Wound Management

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Wound Management

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Contents

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Introduction

ABOUT THIS BOOK

This book is primarily for pre-registration and newly qualified nurses. There is currently no recognised core curriculum for wound care in the pre-registration curriculum programme and wound management can often appear somewhat daunting to novice practitioners. The focus of this book is to educate practitioners in the general principles of wound care as well as techniques associated with the assessment, planning and management of different types of wound.

All aspects of healthcare delivery should be evidence based with a clear rationale for treatment. This book will provide the practitioner with an understanding of the principles of wound care and the ability to use a systematic approach to wound management.

LAYOUT OF THE BOOK

The book is set out in 10 chapters that follow a logical progression starting with the physiology of wound healing and continuing with information about the epidemiology of acute and chronic wounds. Using a holistic approach, the following seven chapters explore the principles and practice of assessing and managing a patient with a wound. The final chapter draws together the threads running throughout the book and shows how they can be applied in clinical practice.

A SYSTEMATIC APPROACH TO WOUND CARE

The information provided within this book will enable the practitioner to develop a systematic approach to learning in general and wound care in particular. The process can be divided into a series of steps thus enabling the practitioner to reach a logical conclusion regarding the most appropriate treatment plan. The steps are:

Step 1: assess the patient, wound and circumstances
Step 2: utilise existing information about the patient
Step 3: explore relevant current best practice
Step 4: make a clinical decision
Step 5: evaluate progress

EVIDENCE-BASED PRACTICE

Evidence-based practice means using current best evidence in a judicious manner to guide healthcare decisions with the aim of improving patient outcomes. In a number of instances the evidence has been used to develop clinical guidelines with recommendations for the assessment and management of specific wound types (see Table 1). The use of an evidence-based clinical guideline provides practitioners with recommendations for effective clinical practice, which can simplify their clinical decision-making, and practice that is not supported by good evidence is discouraged (McInnes *et al.*, 2000). There are national guidelines which many areas are adapting for local use (Graham *et al.*, 2005). The effectiveness of using clinical guidelines can be seen in terms of improved patient outcomes. For example, implementation of clinical guidelines for

Table 1 Different types of national and international clinical guidelines for wound care.

Wound type	Guideline issued by	Website address
Leg ulcers	Royal College of Nursing	www.rcn.org.uk
	Scottish Intercollegiate Guidelines Network (SIGN)	www.sign.ac.uk
Pressure ulcers	National Institute for Health and Clinical Excellence (NICE)	www.nice.org.uk
	European Pressure Ulcer Advisory Panel (EPUAP)	www.epuap.org
	National Pressure Ulcer Advisory Panel (NPUAP)	www.npuap.org
Diabetic foot ulcers	NICE	www.nice.org.uk
Surgical wound debridement	NICE	www.nice.org.uk

venous leg ulcers has been shown to improve healing rates and reduce costs (McGuckin *et al.*, 2002).

INTEGRATING THEORY AND PRACTICE

It is important to integrate theory and practice, and, therefore, there are case scenarios throughout the book for you to assess your knowledge. Using a systematic approach you can work through the different stages of assessment, planning and management using the knowledge you have gained from the

preceding chapter. The suggested management section after each case scenario discusses the various options, where you can compare your answers. All qualified nurses are accountable for their actions and as such should ensure that they have the required knowledge to provide optimum care for their patients.

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Wound Physiology

1

INTRODUCTION

A wound is a loss of continuity of the skin, which may occur as a result of injury, impaired blood supply or deliberate wounding such as surgery. Healing time varies according to the type of injury and the extent of tissue loss. Superficial wounds in which only epithelial tissue is damaged require a relatively short time to heal, whereas healing takes longer and is more complex in deep wounds in which vessels have been damaged.

It is essential that those involved in the care and management of wounds should have at the very least a basic understanding of the physiology of the natural processes involved in wound healing. Understanding and recognition of the different stages of healing will assist the healthcare practitioner in assessment and decision-making in the care and management of the patient with a wound. This chapter presents an overview of the physiology of wound healing. The structure and functions of the skin are described in Chapter 6.

HEALING BY PRIMARY AND SECONDARY INTENTION

- Healing by primary intention is when there is no tissue loss and the skin edges are brought together, such as in a sutured wound.

- Healing by secondary intention is when there is tissue loss and the skin edges are far apart. The wound heals from the base upwards. Examples of this type of wound are a pressure ulcer, a leg ulcer or an open excision such as an abdominal wound.

PARTIAL AND FULL THICKNESS WOUNDS

- Superficial wounds involving the epidermis (and sometimes the upper dermis), leaving lower levels of skin intact, are referred to as partial thickness wounds (Fig. 1.1). Examples are a simple skin abrasion or a donor site from a split thickness skin graft. Epithelial cells migrate towards each other from the

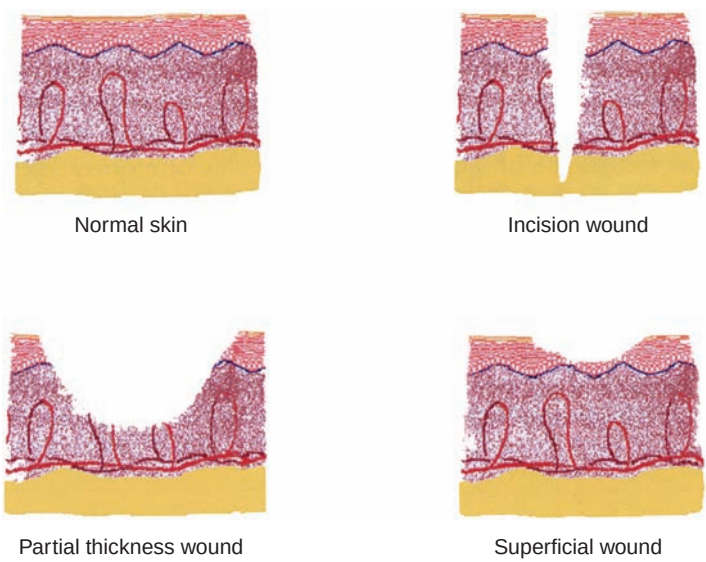

Fig. 1.1 Types of wound. Reproduced with kind permission from Dr George Cherry.

edges of the wound and from the hair follicles, sebaceous glands and sweat glands. This part of the healing process is known as epithelialisation.
- In a full thickness wound all of the dermis is destroyed and the deeper layers may also be involved. These wounds heal by secondary intention, in which granulation tissue is formed to fill the wound space and new epidermis grows over it (Knighton *et al.*, 1990).

THE HEALING PROCESS

The healing process occurs as a natural response to injury and initiates a highly complex cascade of events, which in the normal healing wound occur in an orderly and timely fashion, resulting in skin repair. Wound healing consists of four main phases that overlap each other and are intricately linked:

- Inflammation
- Reconstruction (granulation tissue production)
- Re-epithelialisation
- Maturation

Phase I – inflammation

Immediately following injury, the body's defence mechanisms produce an inflammatory response. This initial phase of healing lasts up to 4–6 days post wounding. Injured blood vessels bleed into the wound and platelets adhere to exposed collagen in the subendothelial layers of the walls of the damaged blood vessels. The platelets flatten and release substances, including proteins, which cause the platelets to become sticky. Fibrin combines with the platelets and trapped

erythrocytes to form a clot, thus occluding the damaged blood vessels (Cooper, 1990).

Activated platelets release growth factors, including platelet-derived growth factor (PDGF) and epidermal growth factor (EGF). The term 'growth factors' is used to describe the various proteins involved in coordinating the cascade of events involved in wound healing (Graham, 1998). However, the number of growth factors that relate to wound healing is unclear and there is ongoing research in this field. Growth factors have a major role in providing the means of cell communication throughout the wound healing process. PDGF supports the initial inflammatory stages of healing and also has an important role in the formation of granulation tissue, collagen and ground substance. EGF has a significant role in epithelialisation and in the formation of granulation tissue (Kunimoto, 2001). These growth factors act as chemoattractant chemicals, which facilitate the migration of neutrophils to the area. Neutrophils and monocytes can be found in the wound on the first day and are responsible for initiating the wound cleansing process by removing bacteria, devitalised tissue and debris from the wound using a process known as phagocytosis (Ovington & Schultz, 2004).

Inflammation leads to an increase in vasodilation and vessel permeability. Histamine and serotonin are released, which increase capillary permeability, allowing plasma leakage, which in turn leads to accumulation of fluid in adjacent tissues (Thomas, 1997). The increased blood supply and oedema produce the inflammatory appearance of erythema and swelling, together with heat, and the patient experiences

localised pain. Excessive fluid drains from the wound tissue as exudate. Wound fluid from acute wounds is rich in growth factors that have been shown to promote tissue repair (Chen *et al.*, 1992).

At the end of the acute inflammatory phase neutrophils decay and are themselves phagocytosed by macrophages that have matured from monocytes. Macrophages continue to phagocytose and digest bacteria, wound debris and necrotic tissue performing autolytic debridement of the wound (Moore, 2003).

Phase II – reconstruction (granulation tissue production)

Macrophages secrete growth factors crucial to wound healing and facilitate angiogenesis (the growth of new blood vessels from damaged vessels), cell migration and proliferation, the restoration of the nutrient blood supply and synthesis of new tissue (Cooper, 1990; Brem, 2001). Oxygen is vital to this process and macrophages can be inactivated by oxygen pressure below 30 mm Hg (Cherry *et al.*, 2000). Fibroblast growth factor (FGF) is released by macrophages and fibroblasts and is an important growth factor for angiogenesis and the formation of new granulation tissue (Kunimoto, 2001). The acute wound fluid stimulates fibroblast and endothelial cell growth (Katz *et al.*, 1991). Fibroblasts have a key role in this phase of healing (Harding *et al.*, 2002). They synthesise and secrete the collagen and ground substance, forming a provisional wound bed matrix (the extracellular matrix), which acts as the scaffolding for repair (Greener *et al.*, 2005).

The wound surface has a relatively low oxygen tension, encouraging the process of angiogenesis,

where new capillaries sprout from blood vessels at the wound periphery (Silver, 1985). They then join together to form a network of capillary loops, infiltrating the extracellular matrix, and supplying oxygen and essential nutrients to the wound (Cutting & Tong, 2003).

The new tissue, composed of the capillary loops, the supporting collagen and the ground substance, is red in colour and has a slightly rough, granular appearance. This is what is termed 'granulation tissue'. In wounds healing by secondary intention granulation tissue can be seen as it gradually fills the wound cavity. As the wound fills with new tissue and a capillary network is formed, the numbers of macrophages and fibroblasts gradually reduce (Dealey, 2005).

Healing may be delayed if infection is present. Healing may also be compromised if damage has been caused due to an unsuitable dressing being applied to the wound.

Wound contraction

Contraction is a normal part of wound healing that may start at around the fifth or sixth day. In this process, cellular forces pull the wound edges towards the centre of the wound. Fibroblasts have been found to be particularly important in wound contraction (Viennet *et al.*, 2004). Contraction can reduce the surface area of open wounds by as much as 40–80% of the closure (Irvin, 1987).

Phase III – re-epithelialisation

Macrophages release EGF, which stimulates both the proliferation and migration of epithelial cells.

Keratinocytes migrate from the wound edges to cover the surface and re-form the layers of destroyed epithelium. Keratinocytes are also largely responsible for reconstituting the basement membrane of the dermal–epidermal junction (Hughes, 2002).

The epithelial cells migrate and proliferate from the wound edges and cover viable tissue with a leap-frogging action. The cells multiply and divide by mitosis and migrate across the surface until they meet in the middle of the wound (Dealey, 2005).

Phase IV – maturation

During the maturation phase the components of the extracellular matrix change and collagen fibres are restructured, becoming thicker and stronger, giving greater tensile strength to the new tissues over time (Hughes, 2002). A newly healed surgical wound has little tensile strength, but will gradually increase to about 50% of the normal tissue after 3 months (Forester *et al.*, 1970). Collagen is constantly degraded and new collagen synthesised. Remodelling of the collagen fibres is a lengthy process that continues for months or years after healing, allowing the wound to continue to strengthen (Moore, 2003). Scars usually flatten and soften and eventually fade (Hughes, 2002). Abnormal scarring may occur if collagen production occurs at a greater rate than its destruction (Smith, 2005).

ACUTE AND CHRONIC WOUNDS

An acute wound is one that heals within a relatively short time frame without complications. The healing process is automatically triggered into action and destroyed tissue is replace by living tissue. A wound

becomes chronic when there is failure of the normal processes of healing, as a result of various underlying problems and pathological conditions. A chronic wound is slow to heal taking months or even years. Falanga (2002) describes chronic wounds as having a complex life of their own.

Wound exudate

Studies on wound exudate have established differences between acute wound fluid and chronic wound fluid. The production of wound exudate is a normal part of the inflammatory process (Thomas, 1997). Wound fluid from acute wounds has beneficial properties that have been shown to stimulate cell proliferation and thus plays an essential part in the healing process (Katz *et al.*, 1991; Chen *et al.*, 1992). Chronic wounds have a prolonged inflammatory response leading to increased and prolonged production of wound exudate that interferes with healing (Moore, 1999; Hart, 2002). Wound fluid from chronic wounds has been shown to have a damaging effect on healing due to sustained high levels of tissue-destructive enzymes (Trengove *et al.*, 1999; Krishnamoorthy *et al.*, 2001). In a normal healing acute wound, high levels of protease activity, which is responsible for clearing the debris from the wound, decrease as the wound heals, whereas in chronic wound fluid protease activity remains increased (Wysocki *et al.*, 1993; Yager & Nwomeh, 1999). Research suggests that wound exudate in non-healing chronic wounds has a damaging effect on wound healing because of the impaired proliferation of key cells in the healing process (Hoffman *et al.*, 1998; Trengove *et al.*, 1999; Krishnamoorthy *et al.*, 2001;