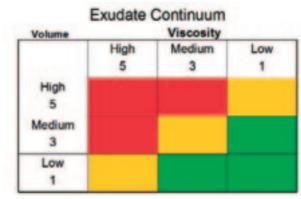


Figure 1. Exudate Continuum

Rogers AA, Burnett S, Moore JC, Shakespeare PG, Chen WY. 1995. Involvement of proteolytic enzymes - plasminogen activators - in the pathophysiology of pressure ulcers. Wound Repair and Regeneration 3:3; 273-283.

Thomas S 1997 Wound exudate-who needs it? Management of Wound Exudate Proceedings. Eds. G.Cherry & KG Harding. Churchill Communications London.

Thomas S, Fear M, Humphreys J, Disley L, Waring MJ. 1996 The effect of dressings on the production of exudate from leg ulcers. Wounds 8:5; 145-150.

Trengrove NJ, Langton SR, Stacey MC. 1996. Biochemical analysis of wound fluid from nonhealing and healing chronic leg ulcers. Wound Repair and Regeneration; 4: 234-239.

Vowden. K, Vowden P, [2003] Understanding exudate management and the role of exudate in the healing process. Brit J Nurs 12:20; Suppl. 4-14.

Vowden. K, Vowden P, [2004] The role of exudate in the healing process: understanding exudate management. K & P Vowden. Chapter 1 in: Trends in Wound Care vol 3, Ed RJ White, Quay Books, Dinton Salisbury UK

Watret L [1997] Know How: Management of Wound Exudate. Nursing Times 93:30 38-39

White R J, Cutting K F [2003]. Interventions to avoid maceration of the skin and wound bed. Brit J Nurs 12:20; 1186-1201.

White [2001] The Management of Exuding Wounds Part 1, Nursing Times 97:9; XI-XIII.

A Review of Different Wound Types and Their Principles Of Management

Abstract

Wounds can be categorised into many different groups and sub groups due to their wide and varied pathologies. However it is possible to categorise most wounds healing by secondary intention into the six following categories; Pressure Ulcers, Leg Ulcers, Diabetic Foot Ulcers, Trauma Wounds, Surgical Wounds and Complex Wounds. Before embarking on any plan of care it is vital that the practioner understands the cause of the wound and considers which of the above categories the wound fits into. An understanding of the basic principles of each type of wound is also essential to ensure well informed clinical decision making.

> The three Wound Continuums, Healing, Infection and Exudate provided a framework for assessing a wound in a systematic manner. However it is essential that when assessing a wound the practitioner understands the underlying pathology of the wound so as to accurately inform clinical decision making. In the case of a pressure ulcer removing the cause e.g. an inappropriate seat cushion can prevent further damage and facilitate healing. In the case of Pressure, Leg and Diabetic Foot Ulcers an understanding of the underlying pathology and key management principles for such wounds can greatly enhance the level of care provided.

> In this article the authors have sought to present a brief overview of common wound types and provide an overview of the key principles of management. It should be borne on mind that within the confines of an article only overviews of each wound type can be provided. Before embarking on any plan of care the practitioner should be aware of the underlying pathology of the wound and the basic principles of management.

PRESSURE ULCERS

Pressure ulcers (pressure sores, decubitus ulcers, bedsore) are areas of tissue death usually located over a bony prominence, which have been caused by external forces of pressure, shear and friction, Allman (1997). These may be further exacerbated

by complications arising from the individuals physical condition, such as altered nutrition, excess moisture etc, Macklebust (1987)

DETERMINING PRESSURE DAMAGE

General visual inspection of all areas of skin should form part of the assessment with special attention to bony prominences, of these areas the sacral and heel areas should be a priority.

Where an area of skin change is noted the following examination may assist in the identification of the early stages of pressure ulcer development.

- Apply light finger pressure for approx. 10 seconds to the area of concern
- Release, if the area is white and then returns to original colour, the area should have an adequate blood supply, but observation should continue. This is often referred to as blanching erythema. Preventative strategies should be considered.
- If it remains the same colour prior to pressure then it indicates the presence of pressure ulcer development, (non-blanching erythema). Preventative strategies should be implemented.
- If there is an alteration in the colour, redness, purple/black, increased heat or swelling then this may imply underlying skin breakdown, and frequency of assessment should be increased.
- For individuals with dark skin pigmentation pressure ulcer development will be indicated in areas where there is localised heat which if tissue is damaged may be replaced with coolness, purple/black discoloration, localised oedema and localised induration.

(Best practice Statement for the Prevention of Pressure Ulcers, 2003)

RISK ASSESSMENT

To consider preventing pressure ulcers we must first determine a persons risk of developing pressure ulcers. All risk assessment tools are based on factors known to predispose an individual to pressure ulcer

Pamela Cooper Clinical Nurse Specialist Grampian University Acute Trust Aberdeen

Fiona Russell Tissue Viability Nurse Grampian University Acute Trust Aberdeen

Sandra Stringfellow Tissue Viability Nurse Grampian University Acute Trust Aberdeen development such as sustained pressure, reduced mobility, incontinence, poor nutrition, age, mental alertness and poor physical condition. There are a number of risk assessment tools available to select from, Norton (1962), Gosnell (1973), Knoll (1982), Waterlow (1984) and Braden (1985). All of which have a research-based rationale dependant on their patient population however it should be remembered that they are to be used as an "aide memoir" alongside clinical judgement and experience. Any intervention adopted following risk assessment should be clearly recorded within the individuals health records.

PRESSURE ULCER CLASSIFICATION

Pressure ulcer classification tools exist to help practitioners describe the severity of tissue damage directly associated to pressure and/or shear and friction, Fletcher J (1997). There are a number of systems out there that each offer a slight variation on the degree of tissue involvement, Torrance (1983), Stirling Pressure Ulcer Severity Scale (1994), and European Pressure Ulcer Advisory Panel.

PRESSURE ULCER.

The vast majority of pressure ulcers that occur are superficial in nature, Gray, Cooper and Clark, (1999), Watret (1999). These are often referred to as Type I - which is non-blanching erythema of intact skin. (Refer to skin assessment). Partial thickness skin loss involving epidermis, dermis or both, Barton & Barton (1981). These are caused by pressure, friction and exacerbated by moisture. Type II pressure ulcers involve full thickness skin loss involving subcutaneous tissue, which may involve fascia, muscle, bone or supporting structures, Barton & Barton (1981). Primarily caused by pressure, shear and deterioration in the individuals physical condition. Due to the differences in aetiology the management of Type I and Type II pressure ulcers is very different.

Sacral Pressure Ulcers Type I

The vast majority of these pressure ulcers occur due to excess moisture often caused by incontinence, Cooper and Gray (2001). There is an increase in the friction co-efficient at the surface of the skin as well as a chemical reaction from the incontinence enzymes, which causes removal of the epidermis.

The treatment of these wounds is usually complicated due to the persistence of incontinence irritation, with no dressing being able to retain itself under those circumstances. A full assessment of the individual's incontinence problem should be carried out and action taken prevent further deterioration of the skin. It is suggested that foam cleansers should be used to cleanse the skin, Cooper and Gray (2001)and application of either a barrier cream or hydrogel depending on degree of tissue damage secured with a body worn pad if appropriate.

Heel Pressure Ulcers Type I

The heel consists of a thin layer of fat that covers the calcaneous bone. High interface pressures are routinely recorded over the heels, Berjain et al (1983), as the pressure is concentrated over a small area, this is further exacerbated by high levels of friction which causes damage to the epidermis, leading to the formation of a blister, Butcher (1999).

If a blister has developed on the heel due to pressure and friction, then it requires to be deroofed using a clean technique and treated thereafter as a moist wound. It may be worth considering the use of heel protectors to reduce the friction co-efficient.

Sacral Pressure Ulcers Type II

Type II pressure ulcers to the sacrum usually manifest as areas of purple discoloration or areas of necrotic tissue. This is due to gravitational forces, where the deep fascia moves downwards, while the sacral fascia remains attached to the sacral dermis, Collier (1999). The pressure ulcer will deteriorate in condition due to the debridement of devitalised tissue. Once debridement occurs the ulcer may extend into a cavity wound, and may involve undermining of the wound as well as the involvement of underlying structures.

When considering the management of a patient with a Type II sacral sore, the focus should not only be the management of the wound but also the appropriate use of pressure reducing support surfaces, positioning and nutritional requirements.

An individual identified with an existing pressure ulcer should be cared for on an appropriate pressure reducing support surface, these are divided into static or alternating systems. Those individuals, who are up sitting, should be sitting on an appropriate support surface. It should also be observed that they are not up to sit for more than two hours before being returned to bed for a minimum of one hour, Gebhart and Bliss (1994).

TABLE 1

Principles of Management – Pressure Ulcers

- Prevention is better than treatment. If at risk of pressure ulcer development then ensure that appropriate preventative strategies have been adopted.
- If a pressure ulcer has occurred, identify, remove or treat the cause
- Treat the wound following the principles of the wound healing continuum based on accurate classification and wound assessment.
- Ensure that the individual is cared for on an appropriate support surface while in bed and up sitting according to the location of the pressure ulcer.
- Ensure that the individuals underlying physical condition does not affect the individual's ability to heal, i.e. poor nutritional status.

TABLE 2

Principles of Management – Leg Ulcers

- All individuals with a leg ulcer should be assessed in line with national clinical guidelines
- A Doppler assessment of the circulation should be carried out by a skilled practitioner and individuals with abnormal readings referred to a specialist
- Compression therapy remains the treatment of choice for venous leg ulceration
- Arterial leg ulceration should be referred for further vascular assessment. This is required to establish the extent of the occlusion and the presence of small vessel disease. A specialist assessment will determine whether the patient is suitable for angioplasty or major vascular surgery
- In mixed ulceration features of venous ulcer in combination with signs of arterial impairment requires assessment by and experienced practitioner. The person conducting the assessment should be aware that ulcers may be arterial, diabetic, rheumatoid or malignant and refer the patient for specialist medical assessment (RCN, 1998). Reduced compression therapy should only be carried out by a competent practitioner.
- Due to the complex nature of diabetic lower leg ulceration it's advisable to obtain specialist referral by the multidisciplinary team ensuring a specialist Doppler assessment and the involvement of the diabetologist.

Heel Pressure Ulcers Type II

Type II pressure ulcers to the heel present with an area of devitalised tissue over the calcaneous bone. Once debridement of the devitalised tissue has occurred, exposure of the calcaneous bone may occur. The wound should be treated with moist wound care products to optimise healing. The dressings should be minimised in thickness so as not to reduce the individual's mobility. Occlusive dressings will reduce the pain however they may not alleviate it and therefore when planning rehabilitation and mobility analgesia should be considered.

To prevent further deterioration of the pressure ulcer the individual should have a pressure reducing heel device for both in bed and whilst up sitting.

CONCLUSION.

The management of pressure ulcers is often complex and fraught, with attention being focused on treating the wound, however when caring for an individual with a pressure ulcer the key management principles should be considered:

LEG ULCERS

What is a leg ulcer?

Definition – a chronic leg ulcer is defined as an open lesion between the knee and the ankle joint that remains unhealed for at least four weeks (SIGN, 1998)

There are numerous causes of lower extremity wounds such as vascular disease, infection, pressure, cancer, connective tissue disorders, metabolic diseases, drugs, insect bites, trauma and autoimmune disease (Bonham, 2003).

Measurement of ankle brachial pressure ratio (index) (ABPI) by hand held Doppler is essential in the assessment of chronic leg ulcers (SIGN, 1998).

The ABPI is calculated for each leg by dividing the highest ankle systolic pressure of each leg by the higher of the two brachial pressures (Jones, 2000)

- Pressures of 0.5 0.8 indicate evidence of significant arterial impairment (0.5 = 50% reduction in arterial blood flow).
- Pressures of 0.6 0.7 may have reduced compression if it has been assessed and applied by an experienced trained leg ulcer care expert (RCN, 1998)
- Pressures of 0.8 and above are suitable for compression (RCN, 1998). Caution should be

taken with diabetic patients and in patients with arteriosclerosis, as abnormally high readings might be because of calcified arteries (Pudner, 1998).

Below are four of the most prevalent types of lower extremity wounds encountered in clinical practice.

Venous Leg ulcer

The condition and symptoms of chronic venous insufficiency are due to impaired drainage in the venous system with subsequent venous hypertension.

Common sites for venous leg ulcers are above medial malleoli and above the lateral malleoli.

Patients with venous leg ulceration (ABPI >0.8) should have elastic multilayer graduated compression bandages applied with a simple non-adherent dressing to the wound. Therapeutic compression provides a minimum of 30 - 40 mmHg pressure at the ankle.

Visual assessment of the skin and lower leg.

Venous leg ulcers tend to be shallow without punched out margins

Lipodermatosclerosis: the characteristic brown staining of the lower leg is suggestive of chronic venous disease. This occurs later on as progressive deposits of fibrous tissue in the deep dermis and fat results in the woody induration of mainly the gaiter area of the shin.

Atrophie blanch is often associated with irregular pigmentation, may also appear and presents as white areas of extremely thin skin dotted with tiny tortuous blood vessels.

Eczema, commonly known as stasis dermatitis, may appear in the gaiter area.

Arterial leg ulcer

Arterial ulcers are less common than those due to venous disease but arterial insufficiency, if present, complicates the healing of the wound. Identified risk factors for arterial disease include smoking or tobacco use, hyperlipaemia, diabetes, hypertension, obesity, advanced age, trauma, sickle cell disease, and cardiovascular disease.

Atherosclerosis reduces the blood flow to the lower limbs during exercise which can cause intermittent claudication so the person has to stop and rest due to lack of blood supply to the muscles.

The majority of patients with claudication have an ABPI between 0.8 and 0.4, while patients with rest pain generally have an ABPI of < 0.4 (Sumner, 1989)

Visual assessment of the skin and lower leg

Pallor foot when the patient is lying flat in their bed is an indication of ischaemia.

In some cases, the skin may appear dusky red or cyanotic blue where impaired perfusion has resulted in blood stagnation within dilated arterioles (Foster, 1987)

Arterial ulcers are mainly found on the anterior shin, over toe joints, over malleoli and under the heel (Donnelly, 2000)

Mixed Venous/Arterial

These will have the features of a venous ulcer in combination with signs of arterial impairment (RCN, 1998)

A full lower leg assessment should be performed and if the ABPI is reduced (for example, <0.8) the patient should be referred for a routine vascular referral. (RCN, 1998). In order to treat the venous component of the disease and promote wound healing without causing further ischaemia or injury, use of reduced compression at levels of 23 to 30 mmHg is indicated if the ABPI is 0.6 - 0.8. The patient must be carefully monitored in these circumstances and careful attention paid to correct application of compression to ensure that tissue injury does not occur (Bonham, 2003).

Diabetes and Neuropathy

Atherosclerosis is common in people with diabetes, occurs bilaterally, and affects the microvascular as well as large vessels. Patients with long-term diabetes commonly suffer from sensory, motor, and autonomic neuropathy due to impaired nerve function from hyperglycaemia. The combination of poor perfusion, altered sensation, and motor/nerve-induced foot deformity from neuropathy results in limited joint mobility and gait alteration, which causes abnormal stress and pressure on the foot leading to callus development. This increased pressure results in ulceration which leads to infection, gangrene, and limb loss far too often in patients with diabetes. Wound infection is particularly troublesome because it can occur without patient awareness of the usual signs of pain, swelling, and erythema resulting in an extensive infection before it is recognised.

DIABETIC WOUNDS

Diabetes is a common health condition. About 1.4 million people in the UK are known to have diabetes — that's about three in every 100 people.

Diabetic Foot Wounds present a significant clinical management challenge and carries a high risk for those who suffer from them.

Patients with diabetic ulceration may have deceptively high-pressure readings and such patient should be referred for specialist assessment. Their ulcers are usually found on the foot and often on bony prominences such as the bunion area or under the metatarsal heads and usually sloughy or necrotic in appearance (Cullum & Roe, 1995). Diabetic patients may have neuropathic, arterial and or venous components (Browse et al 1988; Nelzen et al 1993). Consequently, all diabetic patients with leg ulcers require a multidisciplinary approach to care ensuring the appropriate specialist referrals are made. It's essential that a diabetologist is involved in this process.

Diabetic patients with type 2 diabetes have a 3-5fold increased risk of developing peripheral arterial disease compared to people without diabetes (Sherman and Chulakadabba, 1999; Hurst & Lee, 2003). For those patients with peripheral arterial disease and diabetes, the risk of myocardial infarction and stroke are raised and the rate of amputation is increased by as much as seven times (Dormandy & Murray, 1991).

Painful diabetic neuropathy's symptoms are often slight at first. Some mild cases may go unnoticed for a long time. Numbness, pain, or tingling in the feet or legs may, after several years, lead to weakness in the muscles of the feet. Occasionally, diabetic neuropathy can flare up suddenly and affect specific nerves so that an affected individual will develop double vision or drooping eyelids, or weakness and atrophy of the thigh muscles. The loss of sensation in the feet may increase the possibility for foot injuries to go unnoticed and develop into ulcers or lesions that become infected. The multi disciplinary approach is required to reduce pain and if a wound is present irradiate infection, fully assess the extent of the vascular damage/ nerve damage and whether surgical intervention is required. The patient requires the appropriate medication to manage their pain and if there is a wound it may be treated with a topical antimicrobial until the confirmed diagnosis has been obtained. (Benbow et al, 2004) (Gray et al, 2003)

Diabetic foot ulcer management is complex in nature with a high rate of amputation. In a two year retrospective study in Gwent they had an amputation prevalence rate of 7% (De P et al,

TABLE 3

Principles of Management – Diabetic wounds

- Diabetic Foot Wounds present a significant clinical management challenge and carries a high risk for those who suffer from them
- The two main features of foot ulceration are ischaemia and neuropathy, both of which predispose to infection and lead to necrosis of the tissue.
- Ensure the appropriate analgesia and antidepressants are prescribed for painful diabetic neuropathy (Benbow et al, 1999).
- Appropriate specialist referral should be made if the wound is infected.
- A multi- disciplinary approach to patient care is required when this problem manifests itself due to its complicated nature.

TABLE 4

Principles of Management – Surgical wounds

- There are many varied surgical techniques that can result in the development of a wound: Incisions or excisions, investigative or corrective, open or keyhole
- Four types of wound healing are generally recognised: Primary closure, healing by first intention ,open granulation, healing by secondary intention, delayed or secondary closure, sometimes called healing by third intention or tertiary intention and grafting or flap formation
- The two main potential complications following surgical intervention are infection and dehiscence
- Early identification of surgical wound infection can reduce the damage to the wound
- The aim for all surgical wounds should be to provide an optimal wound healing environment which involves minimal disturbance of the wound and prevention of bacterial invasion

2000). This has a huge psychosocial impact on the patient as well as the cost implications. Krentz et al estimated an annual hospital cost of £4000 000 in a prospective survey conducted in 1997 (Krentz AM et al 1997).

SURGICAL WOUNDS

There are many varied surgical techniques that can result in the development of a wound:

- Incisions or excisions
- Investigative or corrective
- Open or Keyhole

The following four types of wound healing are generally recognised: (Thomas 1990)

- Primary closure, healing by first intention
- Open granulation, healing by secondary intention
- Delayed or secondary closure, sometimes called healing by third intention or tertiary intention
- Grafting or flap formation

Primary Closure

Most clean surgical wounds are managed by primary closure. In this technique the surgeon approximates the edges of the wound and individually sutures the different layers of tissue together. Primary Closure is achieved by using either sutures, staples, steri-strips, tissue adhesives or a combination of these. These wounds usually seal within 24 to 48 hours and heal in 8 to 10 days, when removal of sutures or staples takes place at the discretion of the surgeon. Usually these wounds are covered in a low-adherent island dressing for the first 24 to 48hrs, then often left exposed.

Secondary intention

In wounds that have sustained a degree of tissue loss as a result of surgery, or where an area has been excised and drained, e.g. due to abscess formation or pilonidal sinus excision. Primary closure may be deemed undesirable or impossible to bring the edges of the wound together. In these situations the surgeon may favour leaving the wound open to heal by secondary intention. The duration of healing will be variable for each individual case, wound healing is often affected by intrinsic and extrinsic factors that may result in complications (Baxter 2003). As with all dressing choices, the primary function of a wound dressing is to promote healing by maintaining a moist wound environment. Due to healing by secondary intention, each dressing will require to be tailored to the size, depth, position and exudate levels of the wound.

Tertiary intention

Delayed primary closure is rather less commonly used. This occurs when the surgeon asserts that primary closure may be unsuccessful at the time of surgery, due to e.g. infection, poor blood supplies or the need for excessive tension during closure. Usually patients will return for primary closure three to four days later. During this time the dressing choice will be similar to those when left to heal by secondary intention.

Grafting or flap formation

A skin graft is an area of skin that is surgically removed from one part of the body and transplanted to another. The skin graft replaces tissue that has been destroyed or creates new tissue where none exists. The major disadvantage of this technique is that another wound is created from the donor site. Skin grafting can be either partial thickness or split skin grafts, which are the most common types. These are removed from a suitable donor site, such as the thigh or buttock; donor sites usually heal rapidly within 10 to 14 days. Full thickness skin grafts are used for more specialist surgery where fat, hair and sebaceous glands are removed for transplanting. Skin flaps differ from grafts where often the donor tissue is not completely removed but rotated to its adjacent position with the blood supply remaining intact.

Surgical wound complications

The two main potential complications following surgical intervention are infection and dehiscence. Early signs of wound infection are defined by redness, pain, heat and swelling of the wound and periwound area. These signs must not be confused with the inflammatory stage of wound healing, usually around days 3 to 7 post operative.

Infection amongst other contributing factors can lead to dehiscence of the surgical wound, this is where the wound can either partially or fully open following primary closure. The options following dehiscence is to allow the wound to heal by secondary intention or by delayed closure.

Post operative wound care will vary from centre to centre and practitioner to practioners, however the treatment aims for all surgical wounds are to provide an optimal wound healing environment which involves minimal disturbance of the wound and prevention of bacterial invasion.

TRAUMATIC WOUNDS

Skin Tears

Skin tears usually occur in the elderly or individuals with friable skin often due to underlying medical conditions or long term use of steroid medications. The trauma is often located to the individuals extremities such as arms and lower leg, where an accidental bump or knock causes a skin tear. The epidermis is displaced but still retains a blood supply.

The most efficient way to mange these wounds is to reapply the skin tear, trying to bring the wound edges together to heal by first intention. This may be achieved by initially moistening the wound to facilitate reapplication of the skin tear. Once reapplied, steri-strip and/or a non-adhesive dressing can retain the skin tear.

Grazes/Abrasions

Grazes and abrasions are a superficial injury where the skin is rubbed or torn usually caused by falling onto a rough or gritty surface, Dealy (1994). Abrasions and grazes should be cleansed thoroughly to ensure that no foreign bodies remain embedded in the wound bed. These wounds are often painful due to the nature of their occurrence.

The majority of these wounds can be effectively treated with a simple non-low adherent dressings, however if painful the individual may benefit from the application of a film or hydrocolloid dressing which occludes the wound, keeps the nerve endings moist therefore reducing the pain.

Laceration

A laceration is a wound caused by blunt trauma, which has split or torn the skin forming jagged wound edges, Collins F et al (2002). A thorough assessment of the wound should be carried out to ensure that there is no underlying structure trauma. If the wound edges are clean then closure can be obtained by first intention through, steri-strips, glue or suturing, Dealey (1994). If the wound is contaminated, primary closure is not indicated, the wound should be treated with a topical antimicrobial until the infection is cleared. The wound should then be encouraged to heal by secondary intention and moist wound management

Penetration/Stab Wounds

Knives, bullets or other sharp missiles may cause penetrating wounds, Thomas (1990). Although the external appearance of a penetrating wound may suggest that the injury is relatively minor, internal damage can be considerable, depending upon the site and depth of the penetration and or the velocity of the bullet or missile, Owen-Smith (1985). Therefore prior to implementing any treatment plan the wound should be thoroughly explored by a doctor as surgery may be necessary.

Burns

Burns require separate consideration from other traumatic wounds due to the specialised nature of care required for their treatment. The following show the varying forms of burns:

Thermal burns, which can either, be caused by dry heat such as fires flash flames, friction or by wet heat that is usually referred to as scalds from hot liquid.

Chemical burns, which involves chemicals of an acid or alkaline nature.

Electrical burns, which can be low voltage usually a domestic accident <1,000 volts. High voltage burns are industrial accidents often involving power lines,>1,000 volts.

Radiation burns, is caused by accidental exposure to ultra-violet light, x-rays and gamma rays.

Extent of Burn.

The extent of a burn is described by a percentage that indicates the amount of the total body surface area (TBSA) involved. There is little consensus to determine what constitutes a minor scald which can be treated as an outpatient and what TBSA should be involved before a referral to a specialist center is made, Fowler (1999). Young patients with 5-10% TBSA should be referred to a specialist center along with adults who have 5-10% burns to their face and hands, Turner (1998). Any individual with 10%, or more of the TBSA as a superficial-partial thickness burn would benefit from a review at a specialist center.

Burn Classification

The depth of tissue penetrated by the thermal insult determines the classification of the burn. The various severity's are:

Superficial, this involves the epidermis only. The skin is dry and intact but very red and painful to touch. May form blisters but will usually heal within 3-7 days with minimal or no treatment dependant on the formation of blister, Fowler (1999). If skin intact it may help to apply a bland

TABLE 5

Principles of Management – Trauma Wounds

- Identify the source of trauma and remove if appropriate.
- If wound contaminated or debris present ensure that wound is cleansed thoroughly.
- Thorough assessment should be carried to ascertain if any underlying structure involvement.
- Treat the wound following the principles of the wound healing continuum based on accurate classification and wound assessment.
- Always document plan of care and action taken in the individuals care records.

TABLE 6

Principles of Management – Complex Wounds

- Always establish the underlying pathology of a wound
- Recognise the impact that underlying conditions such as Rheumatoid Arthritis
- Seek specialist advice or referral to ensure underlying pathology is managed effectively

moisturiser or after-sun to keep skin supple and rehydrated.

Superficial –partial thickness burns involves the epidermis and the superficial layer of the dermis. The skin usually shows immediate blistering, moist and exudes haemo-serous fluid. These burns are very painful for the individual, Fowler (1999). Will require debridement of the blistered areas, if the wound was contaminated at time of injury treatment with a topical antimicrobial wound be recommended. If the wound is clean then limit dressing changes by applying an absorbent nonadherent dressing but also ensuring that it does not incapacitate the individuals ability to move and excersise the affected area if possible.

Deep –partial thickness, deep dermal burns are burns, which involve the epidermis, dermis but leave the hair follicles and sebaceous glands intact, Fowler (1999). The burn appears as white/creamy colour with blistering. These can be treated by either debridement and grafting or if the area is small autolytic debridement of the burn tissue and active treatment with moist dressings.

Full thickness burns, involves all structural layer, epidermis, dermis, subcutaneous layer and/or deeper structures. The appearance of the skin is one of a waxy white grey area or yellow/black translucent leathery appearance, Fowler (1999). There is little pain associated with these wounds due to the damage of the nerve endings but wound edges can be very sensitive. These wound require surgical intervention with extensive debridement and grafting.

COMPLEX WOUNDS

The title Complex Wounds refers to wounds which may have an underlying pathophysiology e.g. Pyoderma gangrenosum or where wounds maybe complicated by the individuals prevalent medical condition such as Rheumatoid Arthritis. It is beyond the scope of this article to discuss all of these types of wounds, however the following example has been selected.

In the case of Pyoderma gangrenosum, an uncommon cause of skin ulceration. It may affect any part of the skin, but the lower legs are the most common sites. It is thought to be an auto immune disorder. Pyoderma gangrenosum often affects a person with an underlying internal disease such as:

- Inflammatory bowel diseases (ulcerative colitis and Crohn's disease)
- Rheumatoid arthritis
- Chronic active hepatitis.

Pyoderma gangrenosum usually starts quite suddenly, often at the site of a minor injury. It may start as a small pustule, red bump or blood-blister. The skin then breaks down resulting in an ulcer. The ulcer can deepen and widen rapidly. Characteristically, the edge of the ulcer is purple and undermined as it enlarges. It is usually very painful. Several ulcers may develop at the same time. Untreated, the ulcers may continue to enlarge, persist unchanged or may slowly heal. Treatment is usually successful in arresting the process, but complete healing may take months. Patients with pyoderma gangrenosum are normally cared for by a dermatologist.

This type of wound is an example of where an underlying condition can result in the development of a wound, this underlines the need to be able to identify the factors that have led to the development of the wound. Where a wound cannot be identified as fitting into the first five categories it can be categorised as a complex wound and consideration given to the need for a specialist referral / advice. Wounds such as fungating lesions, vasculitic ulcers, wounds of unknown pathology are other examples of complex wounds.

CONCLUSION

Within this article the authors have sought to provide an overview of some common wounds types and highlight their key management principles. Each wound type of worthy of an article on its own and the authors recognise the limitations of this article

REFERENCES

Abruzzese, R. the effectiveness of an assessment tool in specifying nursing care to prevent decubitus ulcers, In: PRN: the Adlephi Report, 1982, Project for research in Nursing. Garden City, New York: Adelphi University, 1982.

Allman R.M. (1997) Pressure ulcer prevalence, incidence, risk factors and impact. Clinical Geriatric medicine 13 (3); pp. 421-436.

Barton S. & Barton M. (1981) The management and prevention of pressure sores. London. Faber and Faber.

Baxter H (2003) Management of Surgical Wounds. Nursing Times. Vol 99, No 13.

Benbow SJ. Daousi C. MacFarlane I A. Diagnosing and managing chronic painful diabetic neuropathy. The Diabetic Foot. 7 (1) 34-44 Spring 2004

Bergstrom, N. Braden, B. Laguzza, A, et al. (1987) The Braden scale for predicting pressure sore risk. Nursing and Residential Care. 36.4.205-210.

Berjain R, Douglass H, Holyoke E, Goodwin P, Priore R (1983) Skin measurements on various mattress surfaces in cancer patients. American Journal of Physical Medicine 62: 21726

Best practice Statement for the prevention of pressure ulcers (2003)

Bonham P (2003) Assessment and Management of Patients with Venous, Arterial, and Diabetic/Neuropathic lower extremity wounds. American Association of Critical-Care Nurse 14, 4, 442- 456

Benbow SJ, Cossins L, Mac Farlane IA (1999) Painful diabetic neuropathy. Diabetic Medicine 16: 632-44

Browse NL, Burns KG, Lea Thomas M. (1988) Diseases of the veins:pathology, diagnosis and treatment. London, Edward Arnold.

Butcher M (1999) identifying and combating the risk of pressure. Nursing Standard 14(3); 5863

Collier M, (1999) in Wound Management theory and practice, edited by Miller,M. and Glover ,D. Nursing times Books. Collins, F. Hampton, S. and White, R. A-Z dictionary of wound care. Quay books. (2002)

Cooper, P and Gray, D. (2001) Comparison of two skin care regimes for incontinence. British Journal of Nursing, 10(6), 6-20.

Cullum N, Roe B (1995) Leg ulcers nursing management – a reasearch-based guide. London, Bailliere Tindall.

Dormandy JA, Murray GD (1991) The fate of the claudicant: a prospective study of 1969 claudicants. Eur J Vasc Surg 5: 131- 3

Dealy, C. The Care of Wounds. Blackwell Science. (1994)

De P, Kunze G, Gibby OM, Harding K (2000) Outcome of diabetic foot ulcers in a specialist foot clinic. The Diabetic Foot 4 (3): 131-36

Elkeles RS. Wolfe JH. ABC of vascular diseases. The diabetic foot. British Medical Journal. 303(6809):1053-5, 1991 Oct 26.

European pressure Ulcer Advisory Panel, Guide to Pressure Ulcer Grading, EPUAP review, 3(3) p75.

Fletcher J, (1997) Pressure sore grading. Journal of Wound Care. Resource file.

Foster A, Edmons ME. Examination of the diabetic foot. Practical Diabetes 1987; 4 (3): 105-106

Fowkes FG, Housley E, Macintyre CC, Prescott RJ, Ruckley CV. Variability of ankle and brachial systolic pressures in the measurement of atherosclerotic peripheral arterial disease. J Epidemiol Community Health 1988: 42 (2) 128-133

Fowler, A. Burns in Wound Management theory and practice, Miller M and Glover D (ed.) Nursing Times books. 1999.

Gebhardt, K. and Bliss, M.R. (1994) Preventing pressure sores in orthopedic patients – is prolonged chair nursing detrimental? Journal of Tissue Viability, 4(2), 51-54.

Gosnell, D (1973) An assessment tool to identify pressure sores. Nursing and Residential Care. 22. Pp55-59. Gray D, White R.J, Cooper P. (2003) The wound healing continuum. In: R.J. White (ed.) The Silver Book. Quay Books Division, MA Healthcare Ltd. Dinton, Wiltshire UK.

Gray, D. Cooper, P. and Clark, M. (1999) Pressure ulcer prevention in an acute hospital. Poster presentation EPUAP, Amsterdam 1999.

Hurst RT, Lee RW (2003) Increased incidence of coronary artherosclerosis in type 2 diabetes mellitus: mechanisms and management. Ann Intern Med 139; 824-34

Jones J (2000) The use of holistic assessment in the treatment of leg ulcers. British Journal of Nursing. 9, 16, 1040- 1052.

King KM. Diabetes. Diabetes: classification and strategies for integrated care. British Journal of Nursing. 12(20):1204-10, 2003 Nov 13-26.

Krentz AM, Acheson P, Basu A, Kilvert A, Wright AD, Nattrass M (1997) Morbidity and mortality associated with diabetic foot disease: a 12 month prospective survey of hospital admissions in a single UK centre. Foot 7: 144-47

London NJ. Donnelly R. (2000). ABC of arterial and venous disease. Ulcerated lower limb. BMJ books. 320(7249):1589-91, ISBN: 0727915614

Macklebust, J. (1987) Pressure ulcers, aetiology and prevention. Nursing clinics of North America. 22, 2.

Nelzen O, Bergqvist D, Lindhagen A (1993) High prevalence of diabetes in chronic leg ulcer patients: a cross-sectional population study, Diabet Med, 10, 345-350.

Norton D, McLaren R, Exton-Smith A, an investigation of geriatric nursing problems in hospital. Edinburgh Churchill, Livingstone, 1962.

Onugha N. Jones AM. Care study. The management of hard-to-heal necrobiosis with PROMOGRAN. British Journal of Nursing. 12(15):Tissue Viability Supplement: S14, S16-20, 2003 Aug.

Owen-Smith, M. wounds caused by the weapons of war, in Wound Care, Westaby S. (ed.), London, Heinemann Medical 1985, 110-120. Pudner R (1998) The management of patients with a leg ulcer. Journal of Community Nursing. 12, 5, 26-33.

Reid J and Morrison M (1994) Towards a consensus: classification of pressure sores. Journal of Wound Care 3(3) 157-160.

Royal College of Nursing (1998) Clinical Practice Guidelines: The Management of Patients with Venous Leg Ulcers. RCN Institute, Centre for Evidence- Based Nursing, University of York and the School of Nursing, Midwifery and Health Visiting, University of Manchester.

Scottish Intercollegiate Guidelines Network. The care of patients with chronic leg ulcer. SIGN 26 1998;July.

Shearman CP, Chulakadabba A (1999) The value of risk factor management in patients with peripheral arterial disease. In: The Evidence for Vascular Surgery. Tfm Publishing, Harley, Shropshire

Sumner, DS. Non-invasive assessment of peripheral arterial occlusive disease. In Rutherford, K.S. (ed.). Vascular Surgery (3rd edn). Philadelphia, Pa: WB Saunders, 1989.

Thomas S (1990) Wound Management and Dressings. London, The Pharmaceutical Press.

Thomas, S. Wound Management and Dressings. The Pharmaceutical Press. (1990)

Torrance C (1983) Pressure sore: Aetiology, treatment and prevention. London Croom Helm

Turner, D.G. (1998) Ambulatory care of the burn patient. In: Carrougher, G.J. Burn Care and Therapy. St Louis: C.V.Mosby.

Waterlow, J (1985) A risk assessment card, Nursing Times 81.48.49-55.

Watret, L. (1999) Using a case-mix-adjusted pressure sore incidence study in a surgical directorate to improve patient outcomes in pressure ulcer prevention, Journal of Tissue Viability, 9(4), 121-125.

WHO (1999) Definition, Diagnosis and Classifications. Report of a WHO consultation. Part 1: Diagnosis and Classification of Diabetes Mellitus. WHO, Geneva

Applied Wound Management: A Clinical Decision Making Framework

ound management is a constantly evolving speciality with regular advances in terms of new products and knowledge. One the most recent advances has been the development of the Wound Bed Preparation [WBP] concept [Jones 2004]. This is intended to direct practitioners to adopt a systematic approach to wound management. The key principles underlying the concept, namely debridement, wound bio burden management and exudate management, have been recognised as good clinical practice for some time [Dealy 1994]. However, WBP seeks to incorporate these issues into a more precise and systematic approach, leading to appropriate dressing selection. As identified by Jones [2004], this could be described as a paradigm shift in wound care, and for those active in the field there is a need to work within this new paradigm. In this paper, the authors present a framework which utilises three different wound continuums; Healing, Infection and Exudate, whereby the practioner can systematically approach wound assessment.

As the workload of nurses and podiatrists increases, and the need to apply best practice remains a cornerstone of the modern NHS, so the need for competent, systematic clinical decision making increases. It is of great benefit to both the practioner and the patient that the decision making process which leads to a treatment plan is clear, consistent, and coherent. Such an approach reduces the risk of poor practice and subsequently the risk of litigation. Within the UK little is known of the true extent of wound healing by secondary intention. National average healing rates for leg ulcers and pressure ulcers simply do not exist; in fact it is very difficulty to even estimate how many of these wounds exist in the first place. The Applied Wound Management concept allows the categorisation of most wounds healing secondary intention and if applied in a clinical setting it can facilitate clinical audit, producing data which could define the true nature of wounds healing by secondary intention in the UK

Abstract

Recent developments in the field of wound care, in particular the development of Wound Bed Preparation, have led to an increased awareness of the need for a systematic approach to management. While the key principles of debridement, wound bio burden management and exudate management, have been long established, Wound Bed Preparation has sought to introduce a more systematic approach. Applied Wound Management seeks to develop Wound Bed preparation by utilising three continuums:- Healing, Infection and Exudate, to facilitate a systematic assessment of wounds healing by secondary intention. This system not only supports clinic decision making, but also facilitates clinical audit by allowing the easy categorising of the majority of wounds healing by secondary intention. It should, however, be recognised that once an assessment has been completed, consideration should be given to the underlying pathology of the wound and the treatment/management plan developed accordingly. Applied Wound Management and its associated software can not only support clinical decision making, but also provide a clinical audit tool, which, if utilised appropriately, can provide valuable clinical outcome information.

APPLIED UTILISING WOUND MANAGEMENT

The Wound Healing Continuum

The wound healing continuum (WHC) is an aid to understanding the type of tissue present in the wound and the progress of the wound according to which type of tissue is of primary clinical importance. As the continuum is followed from left to right, i.e. from black to pink, it correlates with healing of the wound. Not all wounds will naturally progress in this fashion; for example not all wounds will exhibit black necrotic tissue at any stage in their existence.

Using the Wound Healing Continuum

Identify the colour that is furthest to the left of the continuum, for example if the wound contains yellow slough and red granulating tissue the wound would be defined as a yellow/red wound. A key objective of the wound management plan would be to remove the yellow tissue and promote growth of red granulating tissue. The management plan

David Gray

Clinical Nurse Specialist Department of Tissue Viability Grampian Acute Health Services Aberdeen

Pam Cooper **Clinical Nurse Specialist** Department of Tissue Viability Grampian Acute Health Services Aberdeen

Richard White Senior Research Fellow Department of Tissue Viability Grampian Acute Health Services Aberdeen

Andrew Kingsley Clinical Nurse Specialist [Infection Control and Tissue Viability] North Devon District Hospital Barnstaple

should therefore focus on removal of the yellow sloughy tissue and promotion of the red granulation tissue. As this objective is achieved so the patient can progress along the wound healing continuum towards the right and pink /healing status.

The Wound Infection Continuum

The wound infection continuum (WIC) is an aid to understanding the bioburden (level of bacteria present) in the wound and its likely effects on healing. There are four criteria which are stages from left to right with the most severe, spreading wound infection on the left. Moving right on the continuum through the stages of local infection, and critical colonization, to colonization. Spreading infection, e.g. cellulitis, can be a life threatening condition. Local signs and symptoms associated with a spreading soft tissue infection include; spreading redness [greater than 2cm around the wound margin], very high exudate levels, pain, malodour, heat in the surrounding tissues and blistering. Localised infection is charcterised by less than 2cm of redness around the wound margin; symptoms similar to spreading infection may also be present but to a lesser degree. Critical colonisation is charcterised by delayed healing, malodour, raised exudate levels and slough may also be present, but the wound will not present as if locally infected. A colonized wound is the normal healing state of a wound healing by secondary intention; a reduction in the wound size over a two week period of 'appropriate treatment' would suggest an acceptable level of colonization.

Using the Wound Infection Continuum

The Wound Infection Continuum is a simple sliding scale which can be used as an aid to clinical decision making regarding the level of bacterial colonisation of a wound. A wound may never move from the furthest point to the right on the continuum, Colonisation, during the entire treatment period. This will lead to optimal healing, however, where a patient is identified as having a wound which has a spreading infection, localized infection or critical colonization , this should be considered when developing wound management plan.

The Wound Exudate Continuum

The wound exudate continuum (WEC) is an aid to quantifying the volume and viscosity of wound exudate. Traditionally, wound exudate has been considered in terms of its volume only with little thought given to its viscosity. However, the viscosity of the exudate can be an important indicator of the wounds status (Cutting 2004). The gradings or stages on this continuum are high, medium and low for both volume and viscosity. This wound exudate to be categorised by a numerical score (see diagram) e.g. a low volume and of medium viscosity would be a Low/Med category and would score 4 placing it in the low exudate portion of the continuum. Any score in the green zone should be seen as advantageous to wound healing, the amber zone would cause concern if the previous recording had been green, but not so if the previous recording had been red. A score in the red zone should be investigated further as this may indicate local or spreading infection

Using the Wound Exudate Continuum

When reviewing the wound the exudate on the dressing and present in the wound should be assessed along with information relating to the number of dressing changes required over a 48 hour period. Any wound assessed as having both high viscosity and high volume of wound exudate wound score a full ten points and must be regarded as causing serious concern. It is likely that such a wound may indicate a spreading infection, sinus or fistula formation or some other cause for concern. Any wound scoring 6 points would be regarded as requiring regular review, it maybe that this finding is entirely consistent with the treatment applied e.g. the liquefying of wound slough, the wound may have previously been scored in the red zone and as such a score of 6 would indicate an improvement. Where a wound had previously been scored as 2-4 points and is observed to be in the amber zone [scoring 6 points] this could be an early signs of critical colonization or of the development of a wound infection and should be acted upon.

Wound Type

Most wounds can be categorised into one of six different types, Pressure Ulcer, Leg Ulcer, Diabetic Foot Ulcer, Surgical Wound, Trauma Wound and Complex Wound. It is vital that before embarking on a plan of care for a person with a wound that the cause of the wound is identified and treated if required, e.g. all leg ulcers should be assessed using national guidelines. Failure to do so can lead to delayed healing and potentially damaging complications. A paper written to support the AWM framework by Cooper, Russell and Stringfellow [2004] provides more detailed information. It is however suffice to say that it is undesirable for a practioner to embark upon a plan of treatment/ management without having an understanding of the pathology of the wound and

its basic principles of management.

Using Applied Wound Management in the Clinical Setting

Where a practioner utilises the three continuums they can define a wound as a black [WHC], Colonised [WIC], Low Exudate [WEC] type wound. It is at this stage consideration must be given to the cause of the wound. Such an assessment in the case of a heel pressure ulcer would necessitate a very different form of management / treatment than if it were a leg ulcer or a diabetic foot ulcer. Each wound would require treatment / management relevant to the underlying pathology despite recording the same initial results following assessment by the three continuums. Table 1 gives further examples of this type of decision making and below there are tow cases studies illustrating the Applied Wound Management

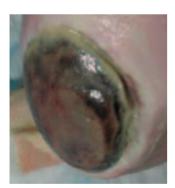
Image				Туре	Treatment Aims
0	Black / Yellow	Locally Infected	High	Trauma / Burn	Facilitate removal of black tissue Antimicrobial therapy High Exudate absorption
()	Yellow / Red	Locally Infected	Moderate	Trauma / Burn	Facilitate removal of yellow tissue Antimicrobial therapy Moderate exudate absorption
No.	Black	Colonised	Low	Pressure Ulcer	Rehydration of black tissue
	Yellow / Red	Colonised	Low	Pressure Ulcer	Facilitate removal of slough Promote granulation tissue Absorption of low exudate
	Red	Critically Colonised	Moderate	Diabetic Foot Ulcer	Antimicrobial therapy Moderate exudate absorption Specialist referral
-	Red	Colonised	Moderate	Trauma	Promotion of granulation Moderate exudate absorbtion
	Red/Pink	Colonised	Low	Trauma	Promotion of epithelialisation Low exudate absorption
~1	Black / Yellow	Critically Colonised	Moderate	Leg Ulcer	Antimicrobial therapy Moderate exudate absorption Specialist referal
0	Yellow / Red	Critically Colonised	Moderate	Pressure Ulcer	Antimicrobial therapy Promotion of desloughing Moderate exudate absorption



Case Study I Pic 1



Case Study I Pic 2



Case Study 2 Pic 1



Case Study 2 Pic 1

Framework in use. APPLIED WOUND MANAGEMENT CASE STUDIES

Case Study I

Eighty-five year old gentleman admitted to orthopedics with a fractured neck of femur. On assessment also presented with a partial thickness dermal burn to his left shoulder blade area. The wound measures 12cm x 5cm on initial assessment

Wound Healing Continuum

Image 1 – The wound presents with a deep dermal burn of black/yellow tissue, which requires rehydrating to facilitate the debridement of the devitalised tissue.

Image 2 – demonstrates an improvement in the wound bed condition, with debridement of necrotic tissue, however the wound healing continuum still demonstrates that there is black/yellow/red and pink tissue present. Of primary importance is the black tissue followed secondly by the yellow tissue. The treatment aim is to focus on the debridement of the black/yellow tissue by rehydration.

Wound Infection Continuum

The wound infection continuum gives the reader the opportunity to consider if the wound has altered healing due to the presence of bacteria. In both images there are no signs of local infection or spreading infection. The wound is progressing and improving which would rule out any form of critical colonisation. However the wound is a chronic wound which was caused by a burn therefore the wound will be colonised, but this will not inhibit healing.

Wound Exudate Continuum

Review of images I and II show that there are low volumes of exudate with low viscosity. This indicates that exudate management is not a problem regarding this wound and can be categorised as Low. However to facilitate debridement a degree of moisture should be provided by the dressing. Once black/yellow tissue begins to soften there may be an increase in the levels of exudate, this does not indicate an infection according the wound infection continuum.

Summary

In summary, the wound presents as a black/yellow wound, which requires debridement, the wound is

colonised with low exudate levels and low viscosity. The management of the wound should be by using a dressing which facilities autolytic debridement by providing a moist environment, but does not have to absorb exudate, or have any antimicrobial properties.

Case Study II

This is a seventy-five year old lady who is cared for in a long-term care of the elderly unit following cerebral vascular accident. She presents with a pressure ulcer to her right heel which occurred due to a deterioration in her physical condition, whilst all preventative strategies had been deployed.

Wound healing Continuum

The wound Healing Continuum clearly demonstrates that Image I is 100% black necrotic tissue. Therefore the initial management of this wound is to facilitate debridement of this devitalised tissue. Image II shows that debridement of the black tissue has occurred and that the wound now presents as a yellow/red wound. The colour of importance now is yellow, with debridement of the yellow tissue pivotal to the wounds ongoing progress. The presence of red tissue indicates that the wound is reaching a stage where debridement can stop and stimulation of granular activity can occur.

Wound Infection Continuum

The initial picture of the wound shows a moist black wound. No clinical signs of infection, but malodorous. The odour comes from the anaerobes present due to the black tissue but does not indicate that the wound is infected, in fact the wound is heavily colonised. The second image shows a wound, which is critically colonised as it has stayed in this condition for a number of weeks without any sign of improvement. The treatment required is the application of a topical antimicrobial, to reduce the bioburden present at the surface of the wound and to kick-start the healing process. No antibiotics are necessary.

Wound Exudate Continuum

The wound starts off with low levels of low viscosity exudate, scoring as low on the continuum but as the wound has progressed the levels of exudate have increased as well as the viscosity scoring medium. This change in exudate should act as a trigger to clinicians that a change has occurred, and trigger a review of the current treatment. The wound according the infection continuum is critically colonised and therefore treatment with an antimicrobial should be commenced. As there is an increased level of exudate an absorbent secondary dressing should be considered.

In summary the wound starts off a black wound with no infection and minimal exudate. As debridement occurs the wound changes to a yellow/red wound which is critically colonised due to the presence of bacteria which has led to an increase in the levels of exudate.

DISCUSSION

At the centre of the Applied Wound Management framework is the Wound Healing Continuum which has been designed to address the shortcomings of previous colour based assessment tools. It recognises the variance in colour and requires to practioner to rate the wound according to the colour closest to the left of the continuum. The Wound Infection Continuum is aimed at providing a structure to wound bio burden assessment as, with the Healing Continuum the aim is to move the patients' status to the right of the continuum. The Wound Exudate Continuum aims to use exudate as an indicator of the wound condition and asks the user to rate both the viscosity and volume of the exudate. Once this assessment has been completed the exudate will fall into one of three categories which can give an indication as to the wounds underlying condition. When all three continuum assessment are taken together they provide the practioner with a clear and coherent assessment of the wounds condition. When these three assessments are consider in line with the type of wound, its pathology and the key principles of its management then the practioner can design a treatment/management plan.

CONCLUSION

Wound Bed Preparation is now recognised as a new paradigm in wound management and it is up to those active in the field to interpret the concept in a manner relevant to their own clinical practice. The Applied Wound Management framework is the authors' response to the need to develop a more systematic approach to wound assessment. Adopting such an approach can prove beneficial to both the Generalist and Specialist practioner. By utilising the AWM framework those unfamiliar with wound management can arrive at a treatment/ management plan which benefit the patient. For the Specialist utilising the framework allows the decision making process to be clear to the observer and also facilitates clinical audit in the future. Software has been developed to support the AWM framework which can generate clinical outcomes data relevant to all practioners. Whatever the level of knowledge of the practioner the Applied Wound Management framework and its associated software can provide guidance and support on a daily basis on the field of wound management

REFERENCES

Cooper P, Russell F, Stringfellow S, A Review of Different Wound Types and Their Principles of Management Applied Wound Management Supp Vol. 1 No 1 pp22-31

Cutting K F (2004). Wound exudate. In: Trends in Wound Care vol 3 ed RJ White, Quay Books, Dinton Salisbury UK>

Dealey C (1994) The care of wounds. Blackwell, Oxford: 83-121

Jones V (2004) Wound bed preparation and its implication for practice $% \left({{{\rm{D}}_{{\rm{D}}}}_{{\rm{D}}}} \right)$

An educationalist's viewpoint, Applied Wound Management Supp Vol. 1 No 1 pp 4-8

The key to progressive wound healing



MAKING SENSE. MAKING PROGRESS.







© copyright Wounds-UK 2004. Further copies can be obatined from www.enquiries@ wounds-uk.com or www.wounds-uk.com