The Royal Marsden Hospital
Handbook
of Wound Management
in Cancer Care

Wayne Naylor
BSC, DIP NURSING, RN, ONC CERT
Wound Management Research Nurse
Directorate of Nursing, Rehabilitation and Quality Assurance
The Royal Marsden Hospital

Diane Laverty
BSC, RGN, ONC CERT
Clinical Nurse Specialist Palliative Care
Directorate of Nursing, Rehabilitation and Quality Assurance
The Royal Marsden Hospital

Jane Mallett
PHD, MSC, BSC, RGN
Nursing and Rehabilitation Research and Development Manager
Directorate of Nursing, Rehabilitation and Quality Assurance
The Royal Marsden Hospital
The Royal Marsden Hospital
Handbook
of Wound Management
in Cancer Care

Wayne Naylor
BSC, DIP NURSING, RN, ONC CERT
Wound Management Research Nurse
Directorate of Nursing, Rehabilitation and Quality Assurance
The Royal Marsden Hospital

Diane Laverty
BSC, RGN, ONC CERT
Clinical Nurse Specialist Palliative Care
Directorate of Nursing, Rehabilitation and Quality Assurance
The Royal Marsden Hospital

Jane Mallett
PHD, MSC, BSC, RGN
Nursing and Rehabilitation Research and Development Manager
Directorate of Nursing, Rehabilitation and Quality Assurance
The Royal Marsden Hospital
Contents

Contributors, viii

Foreword, ix

Acknowledgements, x

Introduction, xi

1 Physiology of Wound Healing, 1
   Structure of the skin and the normal healing process, 1
   Factors that influence wound healing, 15
   Conclusion, 21

2 Wound Assessment, 22
   Principles of assessment, 22
   Assessing the patient with a wound, 25
   Conclusion, 45

3 Management of Specific Wound Types, 46
   Necrotic wounds, 46
   Sloughy wounds, 50
   Granulating and epithelialising wounds, 55
   Infected wounds, 58
   Cavity wounds, 63
   Sinus wounds, 65
   Fistulae, 67
   Conclusion, 72
4 Management of Wounds Related to Cancer and Cancer Therapies, 73
Surgical wounds, 73
Plastic surgery wounds, 75
Skin grafts, 85
Scarring, 90
Graft versus host disease of the skin, 93
Acute skin reactions to radiotherapy, 95
Additional advice for specific skin areas, 101
Fungating wounds, 106
Supply and administration of wound management products, 117
Psychological, sexual and social problems related to chronic wounds, 119
Conclusion, 122

5 A Guide to Wound Management Products, 123
Activated charcoal dressings, 123
Adhesive island dressings, 124
Alginates, 125
Debriding enzymes, 127
Foams, 128
Honey, 132
Hydrocolloids, 133
Hydrogels, 135
Hydrofibre, 138
Larval therapy, 139
Metronidazole gel, 141
Paraffin gauze, 142
Semi-permeable films, 143
Silicone gel sheeting, 144
Contents

Skin barrier films, 146
Sugar paste, 147
Topical negative pressure therapy, 149
Secondary dressings, 151
Tapes, 152
Wound management products to be avoided or for restricted use only, 153
Conclusion, 157

References, 158

Further Reading, 175

Appendix, 177

Index, 199
Contributors

Amanda Baxter  BSc, RN, RMN (Onc Cert)
Clinical Nurse Specialist Pelvic Care (Chapter 3)

Sarah Hart  MSc, BSc, RN, FETC, Onc Cert
Clinical Nurse Specialist Infection Control and Radiation Protection (Chapter 3)

Diane Laverty  BSc, RN, Onc Cert
Clinical Nurse Specialist Palliative Care (Chapters 2, 3, 4 and 5)

Jane Mallett  PhD, MSc, BSc, RN
Nursing and Rehabilitation Research and Development Manager (Chapter 4)

Wayne Naylor  BSc, Dip Nursing, RN, Onc Cert
Wound Management Research Nurse (Chapters 1, 2, 3, 4 and 5)

Caroline Soady  BSc, RN
Clinical Nurse Specialist Head, Neck and Thyroid (Chapters 4 and 5)

Miriam Wood  BSc, RN, Onc Cert
Informatics Project Nurse (Chapter 2)

(All at The Royal Marsden Hospital)

PREVIOUS CONTRIBUTORS (to The Royal Marsden Hospital wound management guidelines)

Jane Mulholland, formerly Principal Pharmacist, The Royal Marsden Hospital

Deborah Fenlon, Lecturer, Centre for Cancer and Palliative Care Studies

Frances Fuller, formerly Clinical Nurse Specialist Lung Cancer, The Royal Marsden Hospital
I have a confession to make! I can now openly admit that early in my clinical career as a Registered Nurse my knowledge of wound management was woefully inadequate. I clearly remember being aware of my limitations in this area at that time. During those first years of my professional practice I gradually cobbled together a somewhat haphazard knowledge base from study days, reading articles and pestering the Ward Sister to help me. I think that eventually I developed a reasonable level of clinical expertise in wound management. However, wound management is a core nursing skill and all patients should be afforded up-to-date and competent nursing care of their wounds. An evidence-based resource like The Royal Marsden Hospital Handbook of Wound Management in Cancer Care would have been enormously useful to me and would have improved the care I offered to my patients. With this in mind, I feel that this work will be welcomed by nurses and other members of the multidisciplinary team who need to find up-to-date and accurate information on wound management quickly and easily.

This Handbook has been published in 2001, a year marking the 150th anniversary of The Royal Marsden Hospital, which opened in 1851 as the first specialist cancer hospital in the world. The sharing of good nursing practice is recognised as an important part of our work and it is certainly a core commitment for the future. This Handbook is an excellent example of that commitment.

My personal experience as a clinical nurse certainly influenced my decision to support the work that produced this Handbook and, in particular, to seek funding from our most generous supporters at The Royal Marsden to complete this project. I would like to take this opportunity to thank the Editors, Wayne Naylor, Diane Laverty and Jane Mallett, for their tenacity, persistence and hard work.

Dickon Weir-Hughes
Chief Nurse and Director of Quality Assurance
The Royal Marsden Hospital (London & Surrey)
Acknowledgements

Dr Karen Broadley, Consultant, Palliative Medicine, The Royal Marsden Hospital
Meriel Ball, Senior Staff Nurse, Weston Ward, The Royal Marsden Hospital
Tracey Cricket, Radiographer, The Royal Marsden Hospital
Dr Janet Hardy, Consultant, Palliative Care, The Royal Marsden Hospital
Rachel Mead, Sister, Ellis Ward, The Royal Marsden Hospital
Sandy Miller, Senior Staff Nurse Outpatient Department, The Royal Marsden Hospital
Diane O’Connell, formerly Sister, Outpatient Department, The Royal Marsden Hospital
Vina Patel, Senior Staff Nurse, Outpatient Department, The Royal Marsden Hospital
Karen Summerville, formerly Clinical Nurse Specialist, The Royal Marsden Hospital
Ann and Tony Rose, The Monte Challenge
Dr Tamara Fishman, Podiatric Wound Consultant, Primary Foot Care Center Inc., Florida, USA
Sara Allen, Sales Representative, Johnson & Johnson Medical, Berkshire, UK

The Royal Marsden Hospital League of Friends (London) has generously supported the post of Wound Management Research Nurse. This has enabled the development of this Handbook along with many other wound management initiatives within The Royal Marsden Hospital. The Editors, on behalf of The Royal Marsden Hospital, would like to thank the League of Friends for their significant contribution to ensuring best practice in the care of oncology patients with chronic and/or complex wounds.

The Editors would like to thank all of those patients and carers who kindly gave permission for us to take and use many of the photographs that illustrate this book. We would also like to thank the two reviewers, Dr Steve Thomas and Ms Judith Coleman, for their insightful and very useful comments, and the Medical Photography Departments at the Chelsea and Sutton sites of the hospital for their hard work in preparing slides and photographs for use in this book.
Introduction

AIM

The Royal Marsden Hospital Handbook of Wound Management in Cancer Care has been developed to promote evidence-based, cost-effective management of wounds.

BACKGROUND

The Handbook has been developed from published research findings and professional opinion papers as well as the accumulated experience of an interdisciplinary team, including nurses, pharmacists and doctors with expertise in wound care, and the Wound Management Group at The Royal Marsden Hospital. The Handbook has been developed for use by both community and hospital-based nurses, particularly those providing care for oncology patients with complex and/or chronic wounds. However, it is anticipated that the Handbook will also be a useful resource for other health care professionals and pharmacists who require guidance and information on wound management. Health care professionals working in clinical areas other than oncology will also be able to utilise the Handbook when wounds require skilled management. While this book is intended to assist nursing practice and is based on the best available evidence, individual patient circumstances and professional judgement should be taken into account when planning patient care in order to provide a high quality and co-ordinated approach to wound management.

The Handbook provides a detailed outline of the management of specific wound symptoms that may appear either in isolation or in combination, including wound necrosis and infection. In addition, the handbook also incorporates the care of wounds that occur as a result of cancer or its treatments. This includes fungating wounds, acute skin reactions to radiotherapy, the management of graft versus host disease of the skin and plastic surgery wounds. To facilitate cost-effectiveness, particular wound care products are identified to manage different symptoms.

The Handbook has been designed to be used in conjunction with The Royal Marsden Hospital Manual of Clinical Nursing Procedures (Mallett & Dougherty 2000) and Patient Group Directions for nurse supply and administration of medicines developed within The Royal Marsden Hospital (Laverty et al. 1997, Mallett
et al. 1997, Mallett & Dougherty 2000). Used together, these resources will ensure the highest quality holistic management for patients with simple or complex wounds (Mallett et al. 1999).

While this book provides detailed information on many different methods of wound management, health care professionals are reminded of the need to carry out patient care according to their level of competency and within their Scope of Practice (UKCC 1992). Finally, every effort has been made in the writing of this book to present accurate and up-to-date information from the best and most reliable sources. However, the result of managing patients' wounds depends upon a variety of factors not under the control of the authors. Therefore the authors do not assume responsibility for, nor make any warranty with respect to, the outcomes achieved from the information described therein.
Physiology of Wound Healing

STRUCTURE OF THE SKIN AND THE NORMAL HEALING PROCESS

Introduction
Knowledge of wound healing physiology is not the sole domain of specialist practitioners, it is a basic area of understanding that all health care professionals involved in wound management need to have. By being aware of the phases that a wound progresses through during healing, and the factors that influence this process, the health care professional is able to maintain an effective and appropriate wound management strategy. This chapter looks at the structure and function of the skin, the normal healing process and factors that may have an influence on tissue repair.

Structure and function of the skin
The skin is one of the largest organs of the body. In the average adult the skin weighs about 5kg and covers an area of around 2m² (Tortora & Grabowski 1996). While it is highly valued for its outward appearance, the physiological functions of the skin as a major body organ are often underestimated. It is a highly complex structure that performs a number of very important functions. The skin and its various appendages, hair, glands, nails and nerves, combine to form the integumentary system.

Skin is composed of two main layers, the epidermis and the dermis. Below the dermis is fatty tissue, known as the subcutaneous layer, which is attached to underlying muscle and bone (Collier 1996, Tortora & Grabowski 1996). Figure 1.1 is a diagrammatic representation of the skin and its structures.

The epidermis
The epidermis can be further divided into four or five layers depending on its location on the body (Fig. 1.1). The outer layer is the 'stratum corneum' and consists of dead cells (keratinocytes) filled with keratin. This layer is waterproof and provides protection from bacteria, heat and a number of chemicals (Tortora & Grabowski 1996). The stratum corneum is constantly replaced as cells slough
off through normal wear and tear: the whole layer is renewed approximately every 24 hours (Collier 1996).

In most other areas of the body, the second layer of the epidermis is the ‘stratum granulosum’. The cells in this layer are also flattened and are in various stages of degeneration; however the cells are still active and contain granules of keratin (Ross et al. 1995).

The third layer is the ‘stratum spinosum’, which is several cells thick. This layer contains live cells that are tightly joined together by numerous processes on their cell membranes; the junctions connecting the cells are called desmosomes. Under a light microscope the cell processes look like spines and hence cells in this layer are commonly called prickle cells; they may also be referred to as squamous cells. The cells in this layer become flattened as they move upwards (Ross et al. 1995, Tortora & Grabowski 1996).

The final layer of the epidermis is the ‘stratum basale’ (also called the stratum germinativum). At this level there is a single layer of cuboidal cells that are multiplying and moving upwards to become part of the previously mentioned layers until they finally reach the stratum corneum and are shed. This layer also extends down to line the surface of hair follicles and sweat glands (Ross et al. 1995, Tortora & Grabowski 1996).
When the epidermis is damaged it is the stratum basale that plays a vital role in the generation of new cells to repair the defect (Collier 1996). In the soles of the feet and palms of the hand, there is an extra layer beneath the stratum corneum called the 'stratum lucidum'. This is a specialised layer only found in thick skin and contains dead, flattened cells filled with a substance called eleidin (Tortora & Grabowski 1996). It provides a cushioning effect to reduce surface impact.

Within the epidermis there are four cell types:

1. **Keratinocytes** – these are the predominant cell of the epidermis (approximately 90%) and produce keratin.
2. **Melanocytes** – these are pigment-producing cells found in the stratum basale, they extend projections between cells of the stratum spinosum and transfer granules of melanin to keratinocytes.
3. **Langerhans cells** – these cells arise in the bone marrow and are involved in cell-mediated immune responses in the skin (including contact sensitivities such as allergic dermatitis).
4. **Merkel cells** – also located in the stratum basale, these cells are closely associated with sensory nerve endings and are involved in touch sensation (Ross et al. 1995, Strete 1995, Tortora & Grabowski 1996).

When damage to the skin is only as deep as the epidermal layer, it is able to 'regenerate' itself from cells in the stratum basale, which divide and fill the defect with cells the same as those that were originally there. Therefore the damage is repaired without formation of a scar (Silver 1994, Calvin 1998).

Lying between the epidermis and dermis is a thin acellular layer of protein fibres called the 'basement membrane'. Cells of the stratum basale are adherent to one side of this membrane while the other side is attached to the extracellular matrix of the dermis, thus tightly bonding the epidermis and dermis together (Stocum 1995).

**The dermis**

The dermis is predominantly a connective tissue layer composed of protein fibres called collagen and elastin. The combination of these two fibres gives the dermis a high tensile strength but also flexibility (Bennett & Moody 1995, Tortora & Grabowski 1996). Surrounding these fibres is a complex matrix of dermal proteoglycans that forms a gel-like material called ground substance (Stocum
Physiology of Wound Healing

There are relatively few cells that are normally present in the dermis, however the principal cells are:

- **Fibroblasts** – these cells produce collagen, elastic fibres and ground substance.
- **Macrophages** – these are phagocytic cells important for fighting infection (in their inactivated form they are present in the blood as monocytes and originate in the bone marrow).
- **Mast cells** – these cells are a part of the immune system and release histamine, they are responsible for allergic and hypersensitivity reactions.
- **Adipocytes** – fat cells (Ross *et al.* 1995).

Originating in the dermal layer are the majority of the skin appendages (Fig. 1.1). These accessory structures include:

- **Hair follicles, roots and hair** – hair is found on all parts of the body except for the sides and palms of the hands, sides and soles of the feet, lips and urogenital orifices. The main functions of body hair are protection and thermal regulation;
- **Sebaceous glands** – these are situated at the base of hair follicles and secrete an oily fluid called sebum that travels up hair follicles to the skin. Sebum acts as a waterproofing agent on the skin.
- **Sweat glands** – there are two types of sweat glands. Eccrine glands, present all over the body except the lips, and apocrine glands present only in the axilla, areola, nipple and external genitalia. These glands are a part of the excretion and temperature control systems of the body.
- **Erector pili muscles** – these are tiny muscles attached to the hair follicle and dermis. When they contract they raise hair into a more upright position ('goose-pimples') (Ross *et al.* 1995, Tortora & Grabowski 1996).

As well as these structures, the blood and lymph vessels, and sensory nerve endings of the skin can be found in the dermis. Finger-like projections called dermal papillae extend into the epidermis and contain blood vessels and nerve endings. Hence, the dermis acts as a supporting structure to the epidermis, being responsible for the supply of oxygen and nutrients to this avascular layer (Thomas Hess 1998).

Damage that extends down into the dermis will result in scar formation. The dermis is unable to regenerate itself and any damaged areas are replaced with
avascular connective tissue resulting in a scar that is devoid of any skin appendages (Flanagan 1996, Calvin 1998).

The subcutaneous layer
The subcutaneous layer (or hypodermis) is a loose connective tissue composed of adipose and areolar connective tissues. It plays an important role in temperature regulation and energy storage (Ross et al. 1995). The depth of this layer is dependent on body site, gender and body composition (Bennett & Moody 1995). This layer contains pressure sensitive nerve endings but only has a minimal blood supply (Tortora & Grabowski 1996). Fibres from the dermis extend into the subcutaneous layer, firmly fixing the skin to it. Because of its poor blood supply, subcutaneous tissue is slow to heal if injured.

Functions of the skin
Skin performs a variety of functions and any damage to, or loss of, skin will affect its ability to carry out these functions effectively. The three main functions of the skin are protection, temperature control and sensation (Mortimer 1998).

Protection
The skin acts as a barrier between the outside environment and the body’s internal structures. It does this by preventing the entry of external hazards, such as bacteria and chemicals, and keeping in those substances needed by the body, for example water and electrolytes (Mortimer 1998). The elasticity and toughness of the skin also protects against mechanical injury. Any damage that breaks this barrier acts as a portal for the entry of external material and the loss of internal substances.

Temperature control
Regulation of body temperature is achieved through the skin’s extensive blood supply and large surface area in combination with the production of sweat. In a hot environment the blood vessels of the skin dilate to increase the flow of blood and hence heat loss. The evaporation of sweat from the skin’s surface enhances this process. In a cold environment blood vessels constrict and reduce the circulation to the skin in order to prevent heat loss. Body hair may stand up (by contraction of the erector pili muscles) to keep a layer of warm air next to the skin (Martin 1996).
Sensation
The third main function of the skin is as a sense organ. The skin is sensitive to touch, temperature, pressure and pain. These senses help to inform an individual about their local environment and protect them from harm (Ross et al. 1995).

There are a number of other functions the skin performs; these include the synthesis of vitamin D, absorption of some lipid-soluble compounds and excretion of water and salts as sweat (Collier 1996, Tortora & Grabowski 1996).

The healing process
A wound may be described as any damage to the structure or continuity of an organ or tissue (Martin 1996). It may involve the skin, soft tissues, muscle, bone or other internal structures and organs (Collier 1996). Damage is commonly caused by an external agent and includes cuts, grazes, bruises, punctures and burns. Internal factors may also cause or contribute to the formation of a wound. For example, a minor traumatic wound may deteriorate as a consequence of an underlying disease process such as circulatory disease. This may result in the formation of a venous or arterial ulcer, whereas diabetes may predispose to the development of diabetic ulcers. Although these wounds are often initiated by external trauma to the skin, progression and deterioration are related to the underlying disease process. Malignant tumours are internal agents that may result in the formation of a fungating wound (Haisfield-Wolfe & Rund 1997).

There are three recognised methods of wound healing; these are primary intention, secondary intention and delayed primary, or tertiary intention, healing (Miller & Dyson 1996, Calvin 1998, Laverty et al. 2000a).

Primary intention healing
Healing by primary intention occurs when the wound edges are brought together and kept in place by the use of sutures, clips, glue or adhesive strips. Surgical incisions are the most common type of wound in this category (Fig. 1.2), although traumatic wounds that have been surgically repaired may also be included (Miller & Dyson 1996).

Secondary intention healing
Secondary intention healing takes place when the edges of the wound cannot be easily brought together. Consequently, these wounds are open and must
A laparotomy wound that is healing by primary intention (sutures have been removed).

An open surgical wound on the lower leg healing by secondary intention following incision and drainage of an infected haematoma.

heal from the bottom up until tissue becomes level with the epidermis. When it has reached this height re-epithelialisation can take place (Flanagan 1998). Some surgical wounds may be left open to heal by secondary intention, particularly if there is a large amount of tissue loss, the wound is heavily infected or a better cosmetic or functional result will be achieved through this method of healing. Predominantly however, these wounds are the result of trauma or a disease process such as leg or pressure ulcers (Fig 1.3) (Miller & Dyson 1996, Calvin 1998).

Delayed primary (or tertiary) intention healing
If a wound is heavily infected or contaminated with foreign bodies, it may be treated as an open wound for several days until it is clean or free of infection, and then the edges brought together (as for primary intention healing). This is known as delayed primary intention healing. This method may also be used for wounds with a large amount of tissue loss. In this instance the wound is closed when it has become small enough that the edges can be kept together without undue tension (Sussman 1998).

The phases of wound healing
Whichever way a wound heals it will progress through a number of phases until healing is complete. It is generally accepted that there are three phases in the healing process: inflammation, proliferation and maturation (Miller & Dyson
Physiology of Wound Healing

1996, Calvin 1998, Moore & Foster 1998b, Thomas Hess 1998, Ehrlich 1999). Some authors also recognise a fourth phase that takes place at the time of injury; this phase is often referred to as haemostasis (Flanagan 2000). Although these phases follow a specific sequence, they do not occur as separate, distinct stages but merge together. The length of each phase depends on the type and nature of the wound.

**Haemostasis**

As soon as tissue is injured, any damaged blood vessels will constrict to stem the blood flow. In a further attempt to prevent blood loss, the 'coagulation cascade' is initiated by the release of chemical messengers from platelets that have come in contact with collagen from damaged blood vessel walls (Fig. 1.4a) (Olde Damink & Soeters 1997, Silver 1994, Flanagan 2000). Thrombokinase and thromboplastin stimulate the formation of fibrin, while serotonin and adenosine triphosphate promote platelet aggregation (Johnson 1988a). This results in the formation of a fibrin and platelet 'plug' that traps blood cells to form a blood clot and hold the wound edges together (Fig. 1.4b). As this clot loses moisture it dries to form a scab. A blood clot usually forms within five to 10 minutes of an injury occurring (Johnson 1988a, Moore & Foster 1998b).

**Inflammation**

This phase is sometimes referred to as the destructive phase and is initiated as soon as injury takes place. The release of enzymes from damaged cells results in the breakdown of noradrenaline, which in turn causes dilation of local capillaries and therefore an increased blood flow to the surrounding tissues (Flanagan 1996, Johnson 1988a). Histamine is released from mast cells and increases the permeability of capillaries allowing leakage of fluid into adjacent tissues. Discomfort may occur due to increased pressure on local nerve endings from fluid accumulation. These processes produce the characteristic signs and symptoms of inflammation such as redness, heat, oedema, discomfort and reduced function (Fig. 1.5a) (Flanagan 1999).

Following these initial reactions, the cells involved in healing are attracted to, and start to infiltrate, the damaged area (Fig. 1.5b). Neutrophils arrive in large numbers, their purpose being to attack and destroy any invading bacteria by phagocytosis (ingestion of the foreign body into the cell) to prevent infection (Calvin 1998). As the level of bacterial contamination drops, the predominant
cell becomes the macrophage. These cells also remove debris from the wound through the process of phagocytosis, but their purpose in wound healing is much more complex than this. The macrophage could be referred to as the ‘conductor’ of wound healing as it is present for the rest of the healing process and orchestrates the healing phases (Silver 1994). It does this through the use of