A PROSPECTIVE RANDOMISED STUDY TO DETERMINE THE EFFECTS OF PULSED SHORTWAVE THERAPY, INFRARED RADIATION, LASER AND ULTRAVIOLET RADIATION ON THE HEALING OF OPEN WOUNDS IN THE HAND

by

NIRMALA NAIDOO

B. Sc (Physio) (UDW)

Submitted in partial fulfilment of the requirements for the degree of

MASTER OF MEDICAL SCIENCE

in the

Department of Plastic and Reconstructive Surgery

Faculty of Medicine

University of Natal

Durban

April 2000

ABSTRACT

Traumatic wounds have been with us since the beginning of mankind. Wound care is a significant and costly healthcare problem with implications for patients and healthcare providers alike. Modern research in wound care is being directed at stimulating the complex cellular and humeral natural defence mechanisms rather than encouraging the increasing and widespread use of antibiotics. Wound healing is traditionally performed by nursing staff, essentially including wound lavage and dressings. However, Physiotherapists frequently treat patients with open wounds. The purpose of this study was to investigate the rate of wound healing in patients with open wounds in the hand having adjuvant physiotherapy wound healing modalities versus wound dressings only.

125 Patients with open wounds on the volar aspect of the hand were included in the study. 5 groups, each consisting of 25 patients, were assessed for zone of injury with 5 patients per zone for all groups. Wounds were exposed and soaked in a warm bath for 10-15 minutes with active exercises. Patients then received either pulsed shortwave therapy (Group A), infrared radiation (Group B), laser (Group C) or ultraviolet radiation (Group D). The control group (Group E) received no adjuvant therapy. All patients received a neutral dressing of Jelonet and gauze. Patients were treated three times a week for 4-6 weeks, or until the wounds were healed. Wounds were measured for the rate of healing by serial tracing and photography. Pain was measured using the numerical rating scale.

Data was analysed using repeated measures analysis of variance with least significant difference.

All groups demonstrated wound healing. Groups treated with pulsed shortwave therapy and ultraviolet radiation demonstrated accelerated rate of wound healing compared to those in the infrared, laser and control groups. In addition to the facilitated wound healing, patients in pulsed shortwave therapy (Group A) showed significant improvement in pain.

The rate of healing of open wounds on the volar aspect of the hand is accelerated by physiotherapy treatment. All groups showed an increase in wound healing rate, and following treatment had decreased pain. Patients treated with pulsed shortwave therapy yielded the most rapid wound healing rate and had the best pain response. Patients thus returned earlier to their daily activities and employment.

In this research the statistical planning and analysis was done in consultation with the Institute of Biostatistics of the Medical Research Council.

PREFACE

The experimental work described in this thesis was carried out at The Workmen's Accident and Rehabilitation Centre, Durban and the Department of Plastic and Reconstructive Surgery, University of Natal Medical School, from March 1997 to March 1999.

This study represents original work by the author and has not been submitted in part or whole to any other University. Where use was made of the work of others, it has been duly acknowledged in the text.

NIRMALA NAIDOO

PAPER PRESENTATIONS

Naidoo N, Madaree A

A study to determine the effect of pulsed shortwave therapy on the healing of open wounds in the hand.

Oral presentation at the 28th conference of the South African Society of Hand Surgery, Cape Town, South Africa (September 1997).

Naidoo N, Madaree A, Thistlethwaite A

A study to determine the effect of laser and pulsed shortwave therapy on the healing of open wounds in the hand.

Oral presentation at the 4th International Federation of Societies for Hand Surgery and Hand Therapy, Vancouver, Canada (May 1998).

Naidoo N, Madaree A

A comparative study to determine the effect of pulsed shortwave therapy, ultraviolet radiation, laser and infrared radiation on the healing of open wounds in the hand.
Oral presentation at the 13th conference of the World Confederation for Physical Therapists, Yokohama, Japan (May 1999).

ACKNOWLEDGEMENTS

I would like to express my sincere appreciation to the following people for their contribution to this thesis:

Professor Anil Madaree - Head of the Department of Plastic and Reconstructive Surgery, University of Natal, and my supervisor, for his expert advice and guidance with the study.

W.A.R.C Board and Mr. Osler - the Manager of W.A.R.C., for approving the study to be carried out at W.A.R.C., and also for their financial support.

Angela Thistlethwaite - Physiotherapist-in-Charge at W.A.R.C., (the Boss), for stimulating my interest in hand rehabilitation and for all her support with this research project.

W.A.R.C. Staff - Angie Thistlethwaite, Melphy Mchunu, Pansy Ntwasa, Marlene Juggarnath, Florence Mdlalose, Petros Ngubane (my extended family), for all the laughter and fun we had working together, especially in the dressings room ('Niri's Room'- my office!).

All the Patients - who so unhesitantly agreed to participate in the study and without who the study would not have been possible.

Dr Glenda Mathews - Senior Lecturer, Department of Mathematical Statistics, University of Natal, for her expert and speedy assistance with the data analysis for the paper presentations.

Professor P Gounden - Head of the Department of Physiotherapy, University of Durban-Westville, for allowing me the opportunity to complete this study, especially toward the bitter end!

Wound Healing Society - for the financial support of this study.

Adcock- Ingram Critical Care - for approval of the purchase of all the saline used in the study, at cost.

Smith and Nephew - for the donation of jelonet wound dressings used in the study.

3 M Medical - for the donation of Tegaderm.

Ballamah Naidoo - my mother, for her encouragement and for always being there for our girls.

CC Naidoo - my father, for his guidance.

Richard Naidoo - my husband, for his unwaivering love, encouragement and support.

Kimera and Narina - my angels, for never failing to remind me that I am a mother too!

ABBREVIATIONS

AIDS	**auto-immune deficiency syndrome**
cm	**centimetres**
cm^2	**square centimetres**
DIPJ	**distal interphalangeal**
DNA	**deoxyribonucleic acid**
E_1	**first degree erythema**
E_2	**second degree erythema**
E_3	**third degree erythema**
E_4	**fourth degree erythema**
FROM	**full range of motion**
GaAlAs	**galium- aluminium-arsenide**
Hb	**haemoglobin**
HIV	**human immuno-deficiency virus**
IRR	**infrared radiation**
J	**Joules**
laser	**Light amplification by the stimulated emission of radiation**
(L)	**left**
Mhz	**megahertz**
MCPJ	**metacarpophalangeal joint**
mm	**millimetres**
mg	**milligrams**
mv	**millivolts**
nm	**nanometres**
no	**number**
NRS	**numerical rating scale**
pts	**patients**

%	percentage
PIPJ	proximal interphalangeal joint
PST	pulsed shortwave therapy
ROM	range of motion
(R)	right
secs	seconds
std. dev	standard deviation
W	watts
WH	wound healing
UVR	ultraviolet radiaton
WARC	Workmen's Accident and Rehabilitation Centre

LIST OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF PLATES

APPENDICES

CHAPTER 1

1. INTRODUCTION

The power of the hand is demonstrated by its ingenious design and construction. It is an efficient tool which we use in many different ways to earn our daily bread and with which we perform a myriad of mundane daily tasks. It is a delicate sensory instrument with which we can judge shape, size, temperature and texture. As Wing-Commander C.B. Wynn Parry writes in the introduction to his distinguished work on *Rehabilitation of the Hand* : 'Only those who have not worked with patients whose hands are seriously disabled do not realise how deep the disaster may penetrate, and how much psychological trauma, often not manifest, can be caused'(Rose, 1968).

Wound care is a significant and costly healthcare problem with implications for patients and healthcare providers alike. Consequently, it is imperative to devise treatment methods for the acceleration of wound healing that are efficacious, cost-effective, easy to apply and one that provides pain relief. Wound healing is a complex process by which all wounds heal in the same sequence of events (Kloth, McCulloch and Feedar, 1990). A combination of vascular responses, cellular and chemical activity, and release of chemical mediators within the wounded tissues are inherent, interrelated components of the healing process.

The basic biology of wound healing should provide a foundation upon which new insights and technological advances can be based to optimise and accelerate the regeneration of injured tissue in the wound repair process. Health professionals involved in wound care must have a thorough knowledge of the patient's medical history, including nutritional status, medications and prescribed treatment regimens for related medical problems.

Understanding the normal cellular activity of wound repair and regeneration is critical to accurate wound assessment, which in turn determines successful wound treatment. The key issue in wound management is understanding the physiological effect of our actions, be they debridement, cleansing, disinfection, dressing, or the use of modalities or motion on the natural response of wound healing. These treatments all contribute, either positively or negatively, to that cellular response (Evans, 1980).

1.1 History of wound management

Traumatic wounds have been with us since the beginning of mankind. Plants, animal parts, excreta and mud were just a few of the early covering agents for traumatic wounds. The first written records of wound management come from the ancient Egyptian papyri (Majno, 1982). The first documented attempt to control medical care through punitive measures was found in Babylon; the written Code of King Hammurabi (1900 BC) mandated amputation of the physician's hands should the patient die from attempted wound management. Subsequently, the Greeks and then the Romans made important contributions to wound care. Hippocrates, the 'father of medicine,' cleansed open wounds with boiling or filtered water. He believed that wound edges should be kept dry and brought together as closely as possible to allow for healing by first intention.

These early approaches to wound care were changed for thousands of years by the misinterpretation of the teachings of Claudius Galen, who believed that the wound was healed when it discharged pus. Ambrose Pare in the 1500s, was the first to rediscover gentle care in the management of traumatic wounds after observing that burning the wounds of gunshot victims seemed to aggravate the wounds. In the 19th century, Semmelweis advocated hand-washing in chlorinated water. Together with Pasteur and Lister, Semmelweis revealed that sepsis and repair were separate phenomena.

Modern research in wound care is being directed at stimulating the complex cellular and humoral natural defence mechanisms rather than encouraging the increasing and widespread use of antibiotics.

1.2 Types of wound healing

Healing of wounds can be divided into 3 types (Figure 1):

1.2.1 Healing by first intention (primary repair)

This involves the primary suture and healing of an aseptic, accurately apposed, incised wound. An example is a healed sutured incision.

1.2.2 Healing by second intention (secondary repair)

Healing occurs by granulation in wounds where primary union fails because of excessive trauma or tissue loss, infection, or because the wound surfaces have not been brought together.

1.2.3 Healing by third intention (delayed primary repair)

If a deep wound has either not been sutured primarily, or later breaks down and then is sutured or resutured several days later when granulation is present, two apposing granulating surfaces are brought together. The result is a wider and deeper scar than is the case with healing by first intention. The amount of eventual scar is a direct function of the amount of granulation tissue that is formed (Harkins, 1965).

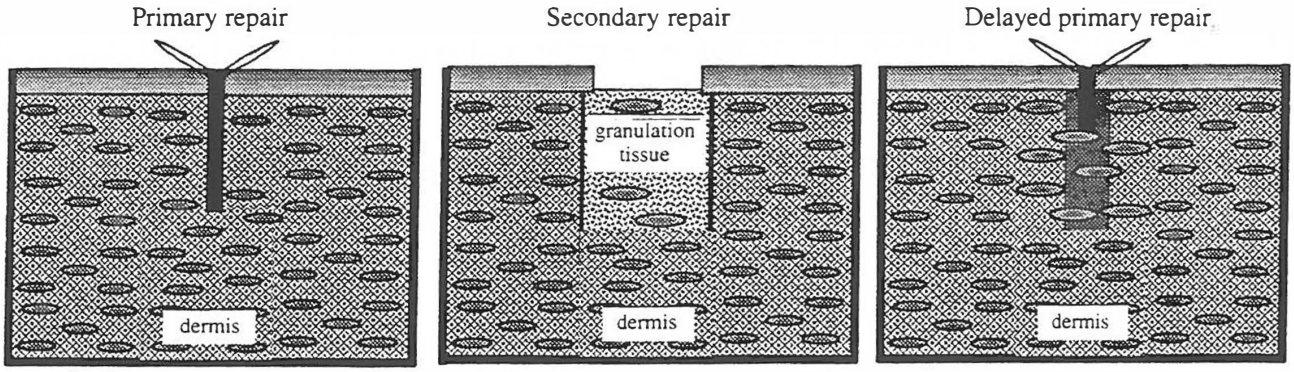


Figure 1 : Three types of wound healing
(Adapted from Harkins, 1965)

1.3 Pathophysiology of wound healing

A wound may be defined as an interruption of tissue to a greater or lesser extent, which may affect skin, mucosa, or organs. The specific sequence of different processes following wounding has one common aim of repair. This is achieved by very complex and dynamic procedures, in which material is degraded and newly synthesised. Wound healing includes aspects concerning certain cell types, biochemical conditions, localisation and time. In every wound type the healing process runs through three stages (Figures 2 and 3), which partly overlap (Hatz 1994).

The overlapping phases in the wound healing process are :-

1.3.1 Inflammatory phase

1.3.2 Migratory phase

1.3.3 Proliferative phase

1.3.4 Maturation phase

More recently, the role of cytokines and growth factors have been investigated intensively for their mechanism in wound healing. Their activity may be influenced by electrotherapy modalities.

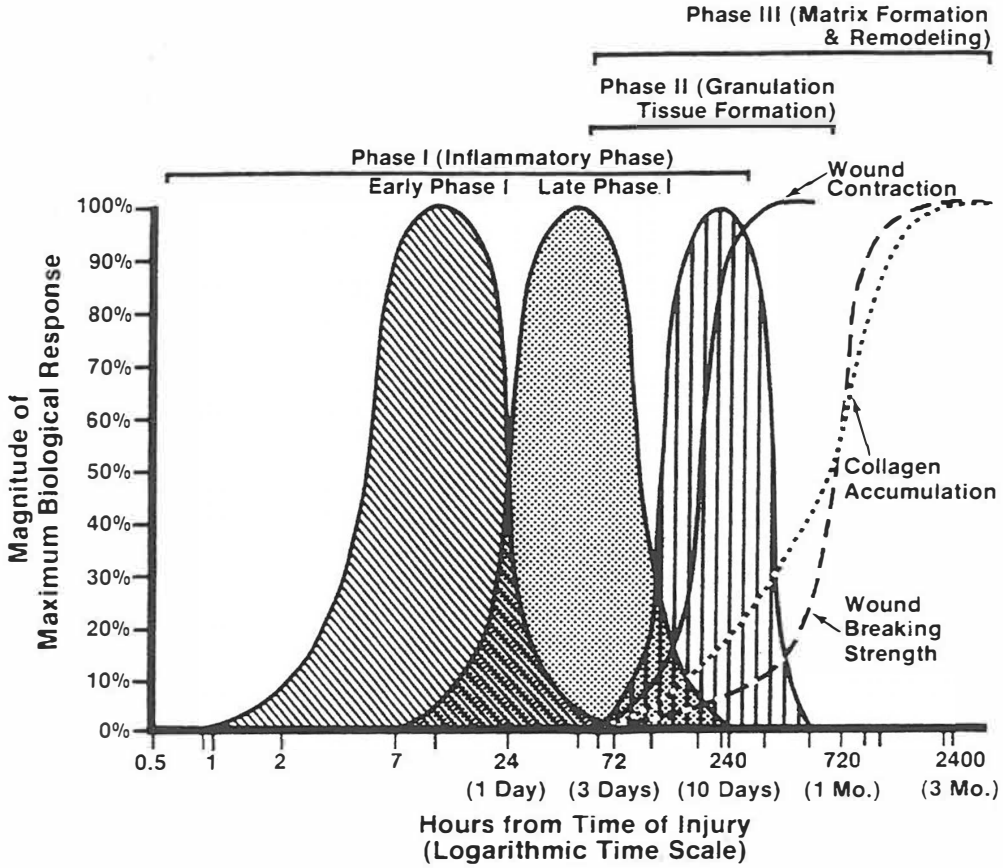


Figure 2 : **Diagrammatic representation of the overlapping phases of wound repair within the first three months following injury**
(Adapted from Kloth *et al*, 1990)

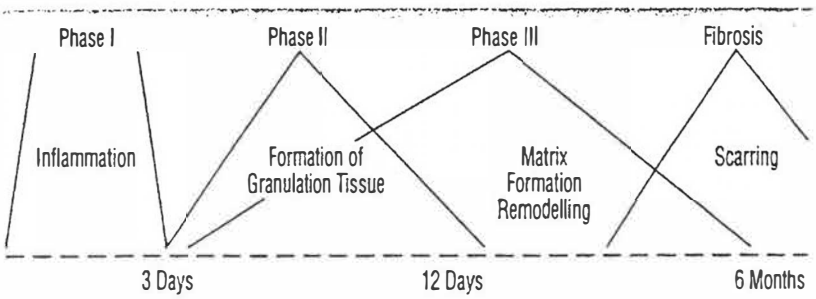


Figure 3 : **Diagrammatic representation of wound healing over a period of 6 months, including fibrosis and scarring**
(Adapted from Hatz *et al*, 1994)

1.3.1 Inflammatory phase

The initial healing reaction to open wounds, the vascular and cellular responses, is the inflammatory phase. This phase is characterised by the formation of clots from platelet plugs and fibrin, local vasoconstriction, wound margin vasodilatation, an influx of neutrophils, and the release of numerous vasoactive mediators. Local vasodilatation, fluid leakage into the extravascular space, and blocking of lymphatic drainage produce redness, swelling and heat. Pain is produced by distension of tissue spaces from swelling and pressure and by chemical irritation of nociceptors. In a new wound, the acute inflammatory response usually lasts between 24 and 48 hours and is completed after two weeks (Zarro 1986). Within the first phase of wound repair cellular processes are determined by immigrating inflammatory cells (after 2-4 hours) and fibroblasts (after 32 hours). Neutrophilic granulocytes and macrophages, which are satellites of the immune system, infiltrate the wound. The inflammatory phase is divided into an early and a late period (Hatz *et al,* 1994). During the first 6 hours, granulocytes migrate into the wound site. As early as 1 hour after wounding, there is increased adherence of granulocytes to the local endothelium. They infiltrate the wound by migrating between endothelial cells and breaking down the basal membrane, attracted by a number of chemotactic substances which result from platelet aggregation and activation of the coagulation system.

The acute inflammation is followed by a subacute phase which lasts approximately 2 weeks. In infected wounds, granulocytes continuously migrate, which obviously prolongs the first phase of repair, leading to marked delay in wound healing. If the wound healing process is delayed in its inflammatory phase, signs of inflammation such as tenderness, erythema, oedema and warmth can be seen. Chronic inflammation may last for months or years and may occur from unresolved acute inflammation. Repeated microtrauma or persistent irritation from the presence of foreign substance in the tissues also contribute to chronic inflammation. Oxygen supply is crucial for progress and quality in wound healing.

The primary goal of biological repair is the termination of blood loss. Platelets adhere to freshly exposed tissue components, such as collagen, and aggregate and by forming a plug, lead to coagulation. Platelets and damaged cells release various mediators activating an entire cascade of events finally transforming fibrinogen to fibrin.

1.3.2 Migratory phase

The migratory phase is characterised by the migration of macrophages and fibroblasts so that wound healing may proceed. The gel-like matrix which forms contains a newly formed vascular network which nourishes the macrophages and fibroblasts that have migrated to the defect in the tissues. Lymphatics also form in this matrix to control wound oedema and infection. Neural tissue may also generate along with lymphatic vessels and the new vascular plexus. The macrophages function as regulators in the late inflammatory phase and by doing so also influence monocyte/macrophage activation. Macrophage function in wound healing is necessary for debridement, stimulation and regulation of the repair sequence. Some weeks after wounding, macrophages are still detectable at the former wound site. As with granulocytes, they produce tissue degrading agents after activation. Overall, macrophages play a prominent role in wound healing processes, and altering their number and function may influence a whole series of regulatory activities.

1.3.3 Proliferative or contraction phase

This phase of wound repair is characterised by proliferation and lasts from day 1 after wounding to a maximum of 14 days (Hatz *et al.*,1994). This phase is characterised by completion of the epithelial phase, collagen synthesis, formation of new capillaries, and an increase in wound strength. Highly vascularised tissue is formed. In addition to leukocytes which are the dominant cells in the inflammatory phase, histocytes, fibrocytes, fibroblasts, plasma cells, mast cells, angioblasts, and myofibroblasts move into the site of the lesion. As a consequence of activated leukocytes adhering to endothelial cells and bradykinin release from the mast cells, vascular permeability increases leading to oedema. Fluid accumulation

is a specific trigger transforming fibrocytes into fibroblasts, inducing cell proliferation, and therefore leading to rapid granulation tissue formation. This dynamic tissue change is fundamental for epithelialisation. Myofibroblasts are modified fibroblasts and contain contractile fibres. Wound contraction, caused by myofibroblasts, occurs at approximately 1-2 mm day and may be the result of contraction itself, fibrosis, adhesions, or other tissue damage (Alvarez, 1987). The wound space is decreased during contraction. Contraction does not always go to completion, except occasionally in small wounds. Contractions usually present functional problems in the healed wound. They allow healing of the wound to proceed to completion, but often prevent normal movement or physiological activity of the injured area. However, wound contraction does not play as decisive a role in the healing process in humans as it does in animals (Hatz *et al.*, 1994).

1.3.4 Maturation or late phase

This phase is the period of remodelling characterised by collagen turnover, increased wound tensile strength, and the formation of blood vessels from capillaries. Connective tissue cells are continually active in the turnover of tissue within the dermis. Open wounds create additional cellular demands for phagocytosis, contraction, generation, and remodelling of new extracellular matrix. A most important connective tissue cell for wound healing is the fibroblast which produces extracellular elements. While the fibroblasts are primarily responsible for fibre production, the interaction of all the connective tissue cells determines the rate of wound healing, and the eventual result and outcome of the wound (Gray, 1980).

During this last phase of wound healing, the production of new connective tissue is of main importance. As soon as fibroblasts synthesise collagen fibrils, their mitotic activity is turned off. Cell density and vascularisation of the wound decrease, while collagen fibrils mature. Scar formation is initiated. Wound disruption strength at this phase is still poor. Three weeks after wounding, the fresh scar has approximately 20% of final strength. Epithelialisation is initiated from the wound margins if all epidermal layers are affected. In superficial wounds, where the basal cell layer is intact, the damaged area can be

reconstructed by mitosis of remaining cells, differentiating into mature epidermis. Re-epithelialisation proceeds with various factors influencing epithelialisation and granulation.

The final step in epidermal wound healing is characterised by cell maturation, leading to regeneration of a defined epidermal layer. The wound proceeds to closure and is covered by mature epidermis. Repeated trauma to the wound surface through dressing changes or flexed skin, interferes with establishing an adequate wound epithelium. In wounds involving the dermal layer, the epidermal cells may develop from small numbers of surviving cells present in the lower parts of hair follicles and sweat glands. This will grow onto the surface of the wound and appear as isolated islands which gradually increase in size and merge. If there is significant loss of tissue and muscle, the survival of these isolated areas of epidermis may be prejudiced by dehydration or an inappropriate method of treatment. When all epidermal elements are lost, without application of a skin graft, resurfacing of the wound can only take place by migration of epithelial cells from the wound margins, and healing is therefore very slow.

Recent studies indicate that the rate of wound repair declines with age. The inflammatory response, proliferative phase and remodelling phase (tertiary bonding to collagen) have all been shown to be retarded with age. The phases of repair tend to start later and proceed more slowly (Hardy, 1989).

1.4 Traumatic wounds

Although some traumatic wounds follow the classic phases of wound healing, the standard phases of wound healing may be substantially altered in a patient with traumatic wounds. The traumatic wound that is left open to close by secondary intention has a prolonged inflammatory, migratory and proliferative phase. The proliferative phase may never terminate, in that complete epithelialisation may not occur. Many conditions and substances, such as hypoxia, ischaemia, infection, dead space, foreign body,. and numerous chemicals have been demonstrated to retard wound healing (Mattox, 1994).

1.5 Formulation of study

The healing of pressure wounds relating to spinal cord injuries and decubitus ulceration has been the subject of many researchers. There is sparse reference in the literature pertaining to any research on the effect of physiotherapy modalities on wound healing. `All studies previously undertaken include only chronic wounds (Nussbaum *et al.*,1994). Other factors relating to chronicity of wounds, for example the time that an ulcer has been present, may influence study results. Wounds which have been present for less than one month may be suitable for acute wound studies (Leaper and Mulder, 1998).

The majority of patients attending the out-patient department at the Workmen's Accident and Rehabilitation Centre, Durban, have the diagnosis of acute crush injury of the hand sustained at the workplace. The study involved the routine treatment of these patients. In addition, the patients were treated with one adjunctive modality for wound healing to compare the effect on the rate of wound healing.

The aim of the study was to establish :

1. the most effective modality of treatment with respect to wound healing in the hand
2. the optimal length of physiotherapy treatment time
3. the most pain-efficient modality of treatment

The hypothesis being proposed is that physiotherapy treatment modalities (pulsed shortwave therapy, infrared radiation, laser and ultraviolet radiation) are efficacious in wound healing and pain relief in open wounds on the volar aspect of the hand.

It is anticipated that the findings of the study would :-

1) establish the most efficient method of treatment of open wounds of the hand by physiotherapy modalities
2) result in efficient use of clinical and treatment time
3) provide evidence that physiotherapy may provide effective treatment of open wounds, and this would therefore broaden the scope of management of patients with open wounds by physiotherapy,
4) impact on the rehabilitation of the hand with regards to activities of daily living and return to work.
5) promote physiotherapy to be included in the multi-disciplinary team in the management of patients with open wounds.

The purpose of the study was to identify and recommend an efficient, economical and effective treatment for open wounds on the volar aspect of the hand. With the ever-increasing promotion of alternate therapeutic approaches to medical care, such as the "laying on of hands" (Bugaj 1982), the treatment of open wounds using physiotherapy measures lends itself most favourably to this process.

As physiotherapists use their hands in so many different ways, in both the assessment and treatment of patients, playing a part in the rehabilitation of the injured or disabled hand is of particular significance.

CHAPTER 2

2. LITERATURE REVIEW

The damage that an injured hand can inflict on the individual and the community is well recognised. The ultimate result of any hand surgery rests on three main factors. These are the quality of the surgical treatment in the early stages; the attitude and determination of the patient and the skilled help and guidance of the rehabilitation experts (Tempest,1980). There are no references in the literature pertaining to any research on the healing of acute wounds of the hand. There is evidence of research on chronic wounds especially related to pressure ulcers, or decubitus ulcers, as seen in spinal cord injured patients. More rapid healing of an acute or chronic wound is significant because it could result in decreased hospitalisation and earlier return of the patient to daily functions. Some wounds take longer to heal and this may contribute to an increased morbidity in these patients. Understanding the fundamentals of wound healing is essential to anticipate and prevent adverse results in this process. As technology and research advances and more care is rendered, the need for therapies aimed at restoring and maintaining structural integrity increases.

2.1 Healing of open wounds

Lazarus *et al.*, (1994) defined a wound as a disruption of normal anatomic structure and function, with healing being a complex dynamic process that results in the restoration of anatomic continuity and function. They characterised an ideally healed wound as one that has returned to normal anatomic structure, function and appearance, while a minimally healed wound has restoration of anatomic continuity, but without a sustained functional result. Wound assessment requires repeated systematic evaluation of wound dimensions.

After an injury, the healed tissue is never the same as it was prior to the injury (Evans,1980).

Epithelium regenerates, but the specialised parts such as the hair and the sweat glands do not. Fibrous connective tissue such as ligament or joint capsule will be replaced, but it will not have the same structure or properties as the original. Moreover, the nerve endorgans do not regenerate. Damaged muscles do not regenerate healing with a scar of fibrous tissue. Serous membranes like synovial joint linings heal well, but there is a lot of exudate in the joint and the fibrin may eventually form a capsular thickening. The general method of healing of soft tissue injuries is by fibrous repair. Fibrous healing will be delayed if there is continued inflammation. Healing also requires a good blood supply, both to support the phagocytes and to supply oxygen and nutrients to the worker cells.

One of the most important factors essential for adequate wound healing is nutritional status. The deleterious effects of malnutrition can be seen in various phases of the wound repair process. An altered inflammatory response can be related to the effects of malnutrition on immune function (Mulder *et al,* 1998). Age is known to affect the repair process and is a consideration when studying wound repair in the surgical and acute wound setting (Leaper and Mulder, 1998). Natural antibodies which aid in humoral immunity have been shown to decrease with age. The failure of any part of the immune and inflammatory responses allows infection to occur and impairs wound healing. The rate of wound dehiscence has been shown to increase 2-3 times in patients over sixty (Mendoza *et al,* 1970).

Drugs such as anti-inflammatory drugs, steroids, and immuno-suppressives have a detrimental effect on wound repair. Tensile strength, as well as healing of open wounds, has been found to be affected by inhibiting several important factors in the repair process, including fibroblast function and collagen synthesis (Mulder *et al,* 1998). Impaired wound healing may be due to local and systemic factors. Systemic diseases by their nature often produce multiple pathological effects.

Vitamin A deficiency was shown to retard wound repair and increase the incidence of wound infection (Brandaleone *et al,* 1941). Experiments in humans have shown that vitamin C deprivation leads to poor wound healing (Hodges *et al,* 1969). Although tobacco smoking is known to result in coronary heart disease, chronic obstructive pulmonary disease, lung cancer and other smoking cancers, many surgeons believe that smoking also impairs wound healing resulting in poor surgical results. Large clinical experiences in several areas of plastic surgery support this suspicion (Chang *et al,* 1996).

2.2 Documentation

For effective monitoring and objective documentation of the status of open wounds, therapists need accurate and practical methods to describe wound size. Several methods of documenting wound size have been reported in the literature. However, not all investigators describe the method they used to determine the surface area of the wounds being measured.

Fischer (1969) took photographs of wounds; such photographs are two dimensional and unipolar, and may produce considerable distortion of three dimensional and multi-planar wound surfaces, particularly when the wounds are located on contoured body surfaces. Documenting wound healing may be incomplete without such a measurement, particularly when wounds are irregularly shaped. Cornwall (1981) calculated surface area by multiplying the dimensions of a wound. The product of the two dimensions of a wound would give an accurate indication of the wound surface area only if the wound is rectangular. Fergusson and Logan (1961) reported a method used by physiotherapists that entailed tracing ulcers on transparent paper, and counting the number of square centimetres within the tracing.

Ramirez *et al.*, (1961) preferred to take photographs of open wounds and to use a planimeter to determine the wound area from the photographs. Howes (1943) projected photographs onto paper and cut out the shape of the wound from an outline he had made

on the paper. By weighing the shapes, he was able to document the extent of wound healing. Bohannon and Pfaller (1983) investigated the practicality and accuracy of three methods of documentation of wound surface area, namely, graph paper counting, weighing and planimeter techniques to determine the area of wound perimeter tracings. Tracings were made by placing sterilised transparency film over the wound and tracing the perimeter of the wound on the film with a fine-tipped transparency marker. The practicality of the three methods depends on what equipment is available to the clinician. The transparent film and marker are of negligible cost and can be used even in determining the area of irregularly shaped wounds. Metric graph paper is also of negligible cost. However, counting the number of square millimetres within a tracing can be a tedious task. Cutting and tracing from the film to transfer the outline to the graph paper may affect the accuracy of the method. The weighing method involves cutting and tracing from the film and requires access to a precise scale. The planimetry method requires access to a planimeter, which can be expensive.

The authors concluded that tracing wounds on clear plastic film provides a viable option for recording wound surface area. The wound area can be determined by a graph paper count, planimetry, or weighing technique. All methods are accurate for confirming known areas. By documenting surface area, it has been possible to establish goals for healing, as well as to assess goal attainment.

2.3 Wound dressings

During the last two decades, many new wound dressings have been developed and marketed for wound care management. Improved technology, research and design of these dressings have yielded a variety of dressings. These dressings can aid in producing a micro-environment that is conducive and supportive to the body's own healing mechanisms, which enable wound repair to occur more efficiently.

The dressing materials that were used throughout the first half of this century were designed with the intention of absorbing and removing all traces of exudate. This philosophy was challenged by Winter (1962) and Hinman *et al* (1963) who demonstrated, both in humans and animals, that wounds which were kept moist healed more rapidly than those left exposed to the air. In dry conditions, the wound has been shown to dehydrate, producing a scab (or eschar) which consists of a dry serous exudate and a layer of devitalised dermis. This scar forms a natural barrier to migrating epidermal cells, forcing them to move deeper beneath the drying eschar, prolonging the healing time and causing an unnecessary loss of healthy tissue (Winter, 1962).

Occlusive dressings occlude the wound and allow moist healing to occur. Wounds covered by an occlusive dressing do not form a scab, so epidermal cells are able to move rapidly over the surface of the dermis, through the exudate which collects at the wound/dressing interface. The application of an occlusive dressing to a wound can also be important in preventing secondary damage as a result of dehydration. The capacity of a deep dermal wound to undergo spontaneous healing depends on the survival of epidermal cells in hair follicles and sweat glands in the base of the wound. If these are allowed to become dehydrated and devitalised, the wound will not heal readily and may have to be grafted.

2.3.1 Bandages

Bandages have a history stretching back thousands of years. The ancient Egyptians, in particular, were skilled in their use, often coating simple woven fabrics with adhesives, resins and other medicinal extracts to aid wound healing. The techniques of bandaging forms an important component in the management of wounds. Bandages vary in structure and performance, and have the functions of dressing retention, support and compression; examples are the cotton conforming bandages and cohesive bandages. The ability of a

bandage to provide dressing retention, support and/or compression is largely determined by its elasticity, although thickness, weight and comfort are important.

Bandages whose primary function is one of support, should have a degree of extensibility to facilitate application and encourage as much active range of motion as necessary. Cohesive bandages combine some of the characteristics of ordinary stretch bandages with those of adhesive products. Whilst they do not adhere to the skin, a special coating on their surface enables the bandages to adhere to themselves, which prevents them from becoming undone under normal conditions of use.

2.3.2 Moist dressings

The use of moist dressings has proved so successful that it is now accepted philosophy in the care of open wounds. Because these dressings all maintain tissue hydration, they are also referred to as occlusive dressings. The importance of preventing wound tissue desiccation in relation to granulation tissue formation and re-epithelialisation is stressed. According to Scotts (1986), efficacious methods of treating pressure ulcers require a moist environment with adequate protection from external irritants. These dressings require frequent changing to maintain moisture. Collwell *et al.* (1993) compared the efficacy and cost-effectiveness of moist gauze dressings and a hydrocolloid wafer dressing and found that there was no statistically significant difference between the two treatment groups, with the moist dressing being the more cost-effective.

Most dressings exhibit some tendency to adhere to the surface of a drying wound. This adherence may be due to the viscosity of the serum itself, but more often it is caused by the penetration into the body of the dressing of blood and exudate, which subsequently dries and hardens to form a scab that incorporates the fibres or threads of the dressing. When the dressing is removed, the bond between the eschar and its underlying tissue will fail, resulting in damage to the newly formed epithelium. If a fabric with an open structure is placed on a wound, granulation tissue can grow through the dressing, so that the material

effectively becomes part of the healing wound. If this occurs, removal of the dressing will result in damage of the healing tissue and thereby delay healing (Thomas 1982).

The adherent properties of four different dressings were described by Malone in 1987 following a study of 40 patients who had an ingrowing big toe-nail removed. The wounds were treated with either a paraffin gauze dressing, a knitted viscose dressing, a hydrocolloid dressing, or a silicone polymer foam. Of the 10 wounds dressed with knitted viscose, 8 bled on removal of the dressing, and 7 of the patients treated with this material complained of pain. No bleeding resulted from the removal of the paraffin gauze, but 5 patients complained of pain. One patient complained of pain on removal of the hydrocolloid, but no pain was reported with the use of the silicone foam.

Pressure ulcers require dressings to maintain their physiological integrity. An ideal dressing should protect the wound, be biocompatible and provide ideal hydration. The cardinal rule is to keep the wound tissue moist and the surrounding intact skin dry. Several investigators studied pressure ulcer healing outcomes by comparing dry wound healing techniques with moist ones. Kurzuk-Howard *et al.* (1985) compared heat lamp treatments with film dressings, and Saydak (1990) compared dry gauze dressings with absorptive powder treatment. Several investigators compared wet-to-dry gauze dressings with various moist wound-healing alternatives (Gorse and Messner, 1987; Sebern, 1986). The results of these studies suggest that the rate of healing is better with the moist wound healing treatment than with dressings or treatments that dry the wound bed.

The normal antibacterial mechanisms present in any wound are able to continue functioning normally by keeping the wound moist. In particular, white blood cells which might otherwise have dehydrated, remain present and functional (Gilchrist and Hutchinson, 1990). It is also likely that in the presence of moisture, natural enzymes such as lysosomes are able to remain functional, thus assisting in autolysis of necrotic debris. In addition, it is probable that antibodies produced by the immune system would assist in the destruction of

potentially harmful bacteria. The importance of preventing wound tissue desiccation in relation to granulation tissue formation and re-epithelialisation is stressed.

2.4 Bacteriology

The skin naturally acts as a barrier to infection, both actively and passively. Sebum, the secretion of the sebaceous glands of the skin, not only acts as a lubricant, but actually destroys streptococci. Any trauma to the skin inactivates sebum through the accumulation of serum. This can lead to an overgrowth of streptococci (Hunt and Dunphy, 1979). Lowden (1974) revealed that the commonest organisms invading the hand and causing septic lesions are the haemolytic streptococcus and *Staphylococcus aureus*. The former tends to give rise to rapidly spreading infections involving the cellular tissues extensively, and inflaming the lymphatic channels and regional lymph glands at an early stage. The *Staphylococcus aureus* set up a local reaction which tends to limit spread. In recent years there have been important changes in the clinical problems set by infections in the hand. These changes are related to the use of antibiotics, the tendency to seek treatment at an early stage, more care in the prophylactic treatment of minor injuries, and increased appreciation of their importance in hospital clinics and surgical units.

The isolation of micro-organisms from a wound is not of itself an indication of the presence of infection, as wounds of all types can rapidly acquire bacteria from any one of a number of sources (Thomas 1990). Such contamination may result from contact with infected or contaminated objects, the ingress of dirt or dust (either at the time of injury or later), or from the patient's own skin or gastro-intestinal tract. The consequences of contamination of a wound will depend on a number of factors: these include the number of organisms, their pathogenicity, and the ability of the patient's own defence system to combat any possible infection. The latter in turn may depend on the patient's age, general health and nutritional status, and other factors such as the administration of immuno-suppressive drugs, which may inhibit the production of leucocytes.

Many wounds may yield a variety of organisms on microbiological investigation, but may never show the classical symptoms of infection, that is, redness and swelling with heat and pain. This was described by Celsus more than two thousand years ago. Lawrence (1985) in a series of publications on the effects of bacteria on burns and wound healing, described the techniques available for detecting and quantifying the number of bacteria present in a wound , and outlined changes in the types of organism that have been isolated from infected wounds over a thirty -year period. The most common pathogen to be isolated from wounds of all types is *Staphylococcus aureus* . This organism, which is found in the nose of 20-30% of normal persons, may be isolated from approximately one-third of all infected wounds.

Other organisms that can cause serious wound infections include *Pseudomonas aeruginosa, Streptococcus pyogenes,* and some *Proteus, Clostridium* and coliform species. Gilliland *et al* (1988) showed that the presence preoperatively of *Pseudomonas* species. and *Staphylococcus aureus* significantly reduced skin graft healing. They also demonstrated that, of 16 ulcers which were slow to heal or which recurred after discharge, 15 (94%) contained *Staphylococcus aureus.* The types of organism present in a given wound may not remain constant, but vary as the condition of the wound itself changes. The number of organisms that might be considered to constitute an infection was discussed by Lawrence (1985).

2.5 Wound debridement

It is commonly accepted that two cornerstones of early wound care are cleansing and debridement. It is believed that the removal of non-viable tissue from the wound bed helps reduce microbes, toxins and other substances that can inhibit wound healing, thereby reducing the inflammatory process and facilitating the wound healing process. This belief has been challenged. Experimental data supporting the efficacy of wound debridement is generally lacking, and clinical trials have failed to consistently show efficacy among commercially available debriding agents (Falabella 1998).

Excessive fibrotic and necrotic tissue as well as debris should be removed from the wound. Surgical debridement has been shown to be optimal in providing a clean wound bed. Natural growth and wound factors may be introduced through fresh bleeding into the debrided wound. Debris and necrotic tissue colonised by pathogenic organisms may also be reduced by aggressive debridement (Knighton *et al*, 1989).

2.6 Wound cleansing agents

Effective wound cleansing is essential if infection is to be avoided and healing is to take place at the optimum rate (Thomas, 1990). In recent years, there has been considerable interest in the effect of hypochlorites and other antiseptic solutions on wound healing. Some studies have shown that hypochlorites have a marked, and in some cases irreversible effect, on the viability of the tissues treated. In a rabbit ear chamber preparation, Eusol and chloramine have been shown to permanently disrupt capillary circulation in granulation tissue and thus cause a significant delay in wound healing (Brennan *et al*, 1985). Further evidence of the adverse effects of chloramine on the healing process was obtained in a second study, in which the antiseptic was applied to a series of standard wounds on the backs of rats (Brennan, 1986). Biochemical and histological examination of the wound site revealed that, compared with the saline control, wounds treated with the hypochlorite solution showed a significant increase in DNA levels, indicative of an increased inflammatory response.

In 1985, Lineweaver *et al.* applied dilutions of four commonly used antiseptics to human fibroblast cells *in vitro*. All the solutions tested (1% povidone-iodine, 0.25% acetic acid, 3% hydrogen peroxide and 0.5% sodium hypochlorite) were found to have marked cytotoxic properties, and with the exception of hydrogen peroxide, were also found to delay wound healing in an animal model. The action of povidone-iodine was particularly marked in this respect. The role of antiseptics and topical agents in wound management has been reviewed previously. It follows that, for routine cleansing of wounds, a sterile solution of 0.9% sodium chloride is probably all that is required. Any benefits that might be derived

from the use of antiseptic agents must be weighed against their possible detrimental effects upon the healing process (Thomas, 1990).

2.7 Pain

Many of the electrical agents and physical treatments applied by the physiotherapist are an attempt to reduce the level of pain perceived by patients. Pain relief is of much current interest and many research articles have been published. Pain is usually considered to be a pathological state in itself by patients (Forster and Palastanger, 1990). Because of the subjective nature of pain, quantification is essential, using some standard scale such as the numerical rating scale (Buck and Paice, 1994).

A variety of instruments for assessing pain is available. Guiffre and associates (1988) suggest that a tool useful for the measurement of pain should meet the following criteria: be easy to understand and use; require little motivation on the part of the subject; be easily scored; require few quantitative terms; be sensitive to fluctuations in the perception being scaled. Whitaker and Warfield (1988) suggested that the numerical rating scale avoids the requirement of mechanical measurement of the point selected by the patient. Jensen *et al.*,(1986) concluded from a study of six pain intensity instruments that the numerical rating scale is the most practical index.

Patients' beliefs about their pain are thought to play a prominent role in pain perception, function and response to treatment (Williams *et al,* 1994). In recent years, increased attention has been placed on the assessment of pain with the advent of specialised scales and methods. One of these methods asks patients to view video tapes of various treatment modalities and to rate the applicability of the modalities to their pain. Patients who believed the treatment was applicable had much better outcomes (Swartz *et al,* 1985 and Shutty *et al,* 1990). Most studies on pain include chronic pain subjects (Bryner 1994).

2.8 Electrotherapy

2.8.1 Pulsed shortwave therapy (PST)

Over the last two decades there has appeared a large amount of published literature relating to pulsed shortwave therapy. Not so widely published is exactly how pulsed shortwave therapy works. Present understanding of its effects must be considered as a hypothesis (Oliver 1984).

In many parts of the world there is increasing interest in the action of pulsed shortwave therapy on living tissues. Studies in Russia, Czechoslovakia, the USA, Canada and Britain have suggested that this form of treatment has a specific biological action in prompting tissue healing. A trial comparing continuous shortwave therapy with pulsed shortwave (Wilson, 1974) on 20 matched pairs of patients with ankle sprains showed clearly superior results in those treated with pulsed shortwave therapy treatment as compared with continuous shortwave therapy in promoting the healing of recently injured soft tissues. It is suggested that the action of pulsed shortwave therapy in promoting the healing of soft tissues is not just a thermal effect, but a more specific biophysical process.

A trial by Bentall and Eckstein (1975) in the use of pulsed high frequency energy therapy on children undergoing orchidopexy concluded that their results further confirmed the benefits of a non-thermal electromagnetic field in the resolution of bruising and increased rate of healing of wounds. Kaplan and Weinstock (1968) stated that pulsed shortwave therapy appears to be an excellent adjunct in the post-operative treatment of foot surgery. This endorsed the views expressed after similar clinical trials by Steinberg (1964) and Braun (1965) and Wilson (1972).

In 1981, Golden *et al* undertook a controlled double blind trial on the clinical behaviour of a standardised wound in humans, when exposed to pulsed shortwave therapy. The wound selected was the donor site of a medium thickness split graft cut. Patients whose donor sites matched the specific criteria were admitted to the trial. Each patient was assigned to one of two groups : the placebo group was treated with a dummy machine and the other

group by a normal Diapuls machine. In the treatment group, 17 out of the 29 patients had wounds which were 90% healed at seven days, compared with only 11 of the 38 in the placebo group. The researchers concluded that approximately twice as many patients were healed in seven days when active treatment was given.

In 1985, Barker *et al* assessed the efficacy of pulsed shortwave therapy in the treatment of lateral ligament sprains of the ankle. A total of 73 patients were randomly allocated to one of two groups and received treatment with a machine which was either functioning normally or was disabled. Their progress was measured by means of goniometry, volumetric measurements, subjective pain scores and gait analysis. The authors concluded that all the quantitative measurements carried out in this trial failed to show a statistically significant difference between the active and the control groups.

2.8.2 Laser

In Europe, the Soviet Union, China and Japan, biostimulation in musculoskeletal and soft-tissue conditions by using the low-energy laser has been greatly touted for decreasing pain and hastening wound repair. In the United States, the concept of biostimulation has been held in less regard (Smith *et al.*, 1992). Mester *et al* (1971) found laser treatment to have "a stimulating effect" on wound healing. Lam *et al* (1983) showed that lasers significantly increase the formation of new capillary paths. Brunner *et al* (1986) determined that the weight of granulation tissue was 10 to 25 percent higher in laser treated wounds than in controls.

It seems evident from these studies that the low-energy laser stimulates the processes that are important in wound repair. In poorly controlled studies involving humans, the use of low-energy lasers has shown remarkable improvements in healing chronic leg ulcers (Goujon *et al.*, 1985). Schjelderup and Klinikk evaluated the treatment of chronic pain with low power laser and to compare the results of needle acupuncture and laser therapy. 40% of the patients estimated laser more effective than acupuncture, while 10% estimated both

methods equally effective. There was very little difference in the duration of pain relief after laser or needle acupuncture.

Assessment of laser therapy by published case histories is difficult. Although some of the studies are well conducted, they are not easy to reproduce in the normal health care with patients who need a solution to their chronic problems (Trelles *et al.*, 1987).

2.8.3 Ultraviolet radiation (UVR)

Ultraviolet radiation forms part of the electromagnetic spectrum and covers the wavelengths between X-rays and visible light. Actinotherapy, the use of ultraviolet light, has been used as a healing agent for centuries; Hippocrates (460-370 B.C.) and Galen (131-201 A.D.) routinely prescribed sun baths for their patients (Kleinkort *et al.*, 1984). Although ultraviolet irradiation has been used extensively to facilitate wound healing, its value has been questioned (Wilkinson 1968, Fugill 1980).

Regardless of the specific mechanism, ultraviolet irradiation does exhibit a bactericidal effect. In open wounds, the effect is lethal to bacteria, and results in a relatively bacterial-free wound, which heals more rapidly than an infected wound. With the availability of topical antibiotics and other agents, such as the low-energy laser for the treatment of wounds, the use of ultraviolet irradiation is not currently in vogue. However, the capabilities and the historic successes should be understood and appreciated. Ultraviolet light is said to improve the healing of skin and is used for this in many centres especially for pressure sores (Dyson and Suckling, 1978).

2.8.4 Infrared radiation

Various forms of light have been used through the ages to facilitate wound healing. While ultraviolet and infrared irradiation have been traditionally used for wound healing, neither agent has provided consistent results. The future of infrared irradiation in the healing of open wounds is bleak (Kloth *et al.*, 1990). There are no well-controlled studies that indicate that infrared irradiation is an effective adjunct in facilitating the healing process of chronic wounds. More definitive studies need to be done in this area..

2.9 Assessment

Objective measurements provide a foundation for hand rehabilitation efforts. A thorough assessment procedure provides information to predict the rehabilitation potential of the traumatised hand, provides data with which subsequent measurements may be compared, and allows the specialist to plan and evaluate treatment programmes and techniques (Fess, 1995).

Specifications and standards of quality are applied to manufactured goods, but there are many variables to apply strict standards in terms of duration of disability when the hand is injured (Glanville, 1982). Correct assessment is a prerequisite of success in any form of rehabilitation. The patient is the most important member of the rehabilitation team. The continuity of treatment from the time of injury until the completion of rehabilitation is important. The treatment time should occupy as much of the patient's time as is required to rehabilitate him in the shortest time. The rehabilitation team should provide a purposeful and confident atmosphere. The patient must be conditioned to concentrate and to work in earnest in order to reach agreed objectives. In theory, the treatment of a hand disability should continue on the basis of a full-time programme of rehabilitation until full recovery has been achieved or until the best possible result in the circumstances has been achieved. In practice, it is usually necessary to strike a compromise and to allow the patient to return to work at a time when the hand is sufficiently recovered for improvement to continue without full-time treatment (Wynn Parry, 1973).

2.10 Adjunctive therapies

The roles of adjunctive therapies in enhancing pressure ulcer healing have been investigated. The therapies considered include electrical stimulation, hyperbaric oxygen, infrared, ultraviolet and low energy laser irradiation, ultrasound, miscellaneous topical agents (including cytokine growth factors), and systemic drugs other than antibiotics.

Data from five clinical trials, involving a total of 147 patients, support the effectiveness of electrotherapy in enhancing the healing rate of pressure ulcers that have been unresponsive to conventional therapy (Carley and Wainpel, 1985; Feedar, Kloth and Gentzkow, 1991, Griffin *et al,* 1991, Kloth and Feedar. 1988). This finding was consistent across a variety of electrical stimulation protocols. To date, this study has been limited to a small number of research centres.

Clinicians considering electrical stimulation therapy should ensure that they have proper equipment and trained personnel who are following protocols shown to be effective and safe in appropriately designed and properly conducted clinical trials (United States Department of Health and Human Services, 1994). The use of various forms of light (infrared, ultraviolet, and low-energy laser) to promote wound healing has been reported in the literature (Freytes, Fernandes and Fleming, 1965; Kahn 1984; MacKinnon and Cleek, 1984 ; Mester, Mester and Mester, 1985). Controlled clinical trials are generally lacking in this area (US Department of Health and Human Services, 1994). The healing rates of hundreds of healthy surgical wounds have been calculated by Marks *et al (*1983) from which they derived an equation which may be used to predict the likely time for a wound of a given size to heal. Using these equations as a baseline, it is possible to monitor the progress of a wound.

Effective hand therapy must be based on a proper understanding of the functional requirements and the muscles controlling them. It is also pertinent to appreciate that certain of the hand deformities seen are associated with disorganisation of the necessary balanced control of these neuromuscular and soft tissue systems (Backhouse 1970).

It is the privilege of physiotherapists to use the power that lies in their hands to restore the hands of their patients to power (Rose 1968). The late Sir Archibald McIndoe once remarked that "It's not what *you* do with the hand that is important, but what the *patient* does with what you have done."

CHAPTER 3

3. ELECTROTHERAPY

Electrotherapy can be defined as the treatment of patients by electrical means (Low and Reed, 1990). This means that electrical forces are applied to the body bringing about physiological changes for physiotherapeutic purposes. This study included the use of a variety of electrotherapy treatment modalities, namely, pulsed shortwave therapy (PST), light amplification by the stimulated emission of radiation (laser), infrared radiation (IRR) and ultraviolet radiation (UVR) for the treatment of open wounds on the volar aspect of the hand. These are the most commonly used modalities in physiotherapy for wound healing (personal experience), and no previous comparative study has been done.

3.1 Pulsed shortwave therapy (PST)

PST is an electromagnetic radiation and is produced by movement of electrons within the atom. If energy is added to an atom, this can cause an atom to move out to a higher-energy electron shell. When the electron returns to its normal level, energy is released as a pulse of electromagnetic energy.

Since the turn of the century, when the beneficial effects of shortwave therapy were discovered, shortwave therapy has been widely used in physiotherapy because it offered the means to achieve deep thermal effects in tissues and joints. Today, with the advancement of physiotherapy, a change of thinking in the use of heat has evolved. PST can be used in the treatment of open wounds in the various phases of wound healing, with little or no production of heat.

3.1.1 Production of PST

PST is the production of high frequency electromagnetic energy. The source of the high frequency current is an oscillator circuit consisting of a capacitor and inductance whose dimensions are so arranged that they will allow electrons to oscillate at a frequency of exactly 27.12 Mhz (Figure 4).

By incorporating a timing circuit with interruptions of oscillations into a series of very short bursts of energy, any trivial heat generated is dissipated during the long intervals between pulses (Figure 5). The electromagnetic energy influences the ions, molecules, membranes and cells, thus speeding up phagocytic activity, enzymatic activity and restoring cell membrane potential in damaged cells (Evans, 1980).

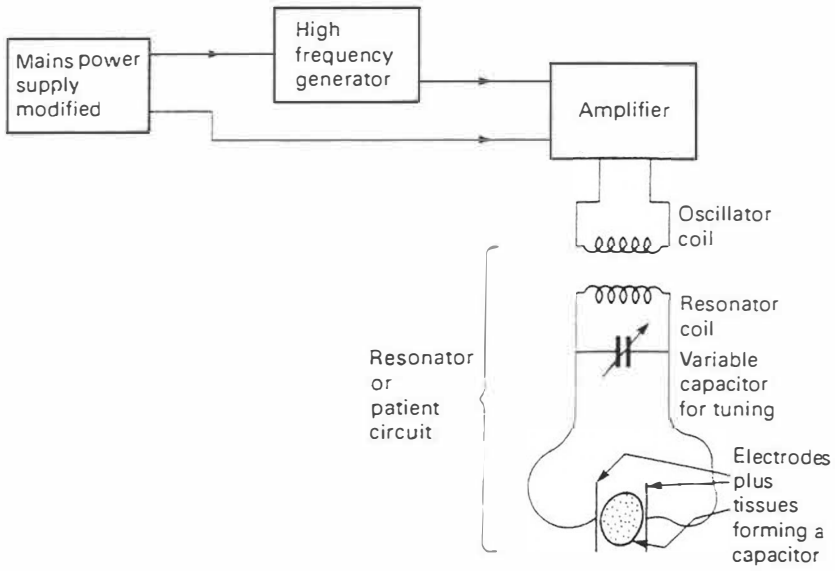


Figure 4: **Block diagram illustrating shortwave therapy generation** (Adapted from Low and Reed, 1990)

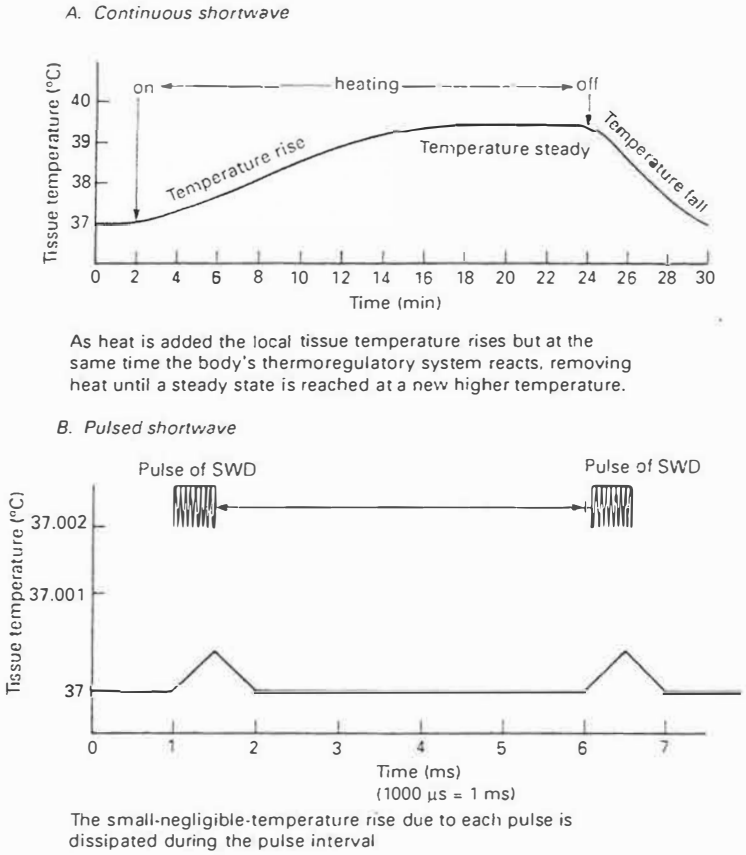


Figure 5 : Diagrammatic representation of shortwave therapy (heat production) and pulsed shortwave therapy (negligible heat production)
(Adapted from Low and Reed, 1990)

All explanations advanced to explain the mechanisms of pulsed shortwave are entirely speculative (Low and Reed, 1990). Some depolarisation of the cell membrane is often associated with cell dysfunction and electrical potentials develop during wound healing. The membrane potential is also involved in the control of cell division and hence in the control of growth, development and repair. It has been proposed that the electromagnetic field could influence the flow of ions through the membrane and therefore restore the normal cell potential in some damaged cells (Hayne, 1984).

An acceptable theory for the effect of PST is the summation theory. Figure 6 illustrates that the non-thermal effects persist longer than heat occurring in the tissue, but since the pulse-repetition frequency is high and the interval between pulses consequently shortened, the heat generated in the tissues will drop to zero. Therefore, when the next pulse is generated, there is still a residual non-thermal effect to which the effect of the second pulse will be added. The negligible heat generated will therefore not accumulate, and hence no temperature increase occurs in the tissue (summating non-thermal effect).

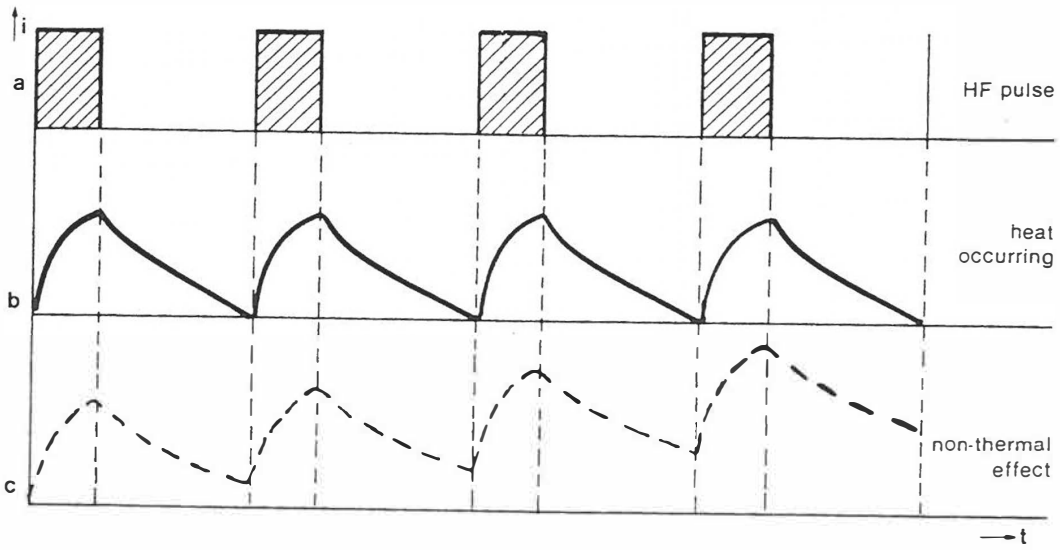


Figure 6 : Effects of pulsed shortwave therapy

(a) five pulses with a certain intensity (i), a certain duration (t) and a very short interval.

(b) increasing thermal effect.

(c) powerfully increasing non-thermal effects.

(Adapted from Meijer *et al*, 1991)

The Curapuls supplies a rectangular pulse with a duration of 0.4 ms. The pulse power (the peak power of the pulse) can be set to a maximum of 1000 W. When PST is used, the aim is to select the highest possible pulse power, while generating as little heat as possible. A low mean power during treatment produces negligible or no heat. The mean power can be

calculated. If, for example, the pulse repetition frequency is 20 Hz, the cycle time (the pulse duration plus the interval) is 1000 : 20 = 50 ms. The percentage of time during which the PST output is present is the 0.4 : 50 = 0.8 %. The mean power for various intensities and pulse-repetition frequencies can be found in Table 1.

Table 1 : Mean power of various pulse-repetition frequencies and pulse powers

	1	2	3	4	5	6	7	8	9	10	Position of intensity control
Pulse-repetition frequency	100W	200W	300W	400W	500W	600W	700W	800W	900W	1000W	Pulse power
15 Hz	0,6	1,2	1,8	2,4	3,0	3,6	4,2	4,8	5,4	6,0	
20 Hz	0,8	1,6	2,4	3,2	4,0	4,8	5,6	6,4	7,2	8,0	
26 Hz	1,0	2,1	3,1	4,2	5,2	6,3	7,3	8,4	9,4	10,4	
35 Hz	1,4	2,8	4,2	5,6	7,0	8,4	9,8	11,2	12,6	14,0	
46 Hz	1,8	3,7	5,5	7,4	9,2	11,0	12,9	14,7	16,7	18,4	
62 Hz	2,5	5,0	7,4	9,0	12,4	14,9	17,4	19,8	22,3	24,8	Mean power
82 Hz	3,3	6,6	9,9	13,2	16,4	19,7	23,0	26,3	29,6	32,8	
110 Hz	4,4	8,8	13,2	17,6	22,0	26,4	30,8	35,2	39,6	44,0	
150 Hz	6,0	12,0	18,0	24,0	30,0	36,0	42,0	48,0	54,0	60,0	
200 Hz	8,0	16,0	24,0	32,0	40,0	48,0	56,0	64,0	72,0	80,0	

(Adapted from Meijer *et al*, 1991)

3.1.2 Physiological effects

The electrical potential across a normal cell membrane is maintained at 60-90 millivolts (mV) due to the relative position of ions on either side of the cell membrane. This potential may be as low as 40 mV in damaged cells (Forster and Palastanga, 1990). It is postulated that when the energy in a pulsed electromagnetic field is applied to a damaged cell, there is a boost to the process to allow the cell membrane potential to return to normal. A normal potential is necessary for the cell to perform its metabolic processes adequately and to increase permeability across the membrane in order that new proteins may be synthesised. This is an essential part of the repair process.

Two main effects of PST have been demonstrated in laboratory experiments, namely, the acceleration of wound healing in animals (Cameron, 1961; Fenn, 1969) and the acceleration of nerve regeneration in rats (Wilson and Jagadeesh,1976; Raji, 1984). A number of controlled clinical studies have demonstrated that PST increases wound healing (Low and Reed, 1990). Various hand injuries have been successfully treated resulting in significant reductions in swelling, disability and pain (Barclay *et al.*, 1983). This was noted during the first 7 days. The study showed that tissues which have a high dielectric constant are good conductors, for example, water and tissues with a high water content.

The main effects of pulsed shortwave therapy include :

1. An increase in the number and activity of cells in the injured region.
2. Resorption of haematoma.
3. Reduced inflammation.
4. Reduced swelling.
5. Increased rate of fibrin deposition and orientation.
6. Increased collagen deposition and organisation.
7. Increased nerve growth and repair.

3.1.3 Therapeutic effects

It seems that PST is an effective treatment for all tissue trauma, both accidental and post-operative. Pain due to a variety of conditions seem to improve with this treatment (Wilson, 1974). Therefore, as already noted, hand injuries could be treated.

3.1.4 Dangers and contraindications

No significant heat is generated by the very short pulses, therefore there is no danger of a burn due to concentration of the field by metal or moisture (Figure 6). PST can therefore be applied through dressings and in the presence of metal implants.

To date there have been no adverse effects reported with the use of PST (Low and Reed, 1990). However, due to the unknown mode of action, it is considered prudent to avoid rapidly dividing tissues, such as the foetus or the uncontrolled growth of precancerous tissues or neoplasms. Similarly, the risk of reactivating encapsulated lesions suggests that tuberculosis should be avoided. Cardiac pacemakers, hearing aids and other electronic equipment can be affected and, although not dangerous to the patient, can be inconvenient.

3.1.5 Technique of application

Pulsed shortwave therapy can be applied to the body tissues using the capacitor field method with rigid and malleable electrodes, and these are positioned by means of supporting arms. Several methods may be used, however the contraplanar method whereby electrodes are placed on each side of the area to be treated is most applicable in the treatment of hands. Both electrodes should be of the same size and the distribution of the electric field in the tissues will depend on the size and position of the electrodes used (Figure 7). The shape of the tissues will also have an effect on the distribution of the electric field. It has been suggested by Scott (1965) that maximum output is achieved when the electrodes are spaced at a maximum distance of 4 cm, and a minimum skin-electrode distance of 2 cm. It is important to position the electrodes parallel to the skin surface so that the skin-electrode distance is constant. The overall effect is to uniformly spread the electric field within the tissues.

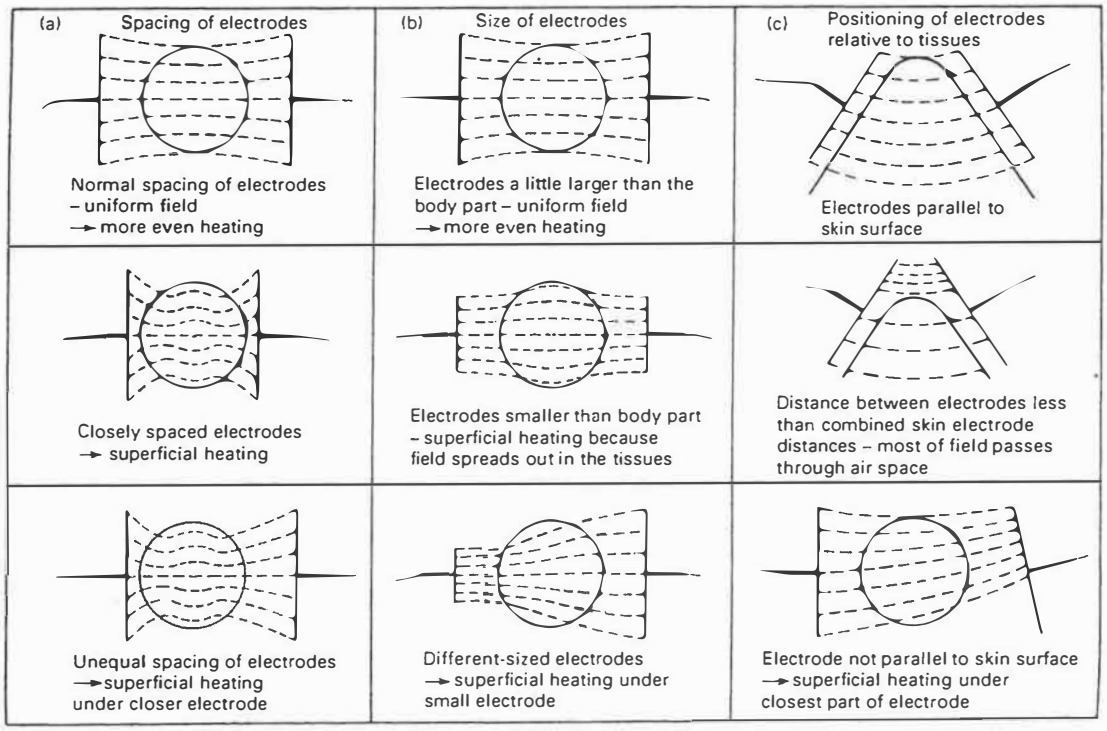


Figure 7 : The effect of positioning electrodes on the electric field through tissues using the contraplanar technique
(Adapted from Low and Reed, 1990)

3.1.6 Dosage

The dose is the total pulsed shortwave energy administered to a patient during a single treatment. The quantity of energy applied can be influenced with the pulse-repetition frequency (refer Table 1). Evidence does seem to suggest that longer treatment times with high pulse repetition rates and short pulses are most effective. This is illustrated by the three studies on sprained ankles (Wilson 1972; Pasila *et al.*,1978; Mcgill, 1989), with an average treatment time of 20 minutes.

Calculation of the dosage is illustrated in Figure 8.

A = 65 μs pulses of 27.12 Mhz oscillations repeated 100 times per second.

Duty cycle = 0.65%.

If peak power is 1000 W, mean power is 6.5 W.

B = 400 μs pulses of 27.12 Mhz oscillations repeated 200 times per second.

Duty cycle = 8%.

If peak power is 1000 W, mean power is 80 W.

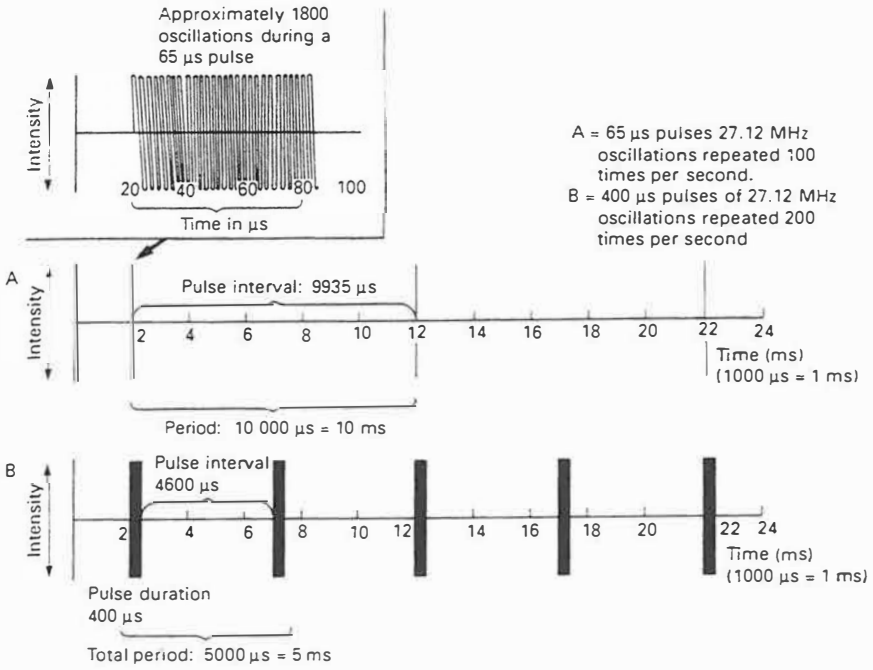


Figure 8 : Pulsed high-frequency oscillations
(Adapted from Oliver, 1984)

3.2 Laser

Laser is an acronym which standards Light Amplification for the Stimulated Emission of Radiation. Although the concept of laser light was first mooted by Einstein, it is only in recent years that its commercial, industrial and therapeutic applications have been explored. High power lasers are used for cutting, drilling and destructive purposes, while low power lasers are used for physiotherapeutic purposes.

Laser is light, or amplified luminous energy, endowed with specific physical characteristics, which is emitted when the atoms within tissues are charged by electromagnetic radiation of a given wavelength. Laser irradiation affects collagen metabolism thus facilitating wound healing (Low and Reed, 1994).

3.2.1 Production of laser

The laser beam is produced when atoms of certain elements are excited by electromagnetic radiation and consequently produce electromagnetic radiation of a particular wavelength. This phenomenon takes place in the so-called laser generator. This energy source is located in the probe of the laser unit (Figure 9).

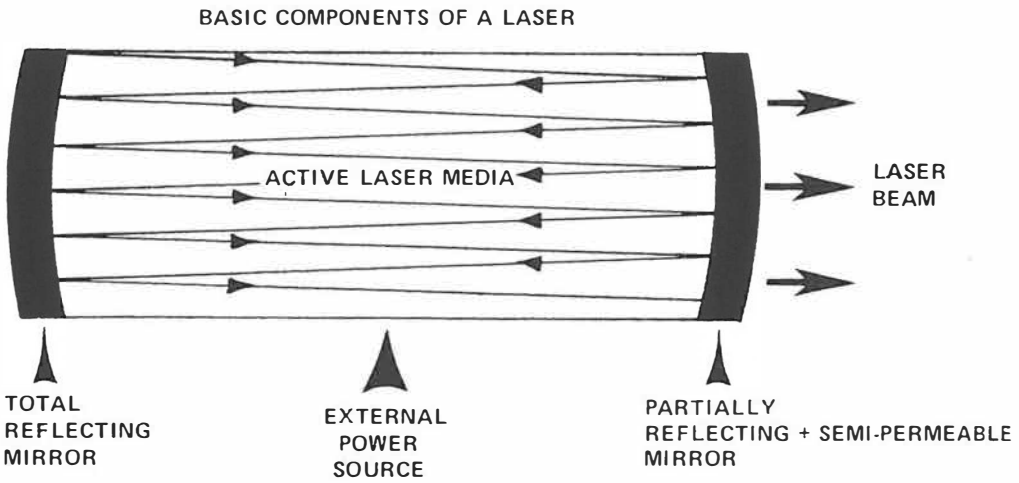


Figure 9 : The generation of laser
(Adapted from Kloth *et al*, 1990)

Lasers are of three types:

- The power laser is used for destructive or surgical purposes;
- Soft lasers have a very superficial effect and are used principally for treatment of the skin;
- Mid-lasers are the type used by physiotherapists as the depth of penetration of this type of laser is sufficient to produce a biological effect on deeper tissues without damaging them. The depth of penetration is up to 1 cm. Lasers are of a specific wavelength and hence of a defined frequency. In the case of visible lasers, a single pure colour is produced (Figure 10).

This supports the unique physical properties of laser energy which are:

1) Monochronocity - energy of a single wavelength which is amplified so that the beam shows as a very narrow line on a spectroscope (Figure 9). This physical property is mainly responsible for the biological response of the body. Visible laser beams give a pure colour which is not normally found in other light sources. As the GaAlAs laser unit falls in the red section of the visible spectrum, its colour is brilliant red.
2) High intensity (compared to ordinary light)
3) Coherence - the rays are in phase so that they oscillate uniformly.
4) Brilliance - this is a measure of light and consists of the power of emission and the directionality of the source of radiation. Laser beams are to a large extent parallel and thus have minimal divergence.

Table 2 : Examples of lasers

Laser type		*Wavelength (nm)*	*Radiation*
Ruby		694.3	Red light
Helium–neon		632.8	Red light
Gallium aluminium arsenide Diodes	Continuous wave	650	Red light
		750	Red light
		780	Infrared
		810	Infrared
		820	Infrared
		850	Infrared
		1300	Infrared
	Pulsed injection	860	Infrared
		904	Infrared
Carbon dioxide		10 000	Infrared

(Adapted from Low and Reed, 1990)

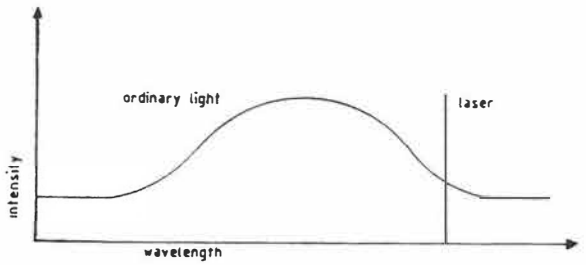


Figure 10 : Production of a single beam of pure laser
(Adapted from Par and Moolenaar, 1994)

Laser sources may be solid, liquid or gas and by varying the type of medium, laser beams of almost every wavelength between ultraviolet and infrared can be produced (Fig. 11). The laser unit used in this study is a type of semiconductor diode laser involving gallium aluminium arsenide (GaAlAs). In this type, electrons flow more readily in one direction than in the other. The electrons are excited by the application of a suitable electrical potential and this leads to the emission of a photon which may then stimulate identical photons. The photons are reflected to and fro and emitted as a laser beam from one partially transparent end (Figure 12). Gallium-aluminium-arsenide is built to give a

specific wavelength. Semiconductor laser diodes can give either a continuous or a pulsed output.

The amount of absorption in the different layers is dependent on the thickness of the skin, blood flow, water content, amino acid content and the presence of chemophores. Chemophores are light absorbing pigments such as melanin, bilirubin, β carotene and haemoglobin. Light energy is converted to other types of energy during absorption (Baxter, 1994). Scattering refers to a directional change of light propagation due to the complex geometry of the tissue involved (Figure 11). Most scattering occurs in the dermis, with minimal scattering in the epidermis (Parrish, 1892). Scattering and absorption determine the depth of penetration of the beam.

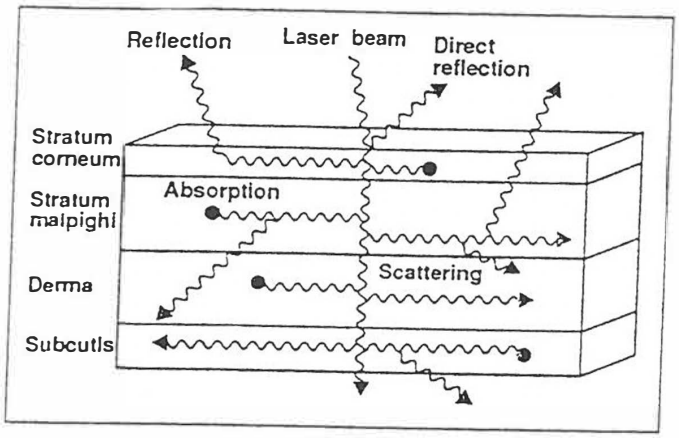


Figure 11 : Illustration of a laser beam passing through tissue
(Adapted from Kloth *et al*, 1990)

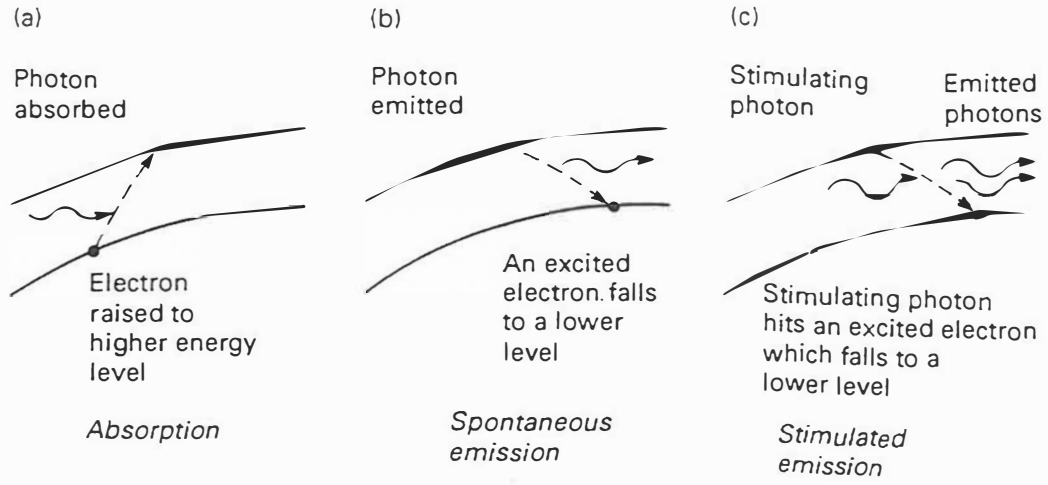


Figure 12 : Diagrammatic representation of the stimulated emission of photons
(Adapted from Low and Reed, 1990)

3.2.2 Physiological effects

The explanations of the physiological effects of laser at a cellular level are still largely theoretical. The following three theories are the most commonly described :

1. Cellular Oscillation Theory :

   This theory suggests that there is a resonance between cells and the laser beam. The laser beam has a definite frequency and when the energy reaches cellular level, this frequency will excite cells of similar frequency. It is considered that this process of resonance increases the biological processes within the cell and ultimately regulates or optimises its function.

2. Biological Field Theory

   It has been postulated that a "biological field" exists around every cell, tissue and organ. Tissue damage results in alteration of the biological and energy state of cells, and it is thought that the effects of the resonance produced by low power laser can restore normal cellular activity.

3. Bioluminescence Theory :

Deoxyribo-nucleic acid (DNA) replication in cells results in emission of "light" at 630 nm. This wavelength is very close to physiotherapeutic laser or "soft" laser. Therefore it has been suggested that such stimulation can accelerate DNA replication.

3.2.3 Therapeutic effects

There are two major therapeutic uses for which laser therapy is used, namely, tissue healing and pain control (Young *et al.*,1989). Other therapeutic effects include acceleration of fracture consolidation (Trelles and Mayo, 1981) and prevention of post-traumatic nerve degeneration (Nissen *et al.*,1986; Schwartz *et al.*,1984).

Consequently, laser irradiation could have a valuable role to play in collagen metabolism, facilitation of wound healing, and anti-inflammatory and analgesic effects in the treatment of a wide range of conditions, including acute soft tissue injuries, chronic musculo-skeletal conditions, benign pain, respiratory conditions, skin conditions, circulatory disorders, stimulation of nerve function and reduction in the formation of fibrous tissue.

3.2.4 Dangers and contraindications

Research has shown that the saturation effect of laser occurs at about 4 J/cm^2 (Thiel, 1987). Increase in cellular activity as a result of irradiation with laser depends on whether the absorbing cells are in a pathogenic or normal state. One study showed that when human skin fibroblast cells with low procollagen levels were irradiated with laser, their synthesis increased 36 times, whereas cells with normal procollagen levels increased their synthesis 4 times. This indicates that with normal use of low power lasers there is no danger of overdose and there is no hazard to the skin.. However due to saturation there is no further benefit to be obtained by exposing tissues to laser energy above 4 J/cm^2. There is the possibility of overstimulation effect if high energy densities are applied (above 8 J/cm^2).

The main danger is the risk of eye damage if the laser beam is applied directly to the eye. Treatment of neoplastic tissue should be avoided as cell stimulation may occur, leading to an increased rate of growth or metastases. Certain patients are not treated with laser, for example, epileptics. Cardiac patients and patients with pacemakers are not treated in the chest region. The treatment head must be sterilised with a suitable solution after being placed on the infected skin, when treating skin infections in contact in order to eliminate the chances of the spread of infection.

3.2.5 Technique of application

There are five techniques of application of laser beams in physiotherapy. In using the grid technique, the surface area of the tissue to be treated is determined. This surface is then divided into square centimetres (Figure 13). The laser probe is then applied at right angles to the tissues for the required time. This technique provides effective coverage of the affected tissue surface). The tip of the laser probe must be in contact with the surface of the skin in order to obtain maximum clinical effect.. However, for the treatment of open wounds, the probe is held at right angles to the wound and as close to the wound as possible (Figure 14). Other techniques of application are the scanning, stroking, open-joint technique, and point application.

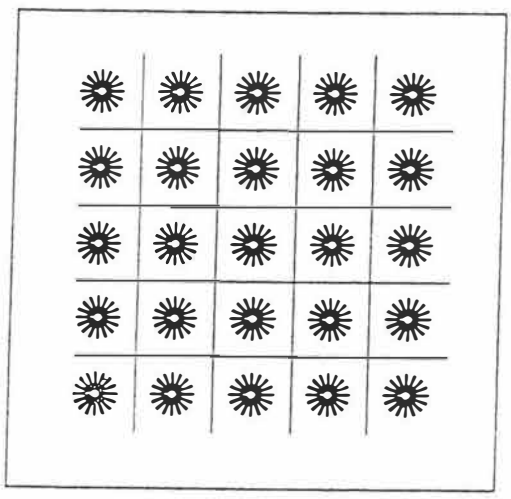


Figure 13 : Laser irradiation of a large wound (The distance between the points of irradiation is 1 cm)
(Adapted from Par and Moolenaar, 1994)

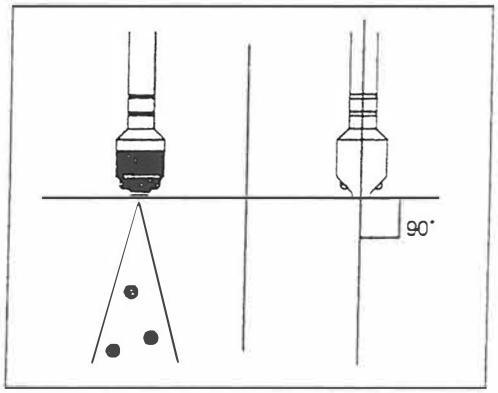


Figure 14 : The incident angle of the laser beam on the skin surface must be perpendicular to the surface for optimum effect
(Adapted from Kloth *et al,* 1990)

Laser application in the management of pain is directly over the site of pain, injury, tender trigger points, reflex points, local and distal acupuncture points, and along the peripheral nerves. Continuous and pulsed are two modes of application of laser. The continuous mode has been recommended for use in acutely painful conditions, superficial conditions and acute wounds. The pulsed mode has been found to be more effective with chronic conditions and deep-lying tissues (Low and Reed, 1990).

3.2.6 Dosage

Laser therapy used in physiotherapy is at a level so low that the localised temperature of the treated tissue does not exceed 36.5°C. The clinical effect of laser therapy depends on the amount of energy put into the tissues at each individual treatment point. Light absorption at different wavelengths in water, haemoglobin and melanin (Figure 15) show that the greatest degree of tissue penetration is obtained with wavelengths between 600 nm and 900 nm (as for the GaAlAs laser). Melanin plays the most important role in the absorption of this type of laser. The longer wavelengths penetrate the dermis better than shorter wavelengths.

The power output may be monitored by the unit itself once energy density or time is chosen, as is the case with the Unilaser 201. The dosage/energy density of laser is expressed in joules per cm2 (J/cm^2). The following equation is used to calculate the dosage :

$$\text{Energy density } (J/cm^2) = \frac{\text{emitted mean power of the laser (W) x duration (secs)}}{\text{area of the laser beam } (cm^2)}$$

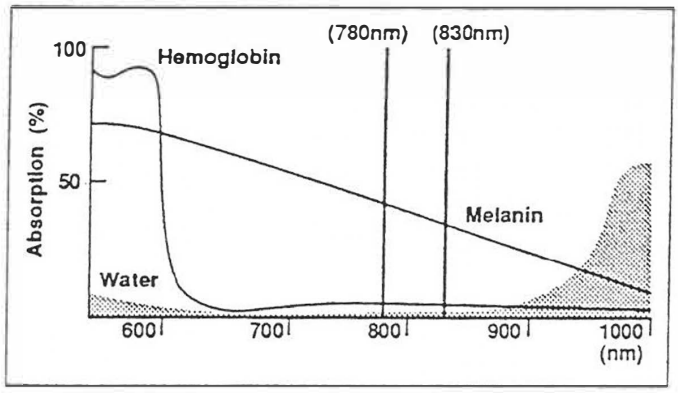


Figure 15 : The light absorption spectrum at different wavelengths
(Adapted from The Scientific Committee of Tokyo Judo Bonesetter Committee, 1984)

3.3 Ultraviolet radiation (UVR)

The sun is the most obvious source of UVR. Ultraviolet radiations are well recognised as the wavelengths that cause sunburn and tanning on exposure to the sun. Ultraviolet is so termed because they are invisible radiations beyond the violet end of the spectrum. The laws of reflection are employed in the design of reflectors used for the redirection of rays towards an appropriate target. In infrared and ultraviolet lamps, a parabolic reflector is normally used as this avoids the danger of the concentration of rays which occurs with some shapes of reflector (Figure 16). A parabolic reflector collects all the rays travelling in an appropriate direction and reflects them from its surface so that they eventually all emerge parallel.

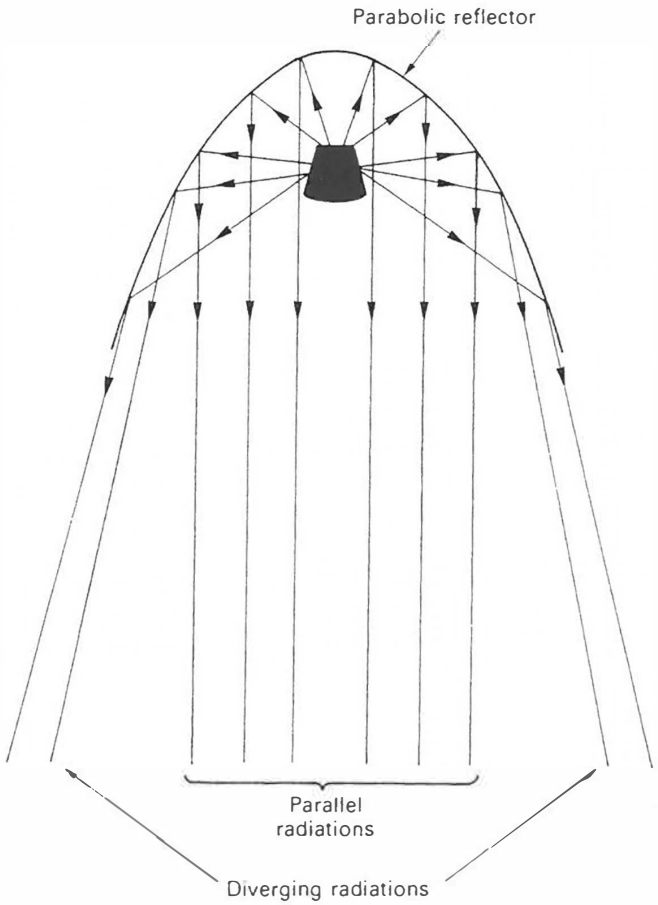


Figure 16 : A parabolic reflector
(Adapted from Scott, 1975)

Ultraviolet radiation is electromagnetic energy which is invisible to the human eye, with wavelengths between 10 - 400 nm. Ultraviolet lies between the visible light and X-rays in the electromagnetic spectrum (Figure 17).

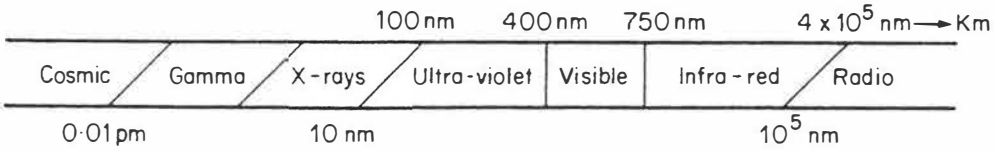


Figure 17 : The electromagnetic spectrum
(Adapted from Klaber, 1980)

3.3.1 Production of UVR

Ultraviolet radiation is produced partly as energy released by the recombination of electrons and positive mercury ions, and partly by photons released when excited electrons return from a higher-energy quantum shell to their normal shell within the mercury atoms. At the same time however, visible and infrared electromagnetic waves are produced, and ultraviolet forms only a part of the total spectrum (Figure 17). UVR is produced by the passage of a current through an ionised vapour, usually mercury vapour, for therapeutic purposes. The tube or envelope must be made of quartz to allow UVR to pass through it (Figure 18).

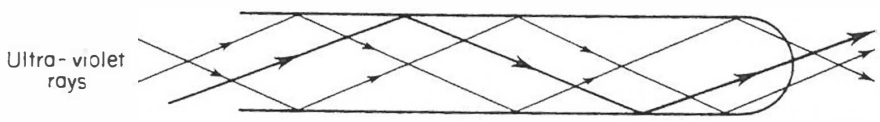


Figure 18 : Mercury vapour gas discharge tube
(Adapted from Scott, 1975)

The Kromayer lamp (Figure 19), used in the study, consists of a high-pressure mercury vapour burner, designed to be used in contact with tissues, both on the skin surface and in body cavities. It is completely enclosed in a jacket of circulating distilled water, the purpose of which is to absorb the infrared rays. A pump and cooling fan are incorporated into the body of the Kromayer lamp in order to cool the water. The water circulation should be continued for 5 minutes after use, to cool the lamp after the burner is switched off. The water circulates between two quartz windows at the front of the Kromayer head, which allow the ultraviolet to emerge. The whole process of argon ionisation, mercury vaporisation and ionisation takes some time, and a period of 5 minutes elapses between starting the burner and ultraviolet emission reaching its peak.

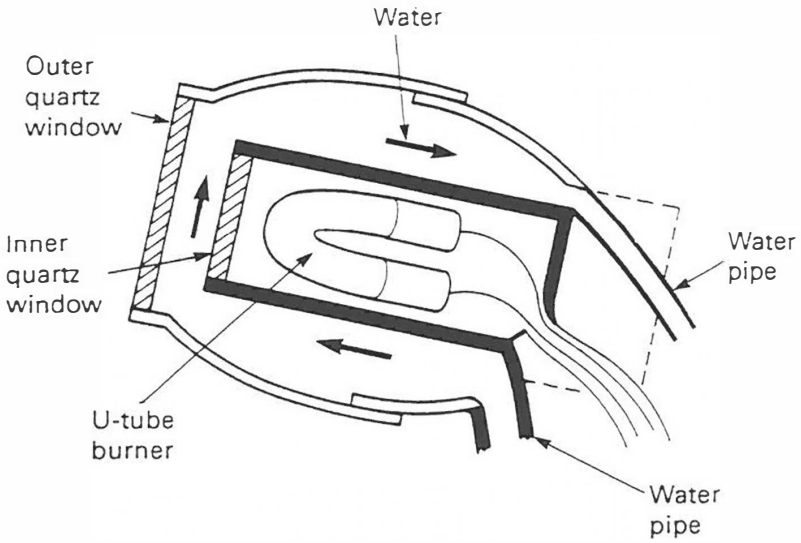


Figure 19 : Kromayer water-cooled lamp
(Adapted from Meijer *et al,* 1991)

The quartz (or special glass) allows UVR to pass through it. Metal electrodes are sealed into the ends (Figure 20). As the pressure in the tube is very low (approximately 400 N/m^2), the mercury will be in vapour form. Some electrodes will be detached from their parent atoms by natural radiation, thus leaving some mercury ions, and will then recombine. If a strong voltage is applied across the electrodes, electrons can be accelerated

and collide with other atoms, splitting off further electrons and leaving many free ions. Recombination of electrons and ions occurs and a steady current flows with electrons being added at one electrode and removed at the other end. This process needs a high voltage to start it, but will continue with a lower voltage and is regulated by limiting the current that is allowed to pass through the tube.

As mains alternating voltage is applied, the process reverses 100 times every second. When many free electrons are being accelerated in the tube, many collisions with neutral mercury vapour atoms will occur; some will be elastic collisions and will not affect the atom. Some will cause ionisation and some will cause excitation. When these excited electrons return to their normal energy level, the energy they lose is emitted as a photon of a characteristic wavelength for that particular transition. Similarly, electrons recombining with ions will give the same effect. The characteristic photon wavelengths given off by mercury atoms are in the green-blue-violet end of the visible spectrum and in the ultraviolet. A line spectrum is thus produced. The wavelengths and intensities emitted are modified at different lamp pressures and filtered by the quartz envelope.

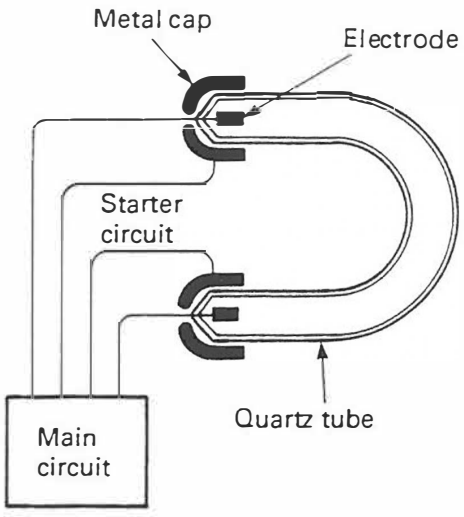


Figure 20 : Mercury vapour gas discharge tube
(Adapted from Low and Reed, 1990)

3.3.2 Physiological effects

The skin acts as a protective layer, in that it absorbs most ultraviolet and prevents its penetration down to vulnerable cells. If ultraviolet waves are absorbed by the skin, the energy they release is sufficient to cause damage, and the consequent reaction depends on the wavelength of ultraviolet and the amount of ultraviolet absorbed (Figure 21).

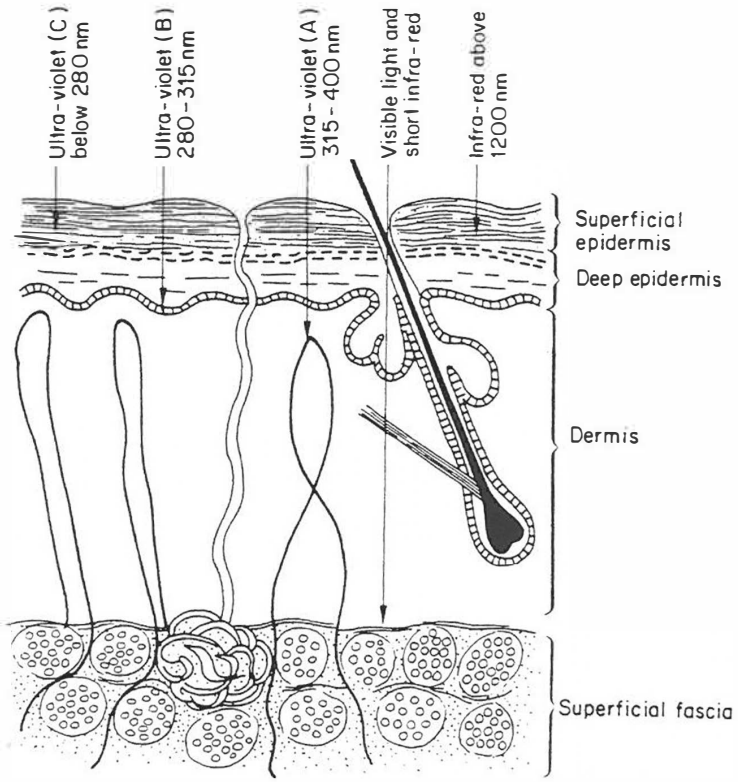


Figure 21 : Cross section of the skin showing the extent of penetration of radiation of different frequencies
(Adapted from Scott, 1975)

Further physiological effects include the following :-

1) Where the skin is intact, the effect of UVR is the same as the acute effects of sunburn.

2) Strong doses of UVR to the eyes can lead to conjunctivitis (inflammation of the tissue over the cornea and lining of the eyelids) and to photokeratitis (inflammation of the cornea).

3) Pigmentation of the skin : Ultraviolet stimulates melanocytes in the skin to produce melanin, which is then passed to numerous adjacent cells. Pigmentation reduces the penetration of ultraviolet rays.

4) Thickening of the epidermis : Stimulation by UVR provokes increased keratinocyte cell turnover. The skin grows more rapidly for a time, leading to shedding of the most superficial cells at an earlier stage in their development than usual so that they can be peeled off. Sudden overactivity of the basal layer of the epidermis causes a marked thickening, particularly of the stratum corneum (the outermost layer). This reduces the ultraviolet penetration. In order for subsequent treatments to have the same effect the dose must be increased (provided that peeling has not occurred). When treating open wounds, that is areas devoid of skin, it is unnecessary to progressively increase the dose as there is no epidermis and therefore no thickening, and hence no resistance to the penetration of ultraviolet radiation.

5) Production of vitamin D : In the presence of ultraviolet, 7-dehydrocholesterol in the sebum is converted to vitamin D in the skin. Vitamin D is necessary for the absorption of calcium which plays a role in the normal formation of bones and teeth.

6) Bactericidal effect : Short ultraviolet rays can destroy bacteria and other small organisms such as fungi commonly found in wounds. Experimental evidence has shown that an E4 dose (refer 3.4.6) effectively destroys all such organisms (High, 1983).

7) Erythema : Damage to cells causes the release of histamine-like substances from the epidermis and the superficial dermis. A gradual diffusion of this chemical takes place until sufficient has accumulated around the blood vessels in the skin to make them dilate. This accounts for the latency in the erythema.

8) Solar elastosis and ageing : The normal ageing process of the skin is accelerated if there is continued exposure to ultraviolet. There is thinning of the epidermis, loss of epidermal ridges, loss of melanocytes, dryness as a result of poor function of sebaceous and sweat glands, and wrinkling from lack of dermal connective tissue.

9) Peeling : The increased thickness of the epidermis is eventually lost as desquamation (peeling). The resistance of the skin to UVR is eventually lowered when this happens.

3.3.3 Therapeutic effects

The following therapeutic effects are derived when using UVR :

1) Bactericidal effect - UVR may be used for the treatment of infected skin wounds. The aim of the ultraviolet is to destroy bacteria, remove the slough and promote repair.
2) Non-infected wounds - the aim of UVR is to stimulate the growth of granulation tissue.
3) Peeling effect - for the treatment of acne and psoriasis.
4) Intact skin may be treated with UVR if it is in a pressure area that is likely to break down.

3.3.4 Dangers and contraindications

Conjunctivitis may occur if ultraviolet rays are allowed to fall on the eye. To prevent this, the physiotherapist always wears protective goggles when the lamp is on. The patient is also provided with goggles or the eyes are screened using cotton wool. UVB and UVC are absorbed by the cornea, but UVA is absorbed by the lens and is implicated in the formation of cataracts.

Further dangers and contraindications are :-

1) Carcinogenesis is a danger if long exposure to ultraviolet rays occurs, as these rays may affect the DNA and thus cell replication.
2) Skin conditions : Certain skin conditions such as eczema, dermatitis, herpes simplex and systemic lupus erythematosus may be exacerbated by UVR.
3) Recent skin grafts.

4) Hypersensitivity to sunlight : Some patients react adversely to sunlight and are not treated with UVR.

5) Deep X-ray therapy : Produces local hypersensitivity to UVR and patients are not treated for three months with UVR following deep X-ray therapy.

6) Erythema : If the patient's skin still presents an erythema from either UVR or IRR, the reaction to UVR is increased. Consequently, UVR is contraindicated until the erythema has subsided.

3.3.5 Technique of application

The lamp requires about 5 minutes to reach operating temperature and should be switched on at least 5 minutes before treatment is commenced. An E_4 dose is given. There is no need for further sterilisation as the UVR emitted will kill off bacteria on the surface. Following treatment, the lamp is cleaned again and the wound is redressed.

3.3.6 Dosage

An E_4 dose is not usually given on skin; it is applied to areas devoid of skin. The aim of the ultraviolet is to destroy bacteria, remove the slough and promote repair in infected wounds. The aim of UVR in non-infected wounds is to stimulate the growth of granulation tissue and thus speed up repair. UVR is applied locally to the lesion using the Kromayer lamp. Every lamp has its average E_1 time and distance as a result of averaged reaction tests on a number of people.

Given the average E_1 of the lamp, the duration of E_2, E_3 and E_4 doses can be calculated as follows :

E_2 time = E_1 time x 2.5

E_3 time = E_1 time x 5

E_4 time = E_1 time x 10

Progression of the E_4 dosage is unnecessary as there is no skin present (Klaber, 1980).

3.4 Infrared radiation (IRR)

Infrared radiation is a form of radiant energy that provides superficial dry heat. The energy for clinical use falls within both near-infrared and far-infrared regions of the electromagnetic spectrum (Figure 17).

Infrared radiation is produced in all matter by various kinds of molecular vibration. The various states of vibration and rotation that a given molecule may have can be altered by absorbed heat leading to the emission of infrared radiation. Heat may be transmitted by infrared electromagnetic radiation.

3.4.1 Production of IRR

Infrared rays are electromagnetic waves with wavelengths of 750 nm - 400 000 nm. Any hot body emits infrared rays; the sun, gas fires, coal fires, etc. Infrared generators may be luminous or non-luminous. The rays emitted from the luminous generators are produced by one or more incandescent lamps. An incandescent lamp consists of a wire filament enclosed in a glass bulb, which may be evacuated or may contain an inert gas at a low pressure. The filament is a coil of fine wire and is usually made of tungsten, as this material withstands repeated heating and cooling.

The loops of wire are supported on hooks, with small springs which take up the slack when the wire expands. The indirectly heated filament also consists of fine loops of wire through which the current is passed, but these are embedded in insulating material and the whole surrounded by a metal cylinder from which the thermionic emission takes place and which acts as the cathode of the valve. The valve may be evacuated or contain an inert gas at a low pressure. The pressure inside the glass bulb must be low so that there is minimal impedance to the electron movement. The passage of an electric current through the filament produces visible infrared rays (Figure 22).

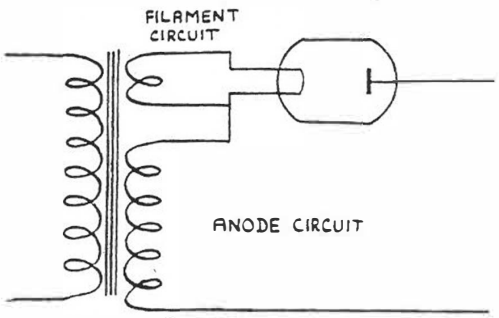


Figure 22 : Production of IRR

The spectrum is from 350 - 4000 nm, the greatest proportion of rays having wavelengths in the region of 1000 nm. The depth of penetration of electromagnetic radiation depends on its wavelength and the nature of the material. The human skin will allow the passage of infrared and ultraviolet rays and their approximate depth of penetration into the skin is up to the superficial fascia (Figure 21).

When an object is heated, the increased vibrations of the molecules causes displacement of the electrons from one orbit to another. As the electrons return to their original orbits, energy is released and electromagnetic waves are set up. All electromagnetic waves are produced by movement of electrons, but different electron movements produce rays of

different wavelengths. The rays travel until they encounter a medium which absorbs them. The effects are produced at the point at which they are absorbed.

Infrared and ultraviolet rays obey the laws of refraction, reflection, absorption and inverse squares. These laws are stated as follows :

The Law of Grottus states that rays must be absorbed to produce their effects. The law of inverse squares states that the intensity of rays from a point source varies inversely with the square of the distance from the source. The angle of the incident ray to the normal is equal to the angle of the reflected ray to normal. This is represented in Figure 23. The penetration of radiation is proportional to the cosine of the angle of incidence of the radiation. This is illustrated in Figure 24. If the radiation is angled to the surface, then a larger area is covered and hence intensity per unit area is reduced (Low and Reed, 1994).

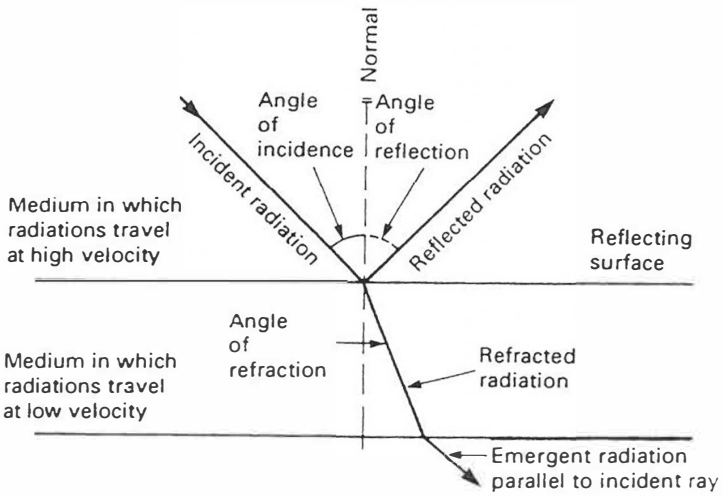


Figure 23 : Reflection and refraction
(Adapted from Low and Reed, 1990)

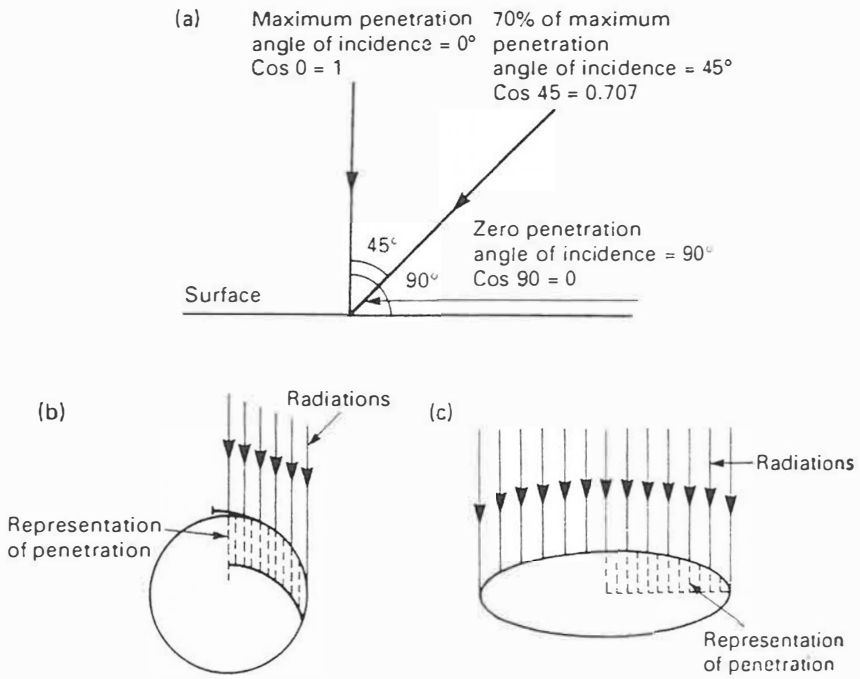


Figure 24 : Ratio of reflected and penetrating radiations

(a) cosine law (b) parallel radiations applied to a curved surface

(c) parallel radiations applied to an ellipsoid surface

(Adapted from Scott, 1975)

Infrared, visible and ultraviolet waves travel in straight lines until they encounter a different medium, when they may be transmitted, reflected or absorbed. As ultraviolet lamps act as point sources, the rays they generate observe the law of inverse squares. In practical terms, this means that the closer a patient is to the source, the greater is the intensity of the radiation being received.

3.4.2 Physiological effects

As a consequence of the heating with infrared radiation, local cutaneous vasodilatation occurs. This is due to the liberation of chemical vasodilators, histamine and similar substances, as well as a possible direct effect on the blood vessels.

Infrared is used therapeutically to relieve pain. The mechanisms by which this occurs could be the stimulation of sensory nerves leading to pain suppression. Muscle spasm of the superficial muscles is reduced as a consequence of the decreased pain. Application of infrared radiation to the surface of open wounds such as pressure sores or ulcers will dry the surface thus diminishing bacterial activity. Vessel dilation induced by heating will allow increased rates of fluid exchange and thus may help to increase the reabsorption of exudate (Wadsworth and Chamnugan, 1980). This effect is limited as infrared heats the superficial tissues. Such heating arrangements coupled with active exercise are valuable in the treatment of hand injuries in general (Low and Reed, 1990).

3.4.3 Therapeutic effects

The therapeutic uses of infrared are :

1) Relief of pain **:** Infrared radiation is frequently an effective means of relieving pain. It has been suggested that pain may be due to the accumulation in the tissues of waste products of metabolism, and an increased flow of blood through the part removes these substances and so relieves pain. In some cases the relief of pain is probably associated with muscle relaxation (Forster and Palastangar, 1990).
2) Muscle relaxation : Muscles relax most readily when tissues are warm, and the relief of pain also facilitates relaxation. Infrared radiation is therefore of value in helping to achieve muscular relaxation and for the relief of muscle spasm associated with injury or inflammation.
3) Increased blood supply : The effect is most marked in the superficial tissues, and may be used in the treatment of superficial wounds and infections. A good blood supply is essential for healing to take place, and if there is infection the increased number of white blood cells and the increased exudation of fluid are of assistance in destroying bacteria.

3.4.4 Dangers and contraindications

1) Burns : There is a danger of heat burn which occurs if the patient is unaware of the heat especially if there is defective or diminished sensation. This is avoided by careful application, adequate warnings to the patient and checking the effects on the skin (which is easily visible with this treatment) several times during the application.
2) Areas in which the arteries and arterioles cannot respond by adequate vasodilatation to the demands of additional heating should not be treated. It is also unwise to apply the treatment to areas where the skin sensation is defective, or on which liniment has recently been applied (Lehman, 1992; Haynes, 1992).
3) Acute skin disease : Dermatitis or eczema.
4) Skin damage due to deep X-ray therapy or other ionising radiation.
5) Patients whose blood pressure regulation is defective : Extensive irradiation is accompanied by a fall in blood pressure which may result in faintness due to hypoxia of the brain.
6) Tumours of the skin may be stimulated to increase growth.
7) Injury to the eyes : It has been suggested that exposure to infrared rays may predispose to cataracts, therefore the eyes should be protected from irradiation.

3.4.5 Technique of application

The luminous lamp is switched on to stabilise, a few minutes before application of the treatment. The lamp is positioned so that it is opposite to the centre of the area to be treated and the rays strike the skin at right angles, thus ensuring maximum absorption. The distance of the lamp from the patient should be measured to 45-60 cm from the area to be treated, to achieve maximum penetration. Care must be taken that the patient's face is not exposed to infrared rays. The skin should feel mildly warm at the end of the treatment.

3.4.6 Dosage

The depth of skin penetration by light from non-luminous lamps is approximately 2 mm compared with 5-10 mm for light emitted from luminous infrared lamps. The output of infrared lamps is determined by the wattage. The intensity of the irradiation is usually controlled by varying the distance of the lamp from the skin. The distance is usually between 45-60 cm. The duration of each treatment is typically 10-15 minutes. Longer exposures may be used for chronic conditions.

Usually electrotherapy is part of an overall treatment plan which is selected and modified on the basis of repeated examination and assessment.

CHAPTER 4

4. PATIENTS AND METHODS

The study was conducted at the Workmen's Accident and Rehabilitation Centre, Durban where patients were referred following crush injuries to the hand sustained at the workplace. The referral source was from orthopaedic and plastic surgeons in private practice (St. Augustine's, Entabeni, Parklands, Umhlanga, Kingsway and Victoria hospitals), via the state hospitals (King Edward VIII, Clairwood, Addington, Prince Mshyeni and other outlying hospitals), industrial medical doctors and nurses, and other therapists in practice.

4.1 Board approval and patient consent

Institutional Board approval was obtained on submission of the protocol for the study. The research was conducted by one researcher with a second obtaining data only on the patient's initial visit when the researcher was not available. Random selection of patients with full thickness open wounds on the volar aspect of the hand was done as follows:

A roster was drawn up for the different treatment groups and zones of injury, and patients were allocated to the treatment groups in the respective zones. For example: the first patient presenting with a zone I injury was allocated to Group A. Subsequent patients with zone I injuries were then allocated each to Group B, C, D and E respectively, until all groups had one zone I injured patient. The process was repeated in the same order for subsequent patients with zone I injury until five patients were accumulated in each group. This format was adopted for all the treatment groups for each zone of injury.

All wounds were sustained less than one month before admission to the study.

4.2 Sample selection

125 patients with open wounds (full thickness) to the volar aspect of the hand participated in the study. All wounds were sustained less than one month before admission to the study. The volar aspect of the hand is the commonest site of work-related hand injuries. All patients were assessed for zone of injury, and zones of flexor tendon injury (Figure 25) were used to match the wounds for zone of injury. The evaluation was done using the Assessment Chart (Appendix 1).

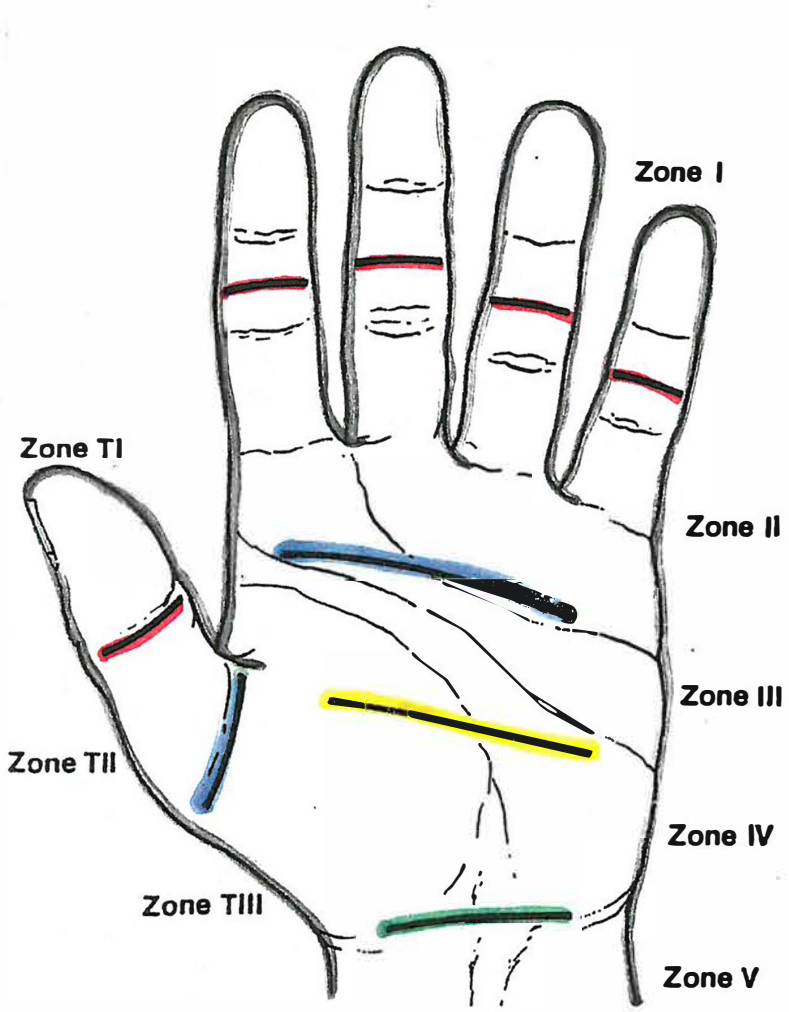


Figure 25 : Zones of flexor tendon injury used to classify wounds for zone of injury

4.2.1 Selection criteria

Patients who had sustained a crush injury with open wounds on the volar aspect of the hand were included in the study. The mode of injury was crush injury sustained at the workplace.

4.2.2 Exclusion criteria

The following patients were excluded from the study :

1. Diabetes
2. Clinical malnutrition
3. Systemic infection
4. Anaemia
5. Haemoglobin level less than 10 g%

4.2.2.1 Haemoglobin level test

Patients were referred to the pathology laboratory for haemoglobin (Hb) level tests. Patients with an Hb level less than 10 g% were excluded from the study and referred to the medical practitioner for possible further medical management. These patients were excluded from the study, as it was felt that the patient was already compromised as a result of the low Hb level and this would affect the rate of wound healing.

4.2.2.2 Wound swab

Wounds were exposed and a wound swab was taken using Sterilin sterile swabs (Smith & Nephew) at the initial assessment (Plate 1). The wound swabs were sent to the pathology laboratory for microbiological analysis for micro-organisms. The referring medical practitioner was informed of the results. Those patients on whom antibiotic therapy was administered were excluded from the study, as it was felt that antibiotics would influence the healing process, and thus the results obtained from the study.

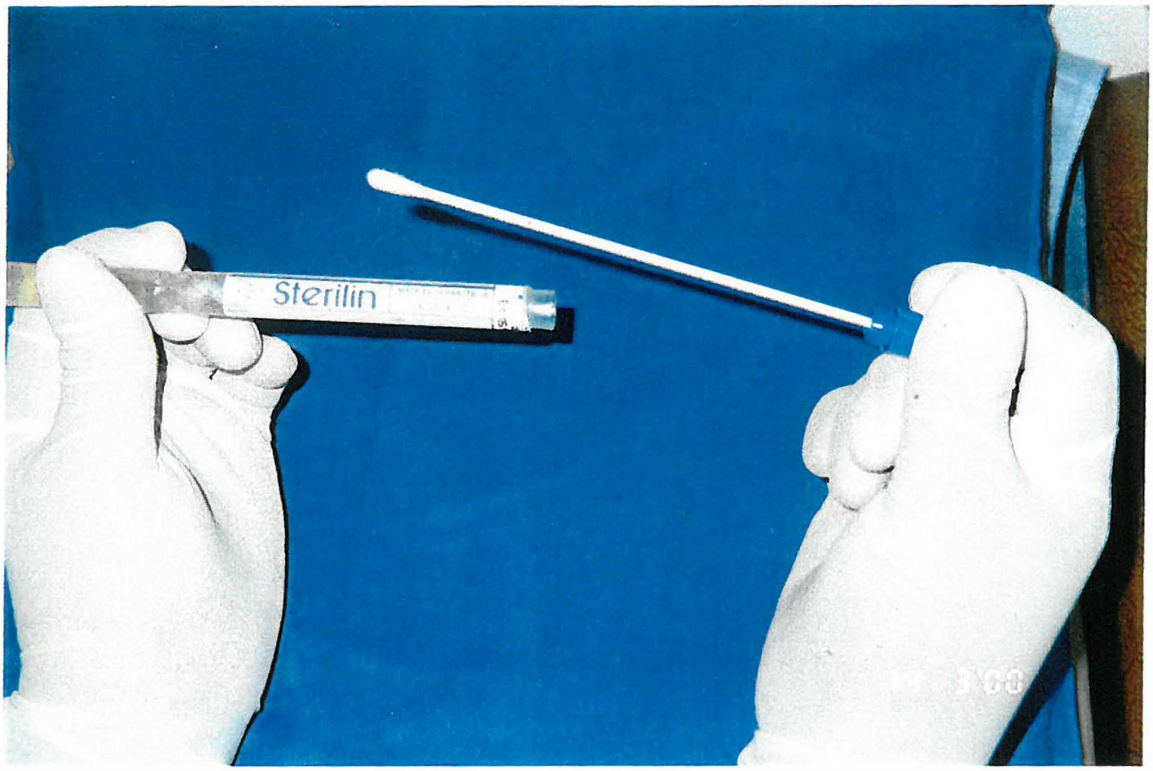


Plate 1 : Photograph of a wound swab

4.3 Randomisation

Following initial assessment and classification for zone of injury, patients were randomly allocated to one of 5 groups :-

Group A - pulsed shortwave therapy (PST)

Group B - light amplification for the simulated emission of radiation (LASER)

Group C - infrared radiation (IRR)

Group D - ultraviolet radiation (UVR)

Group E - control group (dressings only)

Patients were randomly assigned to one of five groups, with 25 open hand wounds in each group and 5 in each zone (Table 3).

Table 3 : Patient distribution

	NUMBER OF PATIENTS				
ZONE OF INJURY	*PST GROUP A*	*LASER GROUP B*	*UVR GROUP C*	*IRR GROUP D*	*CONTROL GROUP E*
I	5	5	5	5	5
II	5	5	5	5	5
III	5	5	5	5	5
IV	5	5	5	5	5
V	5	5	5	5	5
TOTAL NO. OF PATIENTS (N = 125)	25	25	25	25	25

4.4 Assessment of patients

All patients were evaluated using the Assessment Chart (Appendix 1). Re-evaluation of wounds was done once a week.

4.4.1 Wound dimensions

Wound size was assessed as follows :

4.4.1.1 Trace

A trace was made of each wound (Plate 2) using Tegaderm with the grid (3M Medical). A trace was done initially and then repeated once a week for all wounds.

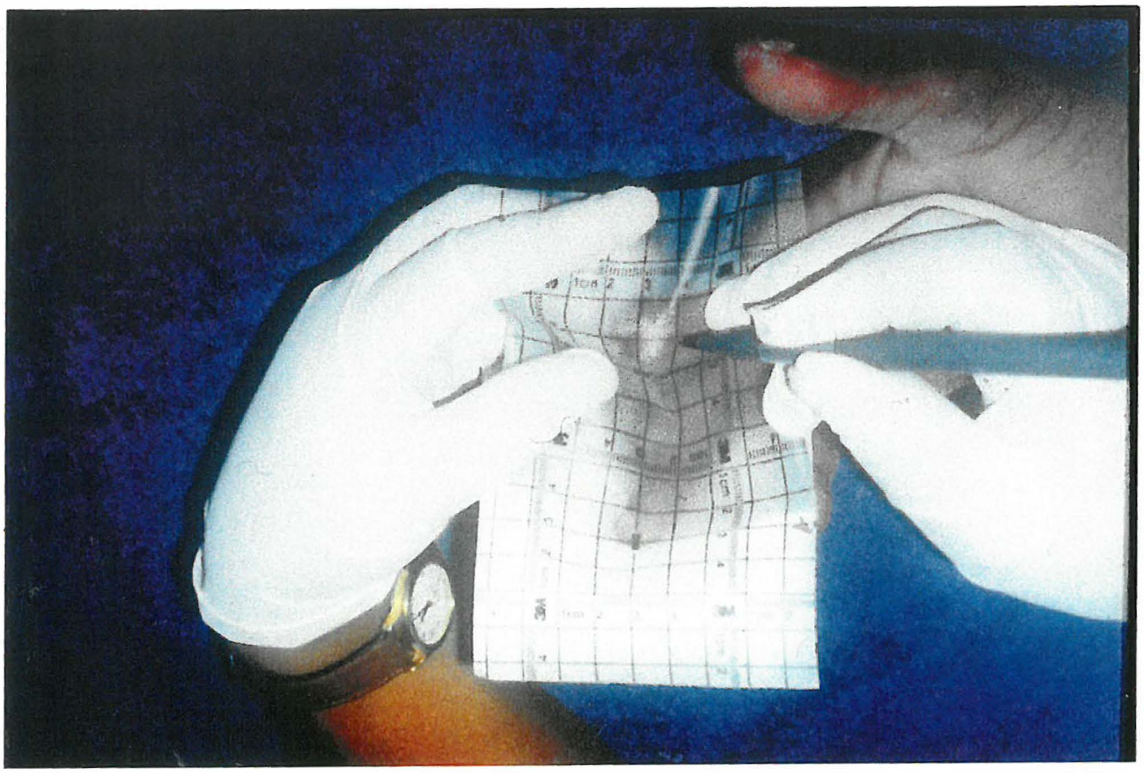


Plate 2 : Photograph showing Tegaderm trace

4.4.1.2 Area of wound

A trace was made of each wound using the sterile Tegaderm made by 3M (Plate 2). The Tegaderm was placed directly over the wound and the outline of the wound was traced onto the Tegaderm. The trace was then cut out and each weighed on a Mettler balance and the wound dimensions were extrapolated. The reading obtained was the weight of the trace of that particular wound, read in milligrams (mg). The area of each wound was calculated from this. The subsequent wound dimensions were calculated as a percentage measurement of the original wound size, and the original wound was taken to be 100%.

Calculation of area of wound : Example :

1 c^{m2} paper weighs 0.08 g on the Mettler balance

therefore cm^2 paper weighs e.g. 3.48g (weight on Mettler balance for wound trace)

therefore Area of wound (x cm^2) = $\frac{3.48g \times 1\ cm^2}{0.08g}$

= 43.5 cm^2

The original size of the wound at **week 0** = 33.79 mg (as measured on the Mettler balance)

The **wound size** was taken to be **100% at week 0**

The size of the wound at **week 1** = 25.68 mg (as measured on the Mettler).

An example of this calculation is as follows :

Area of the wound = $\frac{\textbf{wound measurement at week 1}}{\textbf{original wound measurement (week 0)}}$ **x 100%**

= $\frac{25.68}{33.79}$ x 100%

= **76 %**

Therefore 76% of the original wound remains.

4.4.2 Photography

Photographs were taken using a Minolta 40X camera at a standard distance of 100 cm. Kodak 100 ASA film was used. Photographs were taken once a week. Visual comparison was made of the wounds on consecutive weeks (Plates 9 and 10)

4.4.3 Wound granulation

The following rating was used when determining the status of wound healing on examination.

1 No granulation

2 Patchy granulation

3 Good granulation

4 Overgranulation

5 Epithelialisation

4.4.4 Pain measurement

Pain was measured using the Numerical Rating Scale (Figure 26). The numerical rating scale (NRS) has 11 points, with 0 as no pain and 10 is the worst pain experienced. When using the NRS, the provider simply asks the patients to rate the intensity of their pain if zero is no pain and 10 is the worst pain possible. Patients select a number reflecting their perception of the degree of pain intensity. Pain intensity was assessed once a week using the NRS, and this was done while the patient attended treatment.

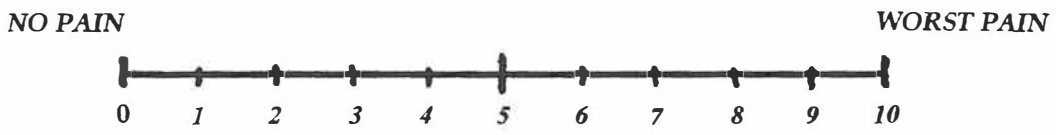


Figure 26 : Numerical Pain Rating Scale

4.5 Technique

The following technique was used for examination, evaluation and assessment of the wounds.

4.5.1 Sterile Procedure

Patients were treated under sterile conditions. Wounds were treated using latex gloves and sterile instruments. Biocide solution (Unilever laboratories) and autoclaving of instruments ensured sterility.

4.5.2 Saline soak

0.9% sodium chloride solution (normal saline) (Adcock Ingram - Critical Care) was used for wound lavage. The seal was broken but not removed at the time of use, to maintain sterility. The bottle of saline was placed in the microwave oven for 2 minutes to warm the solution for the comfort of the patient. Patients soaked the hand in the saline solution for 10-15 minutes (Plate 3). This was used for wound lavage and to encourage active range of motion exercises of the affected hand.

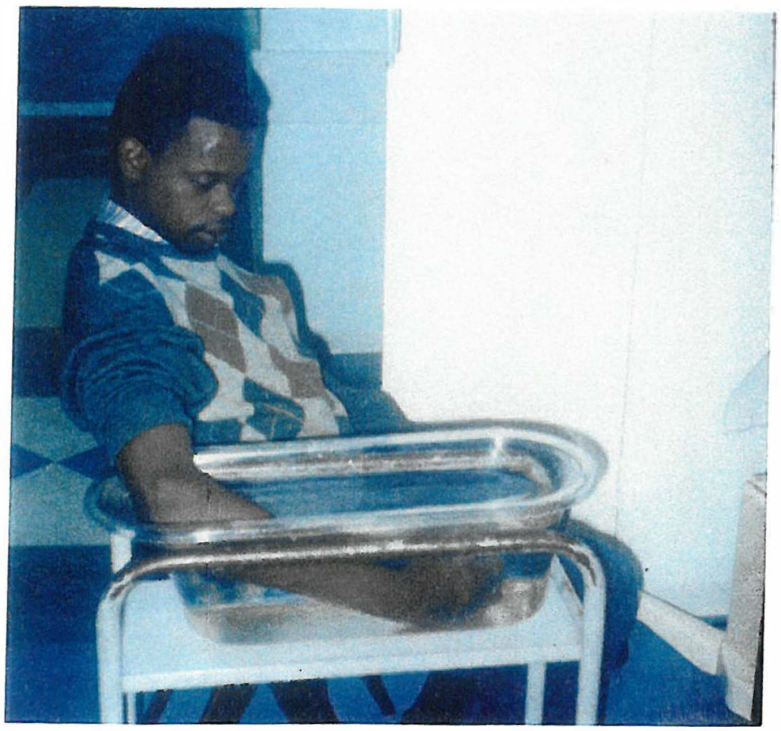


Plate 3 : Photograph showing normal saline soak of the hand

4.5.3 Wound dressings

A neutral dressing of Jelonet and dry gauze (Smith & Nephew) was used for all wounds in all groups (Plate 4). A minimal dressing was used in order to provide cover only for the area of the wound, and to encourage as much active range of motion as possible of the joints. Dressings were changed at each treatment session.

Jelonet consists of leno-weave fabric of cotton which has been impregnated with white soft parrafin. This dressing is used as a primary wound contact layer and the parrafin is present to reduce adherence of the product to the surface of a granulating wound.

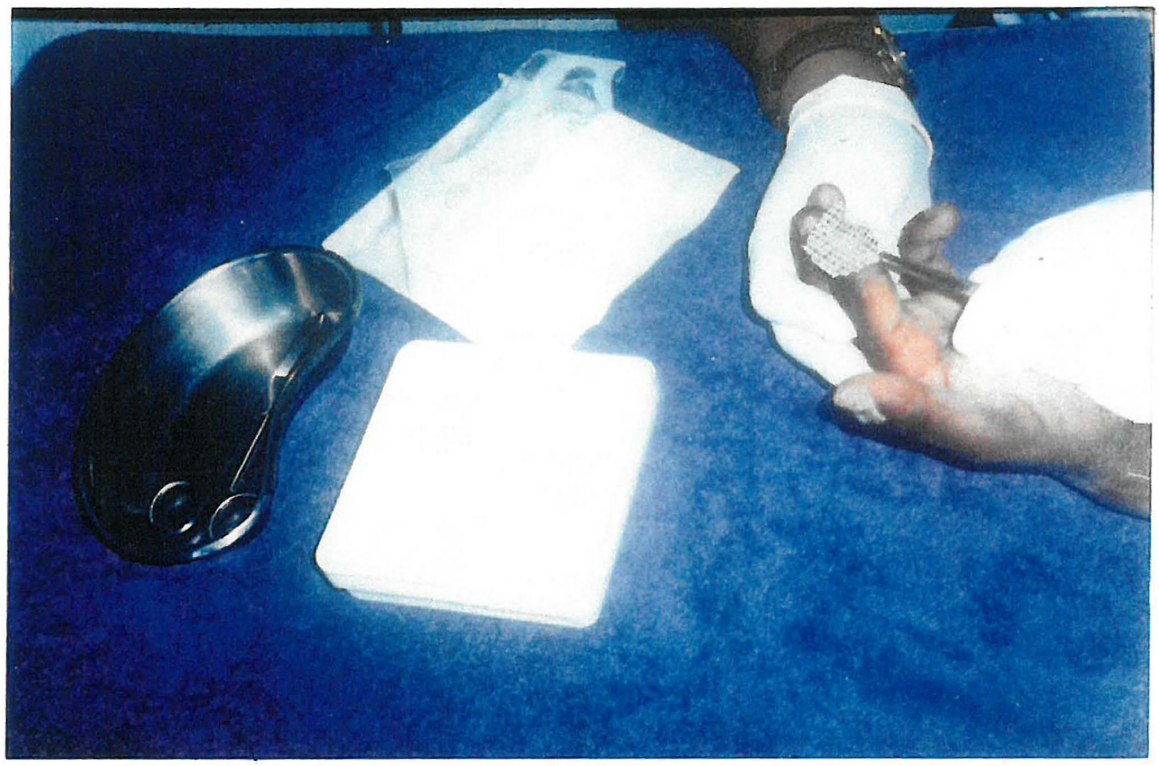


Plate 4 : Photograph showing Jelonet wound dressing

4.5.4 Frequency of treatment

All patients were treated three times weekly for 5-8 weeks, or until the wounds were healed. Assessments were done once a week.

4.6 Technique of application of electrotherapy modalities

There are some principles in the application of electrotherapy that are similar, and those that are unique to a particular modality. The technique of application and the respective dosages for each of the modalities used in this study is described.

4.6.1 Pulsed shortwave therapy

4.6.1.1 Technique of application

The patient is positioned in a comfortable, supported position and the nature of the treatment explained. The affected hand is adequately exposed and placed between a malleable and a rigid electrode (Plate 5), and the parameters of treatment are selected and set. There is no thermal effect and the patient will feel nothing, however the patient is warned to keep still during the treatment, as movement of the area being treated may alter the electrode position. The resonance is shown by the bright illumination of light on the machine.

Plate 5: Photograph showing the application of PST

4.6.1.2 Dosage

PST was administered using the Curapuls 419 model (Enraf-Nonius-Delft). The pulse power was set at 400 W, with a pulse repetition frequency of 35-62 Hz for a duration of 10-20 minutes.

4.6.2 Laser

4.6.2.1 Technique of application

The nature of the treatment and the need to wear goggles are explained to the patient. The surrounding skin is cleaned to ensure that the skin is dry and clean. The skin may be cleaned with alcohol. In this way the reflection of the laser beam on the skin surface is reduced, and as much energy as possible is deposited in the tissue.

The patient is positioned in sitting so that the wounds are accessible and supported. Laser is applied by a hand held applicator, and the probe is held perpendicularly and as close as possible over the affected area (Plate 6). For large wounds, the area is divided into square centimetres and each area is separately stimulated (Figure 13).

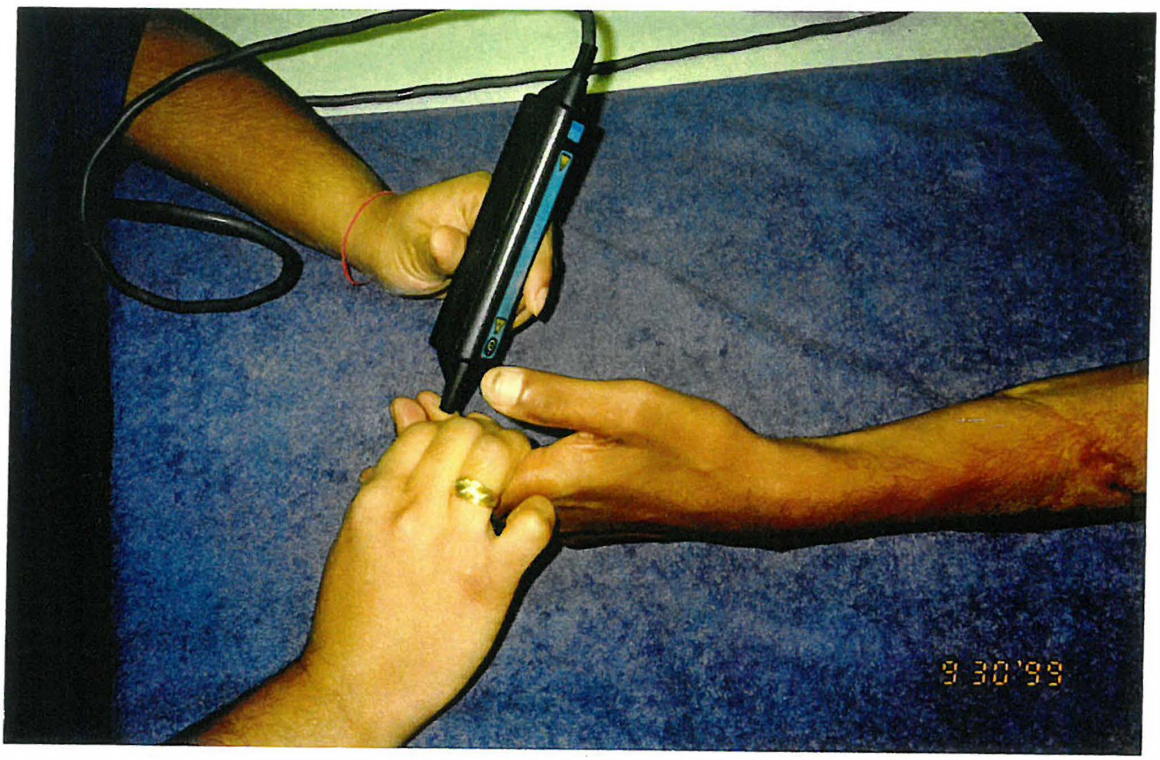


Plate 6: Photograph showing the application of laser

4.6.2.2 Dosage

Laser was administered using the Unilaser 201 model. Each wound was irradiated for 30 seconds per square centimetre (secs/cm^2), using the continuous mode and calculated using the following equation :

$$\text{Energy density (J/cm}^2\text{)} = \frac{\text{emitted mean power of laser (W) x duration (seconds)}}{\text{area of laser beam (cm}^2\text{)}}$$

$$\text{Duration (seconds)} = \frac{\text{Energy density (J/cm}^2\text{) x area of laser beam (cm}^2\text{)}}{\text{emitted mean power of laser (W)}}$$

$$= \frac{30 \text{ J/ cm}^2 \text{ x } 1 \text{ cm}^2}{1 \text{ W}}$$

$$= 30 \text{ seconds/cm}^2$$

4.6.3 Ultraviolet radiation

4.6.3.1 Technique of application

The lamp requires 5 minutes to reach operating temperature and should be switched on at least 5 minutes before treatment is commenced. The patient is positioned in a supported position. All sterile precautions are observed in exposure and handling of the wound is done prior to the treatment. The wound is screened up to its edge, including normal skin, using ultraviolet-resistant material (gauze wet with saline solution). The face of the Kromayer lamp is cleaned with methylated spirits, and when it has had the full 5 minute warming-up period, the lamp is ready for use. The front of the lamp is held as close as possible to the wound and the wound is irradiated for the calculated time. Following treatment the wound is redressed and the lamp is cleaned.

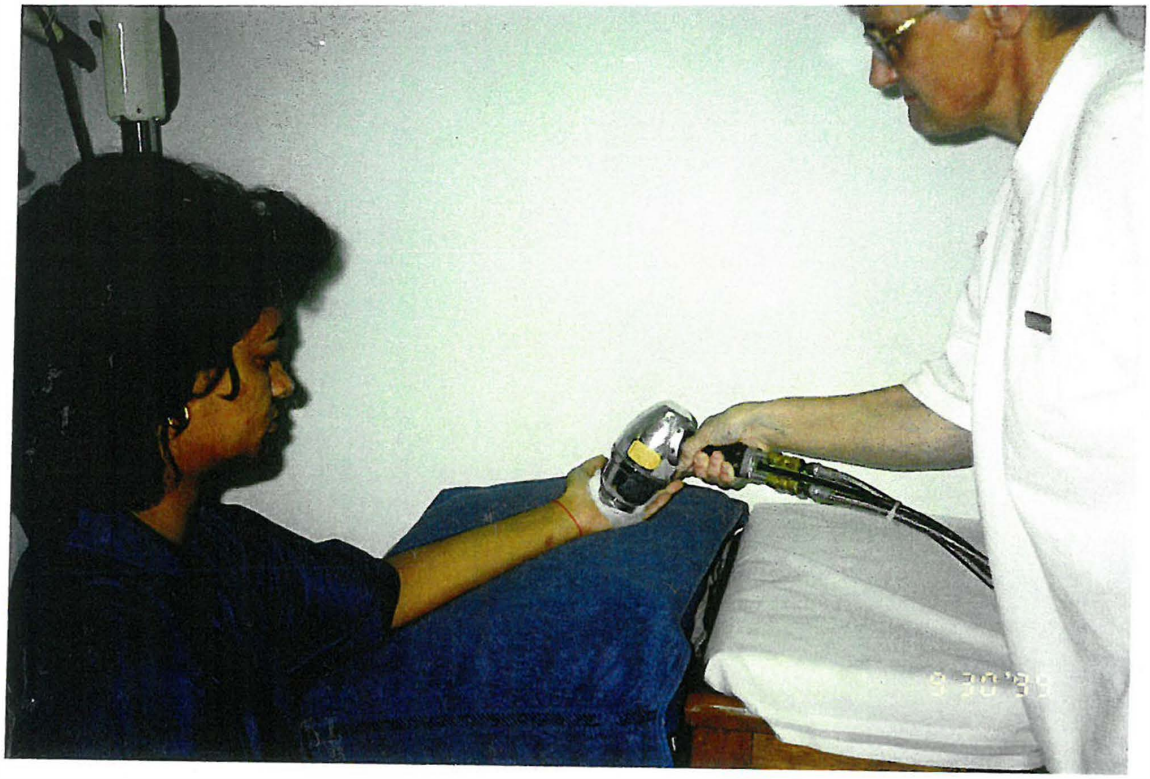


Plate 7: Photograph showing the application of UVR

4.6.3.2 Dosage

Each wound was irradiated for 60 seconds using the Kromayer lamp in contact with the wound.

4.6.4 Infrared radiation

4.6.4.1 Technique of application

The luminous lamp is switched on a few minutes before application to stabilise. The patient is placed in a well-supported position with the area to be treated exposed. The nature and effects of treatment are explained to the patient. The lamp is positioned so that the radiation strikes the surface at right angles and at a distance of 60 cm from the area being treated (Plate 8). The patient is warned that he/she should not touch the lamp or move nearer to it. At the end of treatment the skin should feel mildly warm.

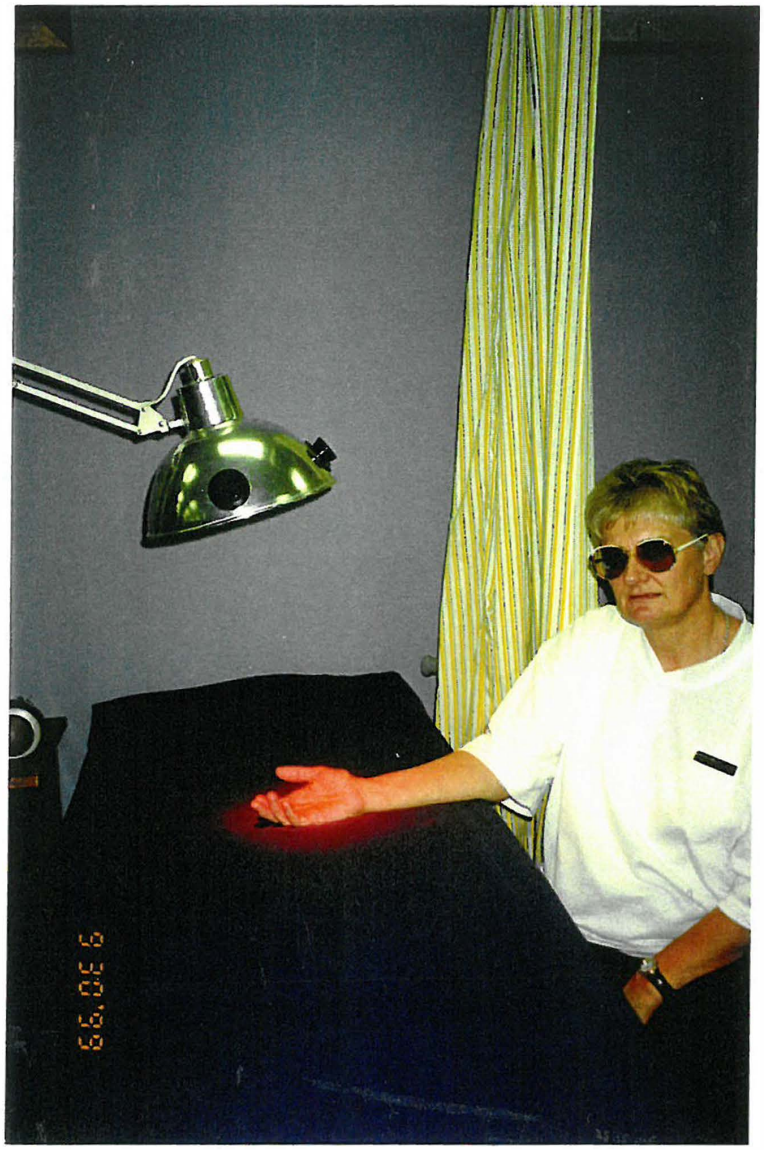


Plate 8: Photograph showing the application of IRR

4.6.4.2 Dosage

Treatment was administered using the Phillips infrared lamp at a distance of 60 cm from the wound being treated, with a treatment time of 10-15 minutes.

Steven L. Wolf, series editor of the well documented text Wound healing : Alternatives in Management (Kloth *et al.*, 1990) writes in the forward "Physiotherapists and other health professionals play a vital role in the rehabilitation of patients with wounds secondary to trauma, metabolic disorders or prolonged pressure." He explains that research has demonstrated the therapeutic value of some electrotherapy modalities to the facilitation of cutaneous and subcutaneous healing.

CHAPTER 5

5. RESULTS

The final sample size consisted of 125 patients with open wounds on the volar aspect of the hand, with 25 patients in each of the five groups. All wounds were sustained less than one month before admission to the study. All injuries were crush injuries to the hand which were sustained at the workplace.

The results are presented in this chapter as follows :-

5.1 Age distribution
5.2 Male : Female ratio
5.3 Traces
5.4 Photographs (Plates)
5.5 Measurement of wound healing
    Tables
    Figures (Line graphs)
5.6 Pain measurement
    Tables
    Figures (Line graphs)

The raw data is presented in Appendix 2 and 3. The data collected was analysed using repeated measures analysis of variance (ANOVA) and pairwise comparisons by least significant differences.

5.1 Age Distribution

The age range of patients included in the study is 20 - 59 years. Table 4 shows the mean age distribution of subjects in the 5 zones and in the 5 groups.

Table 4 : Mean age and standard deviation of subjects for all groups in zone I to zone V

		ZONE I		ZONE II		ZONE III		ZONE IV		ZONE V	
TREATMENT GROUP	No. of pts. per zone	MEAN AGE	STD. DEV.	MEAN AGE	STD. DEV.	MEAN AGE	STD. DEV.	MEAN AGE	STD. DEV.	MEAN AGE	STD. DEV.
PST	5	37.6	6.5	40.4	11.4	33.6	8.6	44.4	9.4	40.6	9.2
LASER	5	37.0	9.5	38.8	9.6	36.2	10.1	35.2	5.9	40.2	11.0
UVR	5	32.8	8.7	40.0	10.5	38.6	7.0	38.0	11.5	37.6	8.0
IRR	5	35.6	11.3	36.2	9.9	36.4	8.6	39.0	9.9	34.2	8.3
CONTROL	5	39.4	10.2	36.8	10.3	31.0	4.9	35.8	8.2	35.8	8.5

Differences were not statistically significant.

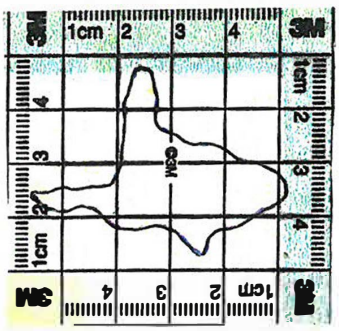
5.2 Male : Female ratio

A total number of 125 patients were included in the study. 120 were male patients and 5 were female patients.

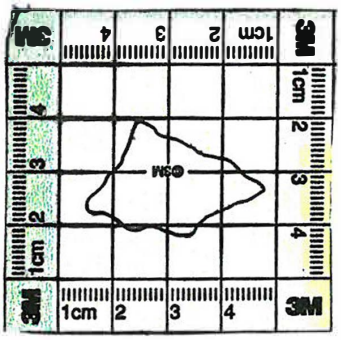
5.3 Traces

Traces were obtained once a week and recorded as in 4.4.1.1 and Plate 2. Traces were taken of all wounds and these were placed together for comparison of wound healing. Examples of the traces obtained at weekly evaluation for the PST and control groups are presented in Plates 9 (a) and (b) respectively. As seen in the two series, there is a decrease in wound size over the consecutive weeks. The traces for all groups in each of the 5 zones showed an decrease in wound size.

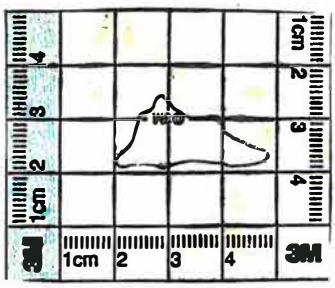
WEEK 0



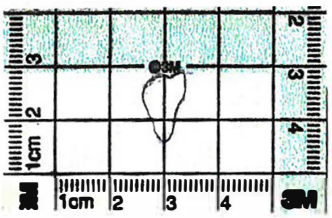
WEEK 1



WEEK 2



WEEK 3



WEEK 4

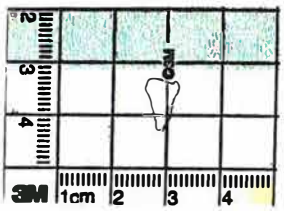
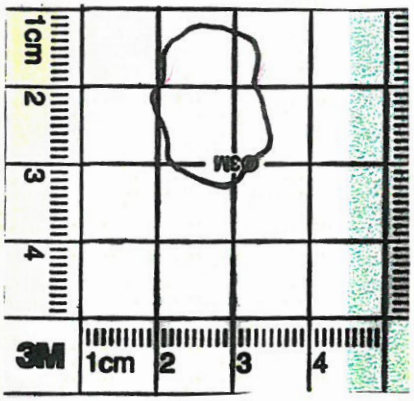
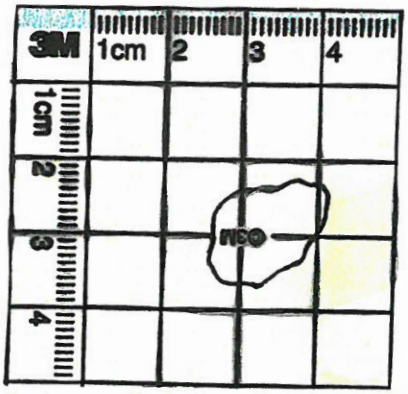


Plate 9 (a) : A series of traces of wounds in zone III in the PST group

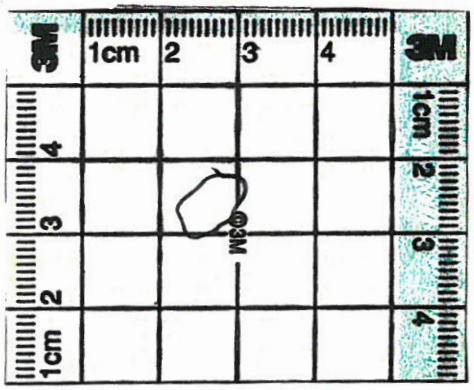
WEEK 0



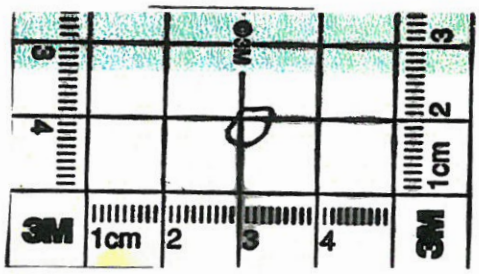
WEEK 1



WEEK 2



WEEK 3



WEEK 4

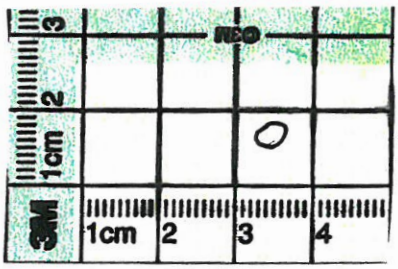


Plate 9 (b) : A series of traces of wounds in zone II in the control group

5.4 Photographs

A series of photographs was taken of each wound at evaluation and at weekly intervals. Not all photographs are shown. The example shown in Plate 10 (a) are a series of photographs taken of a patient in the PST group in Zone II. Plate 10 (b) shows a series of photographs taken of a patient in the control group in Zone V. There is a decrease in wound size from week 0 to week 5. Serial photographs of patients in all groups were taken. Visual examination of the photographs showed a decrease in wound size over time.

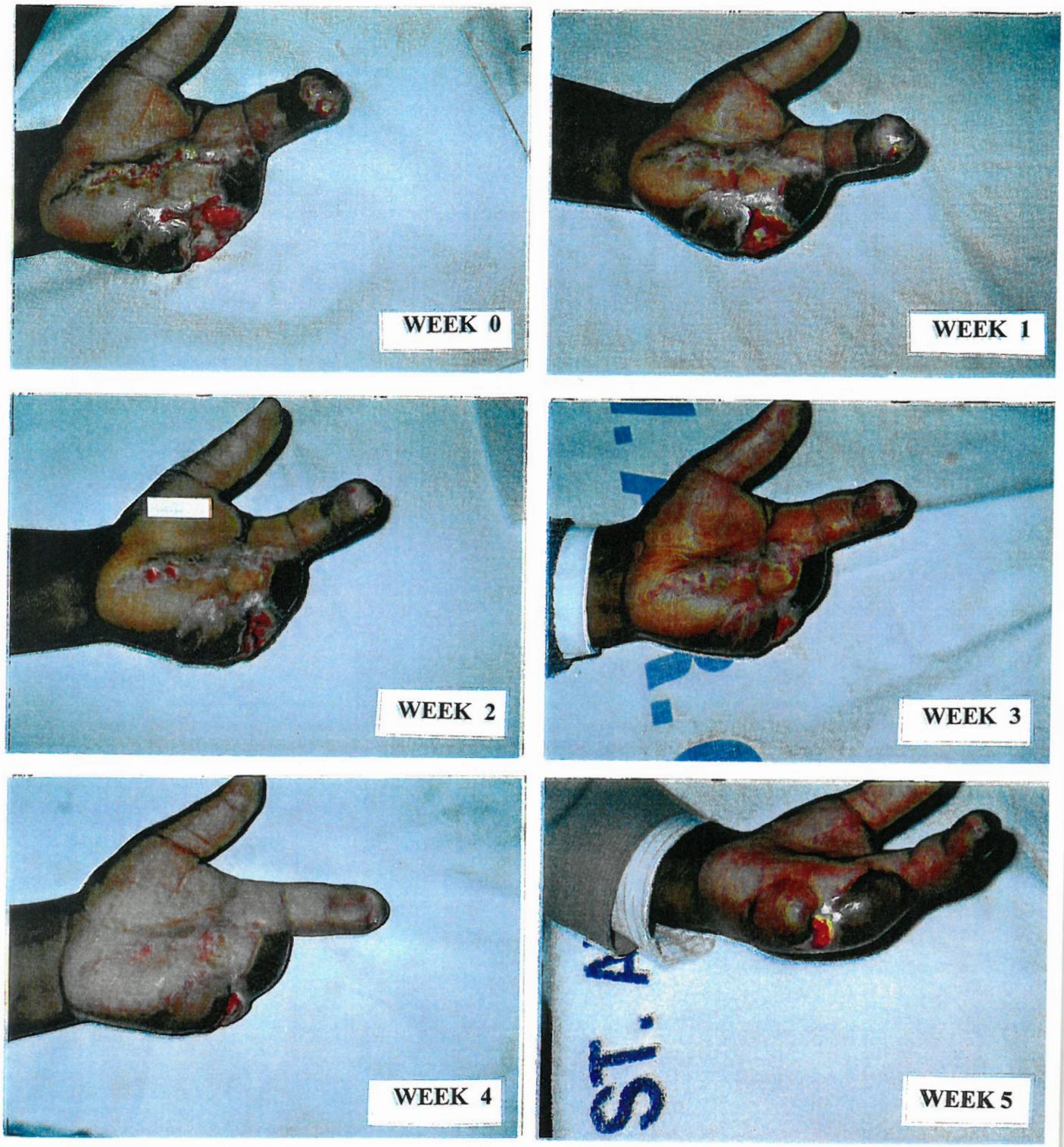


Plate 10 (a) : A series of photographs taken of a patient in zone II in the PST group

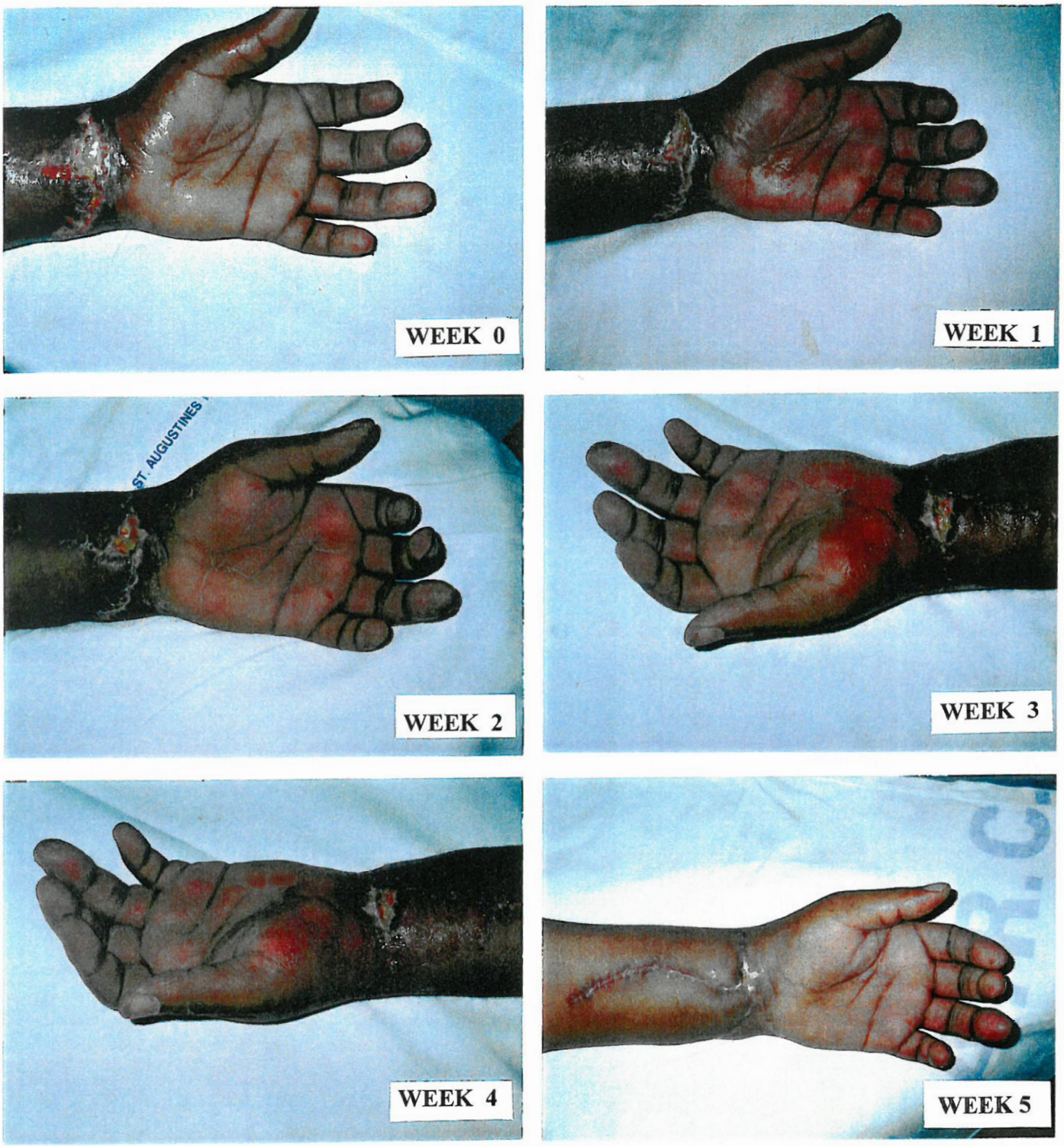


Plate 10 (b) : A series of photographs taken of a patient in zone V in the control group

5.5 Measurement of wound healing

The papers traces of the wounds were weighed and recorded. An example is listed in Table 5(a). Assuming that the wound at the commencement of treatment was 100%, the percentage wound surface area was calculated for each wound once weekly. This is represented in Table 5(b).

Table 5 (a): Table showing weight of traces for each patient in Zone I in the PST group

PATIENT	*WEEK 0*	*WEEK 1*	*WEEK 2*	*WEEK 3*	*WEEK 4*
1	0.37	0.22	0.17	0.11	0.06
2	0.93	0.61	0.40	0.15	0.07
3	2.66	1.76	0.98	0.23	0.08
4	0.98	0.69	0.26	0.06	0.02
5	3.49	2.52	1.71	0.67	0.10

Table 5 (b) : Percentage remaining wound surface area for each patient in Zone I in PST group

PATIENT	*WEEK 0*	*WEEK 1*	*WEEK 2*	*WEEK 3*	*WEEK 4*
1	100	59.5	45.9	29.7	16.2
2	100	65.6	43.0	16.1	7.5
3	100	66.2	36.8	8.6	3.0
4	100	70.4	26.5	6.1	2.0
5	100	72.2	49.0	18.3	5.1
AVERAGE	**100**	**66.8**	**40.2**	**15.8**	**6.8**

Week 0 shows the original wound size to be 100 %, and the subsequent wound size is expressed as a percentage of the original wound. The average wound healing was calculated for all the patients per week. The average percentage wound healing (W.H.) for each group in each zone per week was calculated. Table 6 shows the calculation for Zone I in the PST group. Figure 27 illustrates graphically the results obtained in zone I for all groups.

Table 6 : **Mean percentage remaining wound surface area and standard deviation per week for all groups in zone I**

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV
PST	100	0	66.8	4.9	40.2	8.9	15.8	9.3	6.8	5.7
LASER	100	0	68.1	14.1	49.3	11.6	27.1	10.4	11.5	4.0
UVR	100	0	71.3	19.7	41.0	30.7	30.4	36.7	20.6	31.8
IRR	100	0	69.9	7.6	50.4	8.1	28.0	6.9	14.3	5.5
CONTROL	100	0	82.7	7.0	61.8	8.6	40.9	9.4	24.9	8.9

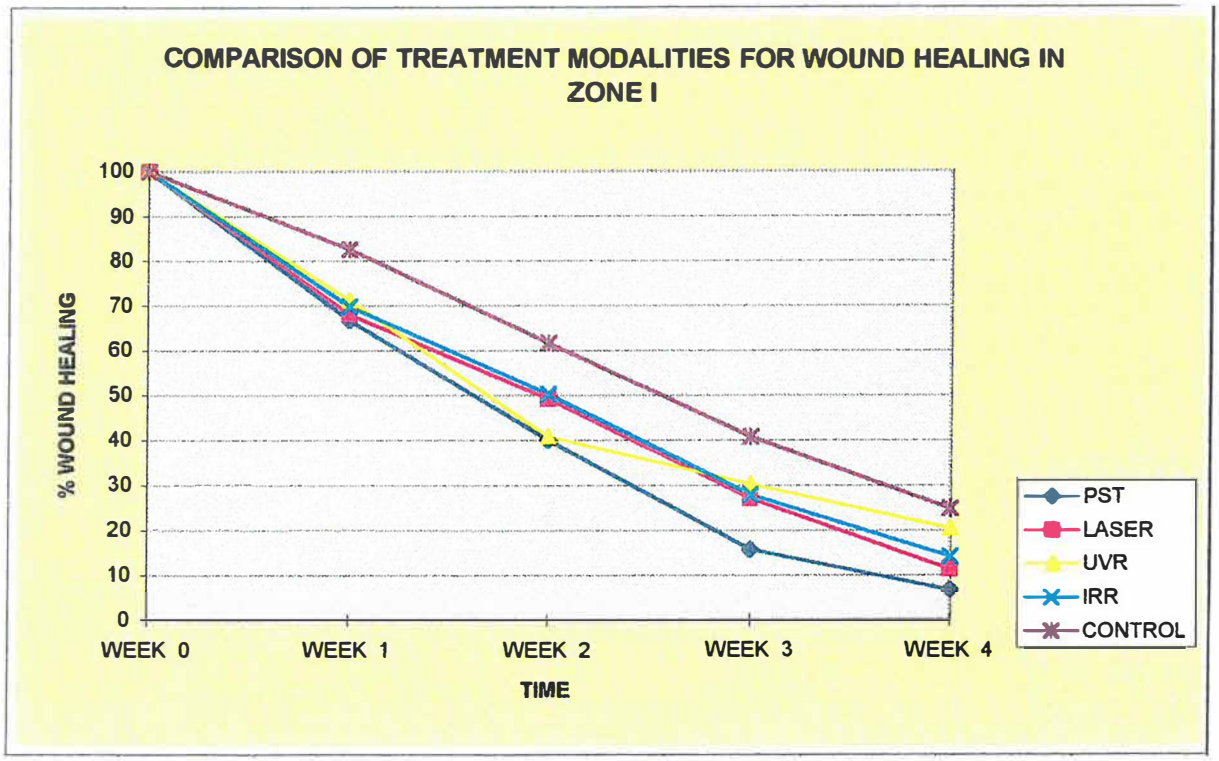


Figure 27 : **Graph illustrating the average percentage remaining wound surface area for all groups in zone I**

Figure 27 illustrates graphically the calculated average percentage wound healing in zone I.

In the analysis of the data for percentage wound healing in all the groups in zone I per week over 4 weeks, no statistical difference was found in the different treatment groups. Repeated measures analysis of variance using MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect in zone I showed significant change over time with a p value of 0.0001 for all groups. TIME*TREATMENT interaction, that is, changes over time, did not differ significantly between the groups.

There was a significant change between the groups in zone I from week 0-1 ($p = 0.0001$), week 0 and 2 ($p = 0.0001$), week 0 and 3 ($p = 0.0001$), but no significant difference was found between the groups at week 1, week 2, week 3 and 4. Differences between treatments with regards to these changes were not statistically significant.

Table 7 : **Mean percentage of remaining wound surface area and standard deviation per week for all groups in zone II**

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV
PST	100	0	50.7	15.4	31.9	18.4	11.7	9.6	3.1	2.9
LASER	100	0	63.1	10.9	48.2	17.8	30.9	14.3	12.2	6.6
UVR	100	0	70.8	9.2	37.1	12.9	17.9	10.5	11.3	7.9
IRR	100	0	79.2	8.0	62.0	12.4	46.1	16.7	30.9	19.2
CONTROL	100	0	74.0	16.6	47.8	10.6	32.2	13.7	20.1	1.0

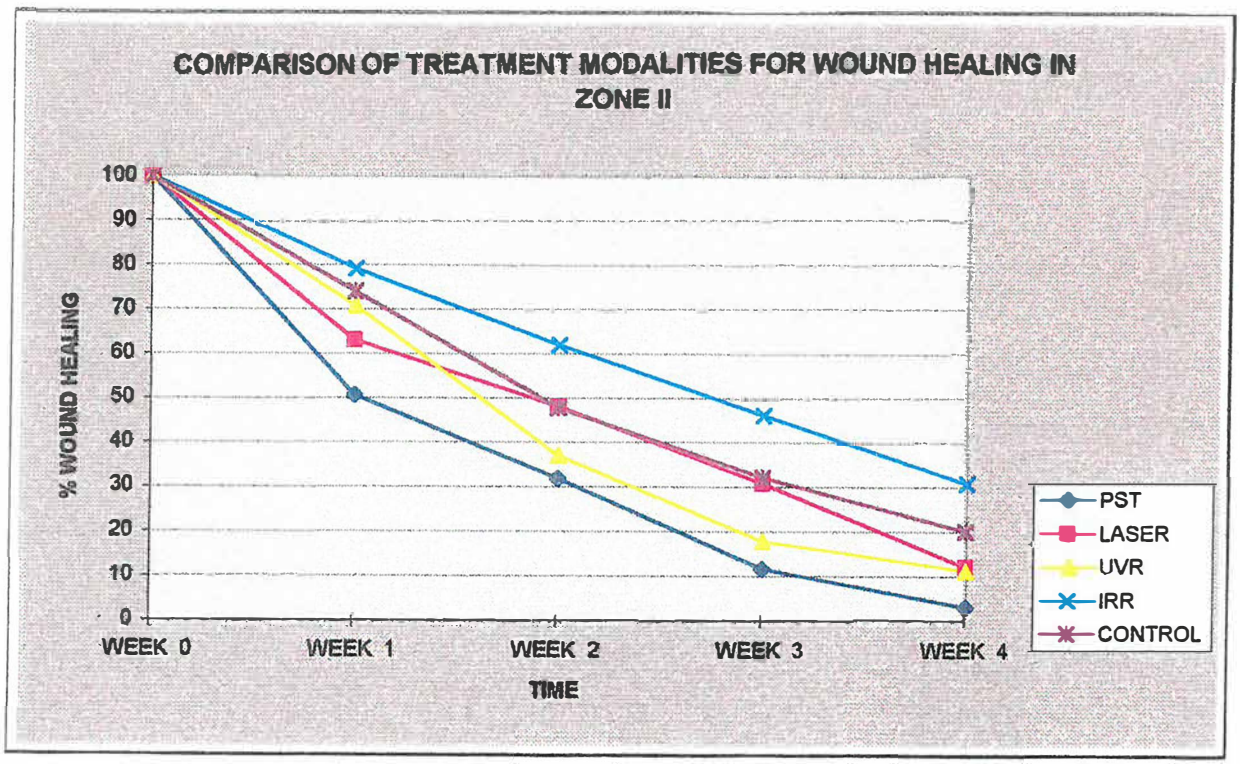


Figure 28 : **Graph illustrating mean percentage of remaining wound surface area per week for all groups in zone II**

Figure 28 illustrates the comparative performance of each group per week in zone II.

Repeated measures analysis of variance for wound healing in zone II yielded the following :-

Percentage wound healing showed statistical significance between treatment groups at week 1 ($p = 0.0166$), week 2 ($p = 0.0393$), week 3 ($p = 0.0051$) and week 4 ($p = 0.0076$). MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect yielded significant change over time ($p = 0.0001$). MANOVA criteria and F approximations for the hypothesis of no TIME * TREATMENT effects showed a significant change over time which was different between groups ($p = 0.0169$).

There was significant change from week 0 to 1 ($p = 0.0001$) with statistically significant group differences at week 1. Week 0 to 2 also showed significant change ($p = 0.0001$), week 0 to 3 ($p = 0.0001$), week 0 to 4 ($p = 0.0001$), with statistically significant group differences at week 2 ($p = 0.0393$), week 3 ($p = 0.0051$) and week 4 ($p = 0.0046$).

Pairwise comparisons using least significant differences for zone II yielded the following :-

Wound healing at week 1 showed the PST group to be significantly better than UVR, IRR and the control treatment groups. At week 2 the PST and UVR treatment groups are significantly better than the IRR group. Week 3 showed the PST group to be significantly better than the laser, IRR and control groups. Week 4 showed statistical significance for the PST group performing better than the IRR and control groups.

Table 8 : **Mean percentage of remaining wound surface area and standard deviation per week for all groups in zone III**

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV
PST	100	0	63.6	17.8	25.3	12.1	10.3	6.4	2.9	3.1
LASER	100	0	73.3	18.0	49.6	14.2	20.7	11.0	6.7	4.4
UVR	100	0	68.8	17.3	56.4	16.8	34.5	12.8	18.3	8.5
IRR	100	0	73.0	5.9	45.6	7.1	30.5	8.1	16.7	5.3
CONTROL	100	0	74.7	14.0	58.7	7.4	37.3	9.7	17.9	8.6

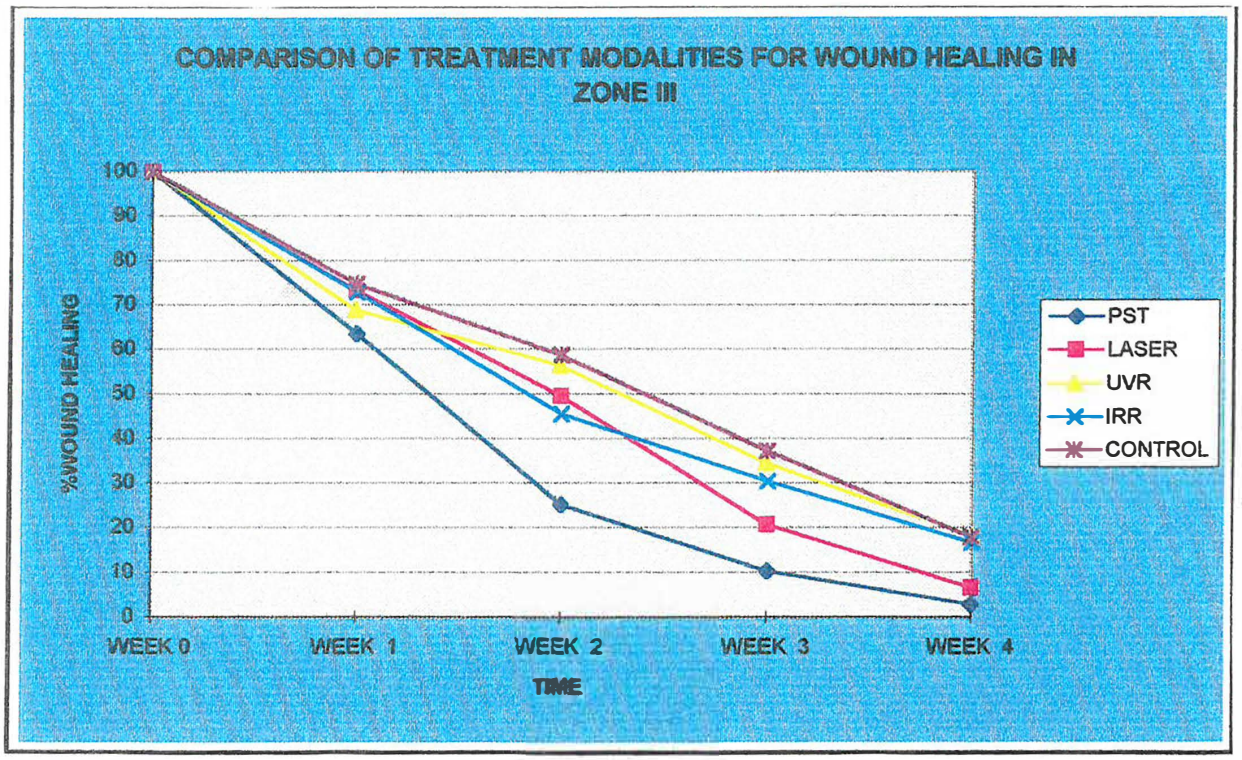


Figure 29 : **Graph illustrating the mean percentage of remaining wound surface area per week for all groups in zone III**

Figure 29 illustrates the graphical representation of the average percentage wound healing in all groups in zone III.

Repeated measures analysis of variance for wound healing in zone III yielded the following :-

There was no significant difference between the treatments at week 1 ($p = 0.7397$) in the different treatment groups. Week 2 ($p = 0.0024$), week 3 ($p = 0.0018$), week 4 ($p = 0.0018$) showed significant change for all groups.

MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect was significant for zone III ($p = 0.0055$).

There was significant change in the treatment groups from week 0 to week 1 ($p = 0.0001$). Change from week 0 to 2 was significant ($p = 0.0001$), week 0 to 3 ($p = 0.0001$) and week 0 to 4 ($p = 0.0001$). Treatments were also different with regards to these changes : week 0 to 2 ($p = 0.0024$), week 0 to 3 ($p = 0.0018$) and week 0 to 4 ($p = 0.0018$).

Pairwise comparisons using least significant differences for zone III yielded no significant difference in the treatment groups for week 1. At week 2, PST yielded significantly better wound healing than laser, UVR, IRR and the control groups. PST was significantly better than UVR, IRR and the control groups at week 3. At week 4, PST and laser performed better than UVR, IRR and the control groups, but there was no significant difference between these two groups.

Table 9 : Mean percentage of remaining wound surface area and standard deviation for all groups in zone IV

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV
PST	100	0	59.2	27.9	36.0	21.6	18.7	14.2	5.4	7.5
LASER	100	0	62.4	12.3	49.5	7.5	24.9	5.7	12.9	3.4
UVR	100	0	74.9	10.0	55.7	9.1	34.4	13.1	23.4	11.1
IRR	100	0	76.7	10.6	54.1	11.1	37.8	8.9	18.3	6.0
CONTROL	100	0	74.7	12.7	54.6	16.1	34.9	25.9	18.7	23.1

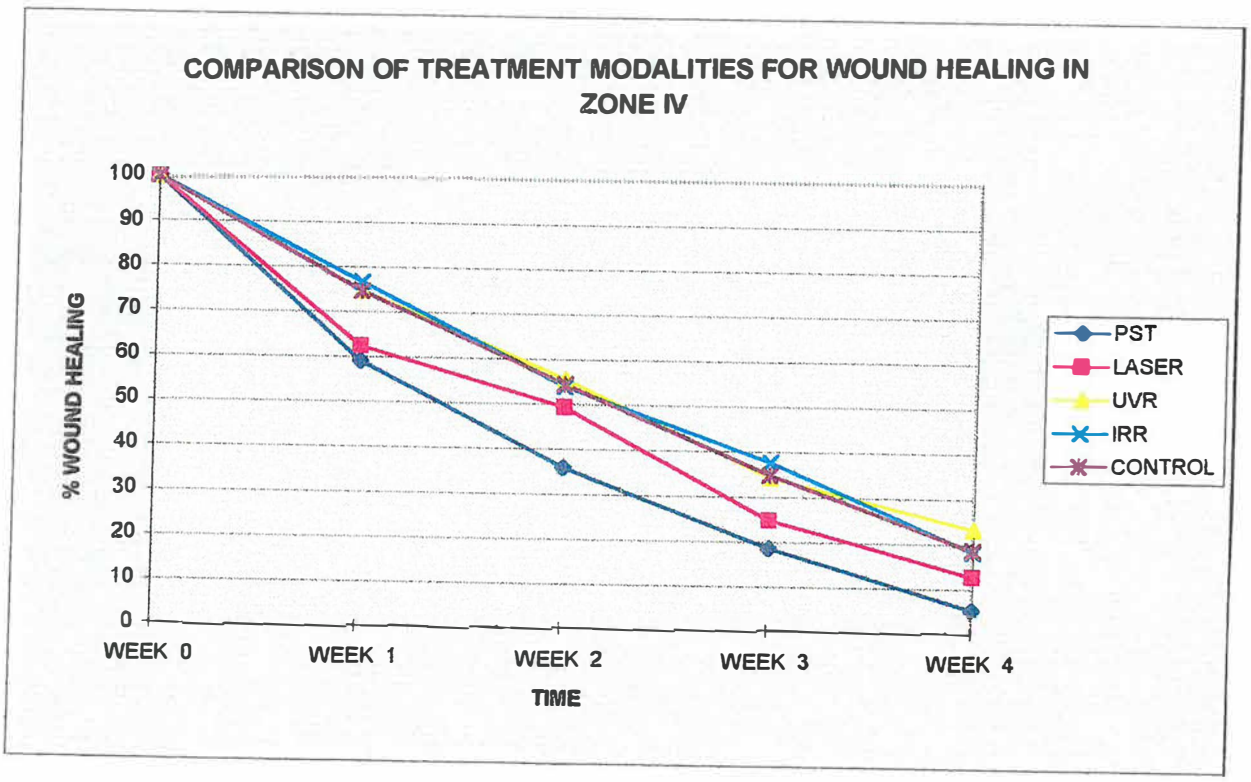


Figure 30 : Graph illustrating mean percentage of remaining wound surface area for all groups in zone IV

Figure 30 illustrates graphically comparative performance of the different treatment groups in zone IV.

Analysis of wound healing for all groups in zone IV per week showed no statistical significance at week 1, week 2, week 3, or 4. MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect yielded a p value of 0.0001, showing a significant change for all groups. MANOVA test criteria and F approximations for the hypothesis of no TIME* TREATMENT interaction found no statistical significance ($p = 0.0934$).

Time changes between week 0 to 1, week 1 to 2, week 2 to 3, and week 3 to 4 were all significant ($p = 0.0001$). However, differences between treatments with regards to these changes were not significant.

Table 10 : Mean percentage of remaining wound surface area and standard deviation for all groups in zone V

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV
PST	100	0	71.1	12.7	39.9	12.4	21.8	12.6	9.4	7.4
LASER	100	0	68.0	14.2	35.6	5.5	18.3	6.7	8.5	4.4
UVR	100	0	64.7	16.7	44.4	26.1	27.1	32.2	16.5	25.4
IRR	100	0	81.7	3.7	53.5	10.7	40.5	6.1	28.8	3.3
CONTROL	100	0	80.7	9.3	60.3	15.1	43.6	13.2	27.8	11.6

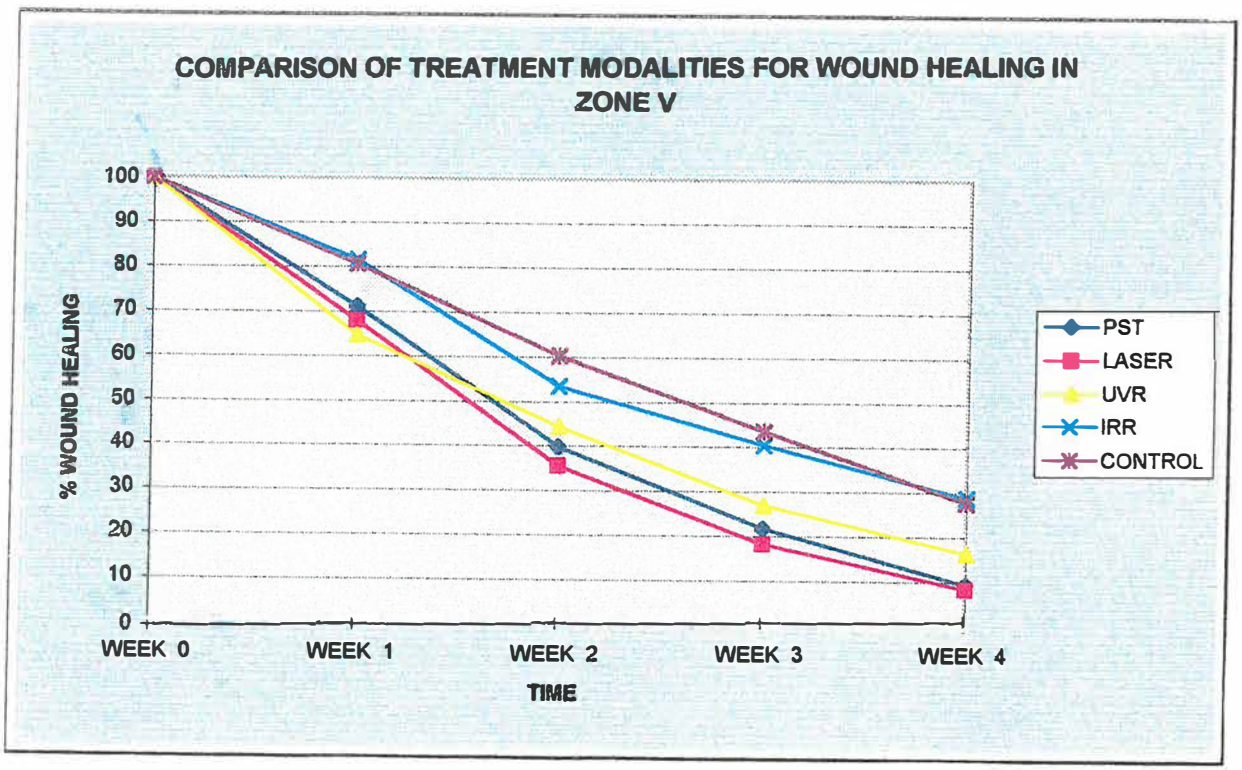


Figure 31 : Graph illustrating the mean percentage of remaining wound surface area for all groups in zone V

Figure 31 is the graphical illustration of wound healing in the different groups in zone V.

Analysis of variance tests for wound healing in zone V at each week yielded the following :-

Percentage wound healing between the treatment groups at week 1, week 2 and 3 were not significant. Marginal statistical significance was found for comparison between groups at week 4 with a p value of 0.059 and an F value of 2.72.

Repeated measures ANOVA for the dependent variable was done. MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect yielded statistical significance with a p value of 0.0001.

MANOVA test criteria and F approximations for the hypothesis of no TIME * TREATMENT effect yielded no statistical significance (p = 0.3568).

Repeated measures ANOVA for the time variable was statistically significant for all the weeks (p = 0.0001). There was significant change from week 0 to 4 (p = 0.0500) supporting the differences between the groups with regards to changes in time.

Table 11 : Mean percentage of remaining wound surface area and standard deviation for all treatment groups in all zones

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV	% WH	STD. DEV
PST	100	0	62.3	17.4	34.7	15.2	15.6	10.7	5.5	5.7
LASER	100	0	67.0	13.5	46.4	12.4	24.4	10.3	10.4	4.9
UVR	100	0	69.7	14.3	46.9	20.6	28.9	22.6	18.0	18.4
IRR	100	0	76.1	8.1	53.1	10.7	36.6	11.4	21.8	11.2
CONTROL	100	0	77.3	11.9	56.6	12.2	37.8	14.8	21.9	13.0

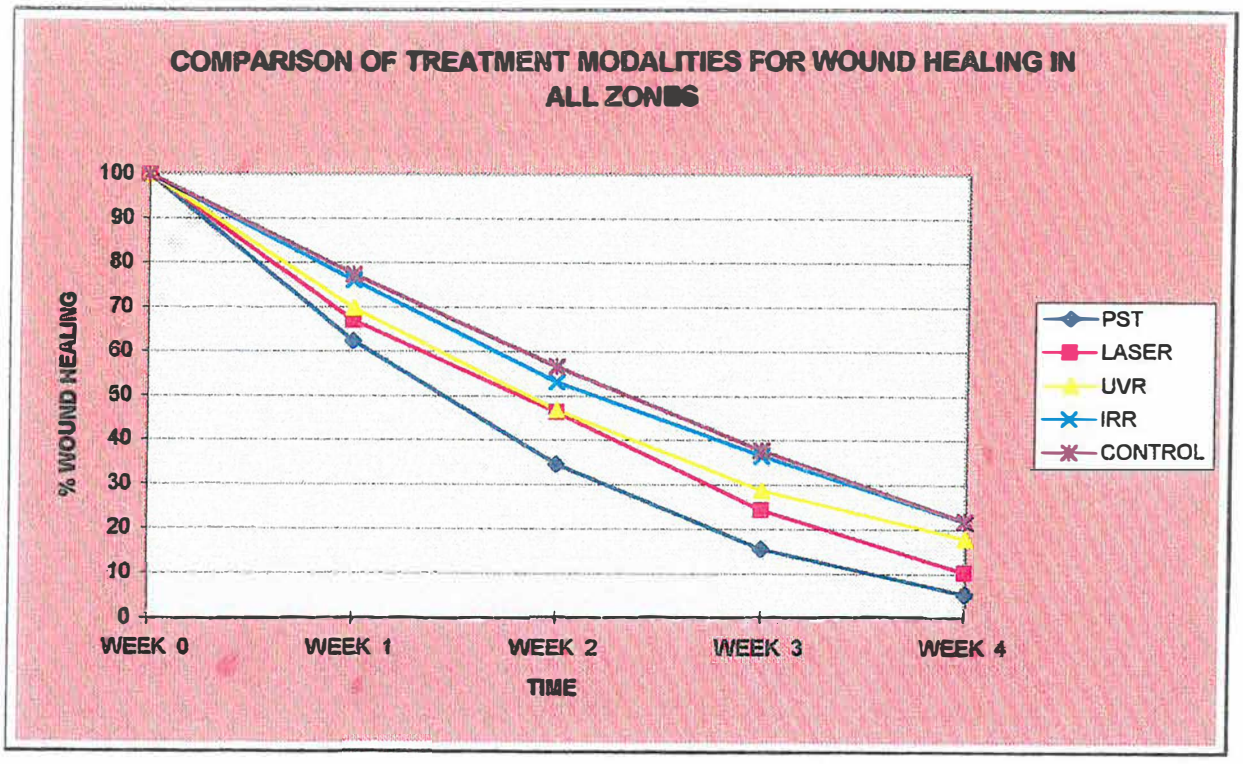


Figure 32 : Graph illustrating mean percentage of remaining wound surface area for all treatment groups in all zones

The calculation of the means by treatment with the combination of the zones, is illustrated in the Figure 32.

Two-way repeated measures analysis of variance testing for differences between treatments, while controlling for zones yielded the following :-
Wound healing at week 1 was significant with a p value of 0.005 and F value of 5.44. Treatments at week 2 ($p = 0.0001$), week 3 ($p = 0.0001$) and week 4 ($p = 0.0001$) were all statistically significant for wound healing.

MANOVA test criteria for hypothesis of no TIME effect and no TIME * TREATMENT effect were significant ($p = 0.0001$).
MANOVA test criteria for hypothesis of TIME * ZONE effect was not significant ($p = 0.6162$). Repeated measures ANOVA for hypotheses of between subjects effects was significant ($p = 0.0001$).

Repeated measures ANOVA for the variables of time, treatment and zone yielded the following:-
There were significant time differences between week 0 and 1, week 0 and 2, week 0 and 3, and week 0 and 4. There were also significant differences between the treatment groups. No significant difference was found between the zones.

T tests for least significant differences between the groups was determined. At week 1 and 4, wound healing in the PST and laser groups were found to be significantly better than UVR, IRR and the control groups. At week 2 and 3 wound healing in the PST group was significantly better than the laser and control treatment groups (but not significant when tested against the UVR and IRR groups). Laser was also significantly different to the control group (but not the UVR and IRR groups) at week 2 and 3.

5.6 PAIN MEASUREMENT

Pain was measured once weekly on the numerical rating scale. The average measurements calculated per week for each group is listed in Appendix 3. Graphical representation is illustrated for each zone.

Table 12 : Mean pain measurement and standard deviation for all groups per week in zone I

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV
PST	6.6	1.14	5.4	1.52	3.8	1.48	1.6	1.34	0.8	1.09
LASER	7.6	2.51	6.6	3.05	6.4	3.21	5.6	3.78	4.8	3.56
UVR	8.4	1.67	8.0	1.87	6.2	1.64	5.6	0.89	4.4	0.89
IRR	6.6	2.07	6.2	2.8	5.2	2.17	5.0	2.74	4.6	2.97
CONTROL	8.2	1.30	7.2	1.64	6.2	2.95	5.8	3.03	5.2	2.59

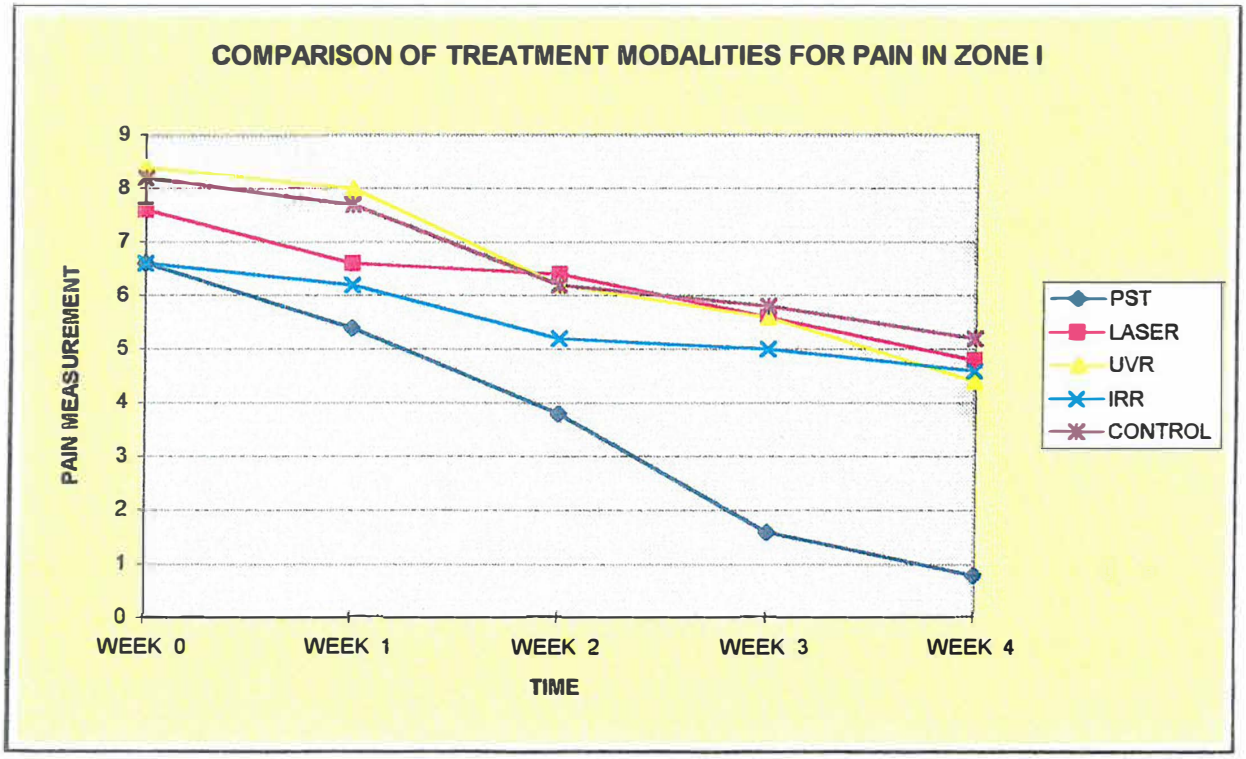


Figure 33 : Graph illustrating mean pain measurement per week for all groups in zone I

The graphical illustration of the pain measurement in all groups in zone I is shown in Figure 33.

Analysis of pain measurement as recorded on NRS for zone I is as follows :-
The dependent variable of pain measurement at week 0, week 1, week 2 and week 3 showed no significance between the treatment groups. There was a significant difference between the groups at week 4.

Repeated measures MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect was significant for zone I ($p = 0.0001$). MANOVA test criteria and F approximations for the hypothesis of no TIME * TREATMENT effect was not significant ($p = 0.1982$), that is, changes over time did not differ significantly between groups. Repeated measures ANOVA tests of hypotheses for between subjects effects was not significant ($p = 0.2136$).

There is a significant change in pain measurement between the groups in zone I from week 0 to 1 ($p = 0.0001$), week 0 to 2 ($p = 0.0001$), week 0 to 3 ($p = 0.0001$) and week 0 to 4 ($p = 0.0001$). Significant differences were found between the groups at week 3 ($p = 0.0539$) and week 4 ($p = 0.0052$), but no significant difference was found at week 1 and week 2.

Pairwise comparisons using least significant differences for zone I yielded the following for the different treatment groups :
Pain measurement at week 0, week 1 and week 2 was not significantly different for the different treatment groups. The PST treatment group was significantly better than the other groups at week 3 and week 4.

TABLE 13 : Mean pain measurement and standard deviation for all groups per week in zone II

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV
PST	7.2	1.79	6.2	1.64	4.4	1.95	3.2	2.17	2.4	1.52
LASER	7.6	1.67	6.8	1.92	6.6	1.95	6.2	1.48	4.8	1.48
UVR	7.4	1.95	6.8	1.92	4.6	0.55	5.6	1.14	4.20	0.84
IRR	8.2	1.79	8.0	1.73	7.0	1.22	6.0	2.00	5.4	1.67
CONTROL	7.8	2.28	7.6	2.07	6.8	1.79	6.0	1.58	6.2	1.30

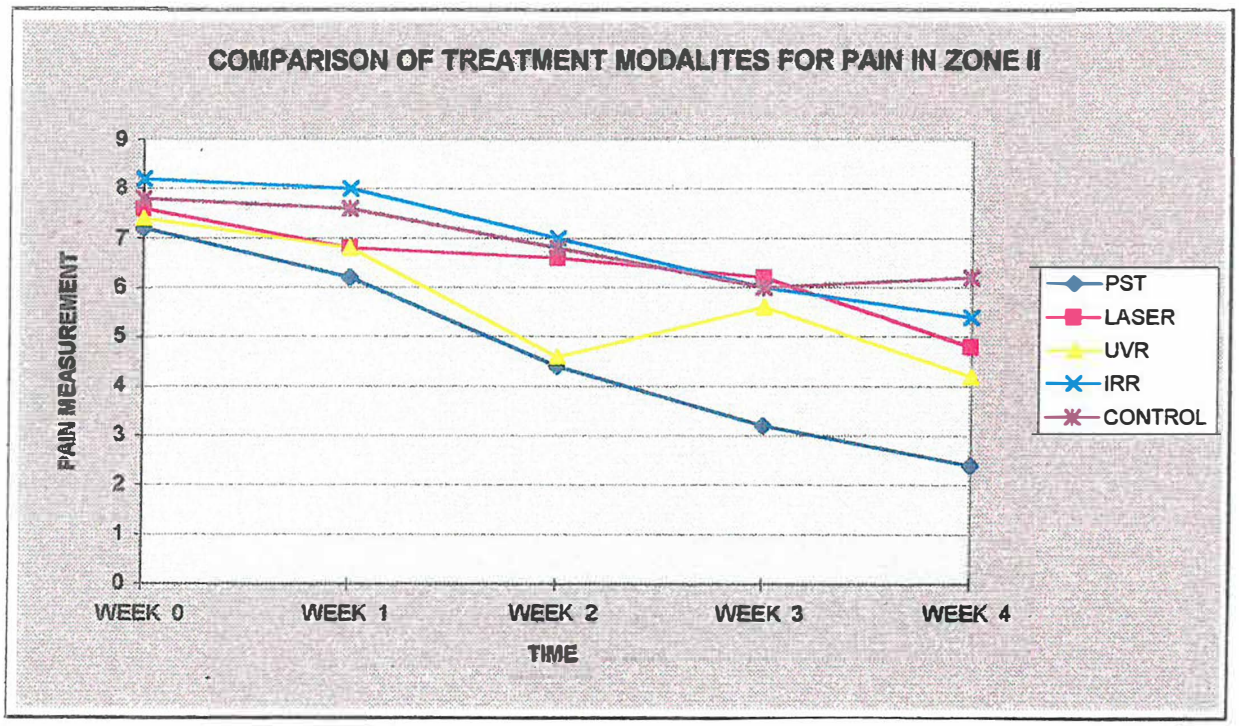


Figure 34 : Graph illustrating mean pain measurement for all groups per week in zone II

Graphic illustration of the pain measurement in the different treatment groups in zone II is shown in Figure 34.

Analysis of pain measurement as recorded on NRS for zone II is as follows :-
The dependent variable of pain measurement at week 0 and week 1 showed no significance between the treatment groups. There was a significant difference between the groups at week 2 ($p = 0.0349$), week 3 ($p = 0.0533$) and week 4 ($p = 0.0044$).

Repeated measures MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect was significant for zone I ($p = 0.0001$). MANOVA test criteria and F approximations for the hypothesis of no TIME * TREATMENT effect was significant ($p = 0.0141$). Repeated measures ANOVA tests of hypotheses for between subjects effects was not significant ($p = 0.1429$).

There was a significant change in pain measurement in zone II from week 0 to 1 ($p = 0.0224$), week 0 to 2 ($p = 0.0001$), week 0 to 3 ($p = 0.0001$) and week 0 to 4 ($p = 0.0001$). Significant differences were found between the groups at week 4 ($p = 0.0057$), but no significant difference was found between the groups at week 1, week 2 and week 3.

Pairwise comparisons using least significant differences for zone II yielded the following for the different treatment groups :
Pain measurement at week 0 and week 1 was not significantly different for the different treatment groups. The PST treatment group had significantly lower pain than the other groups at week 2, week 3 and week 4.

Table 14 : Mean pain measurement and standard deviation for all groups per week in zone III

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DE
PST	7.6	2.07	6.0	1.58	4.6	1.67	3.4	1.14	0.6	0.89
LASER	8.2	2.05	7.4	1.67	6.2	1.79	5.4	2.41	4.4	1.95
UVR	8.0	1.58	8.0	1.58	6.4	1.67	6.2	1.79	5.0	0.71
IRR	7.8	2.59	7.0	2.55	6.2	1.64	5.6	2.70	5.4	2.30
CONTROL	6.8	1.64	6.0	2.45	5.6	1.82	5.0	2.82	4.8	2.59

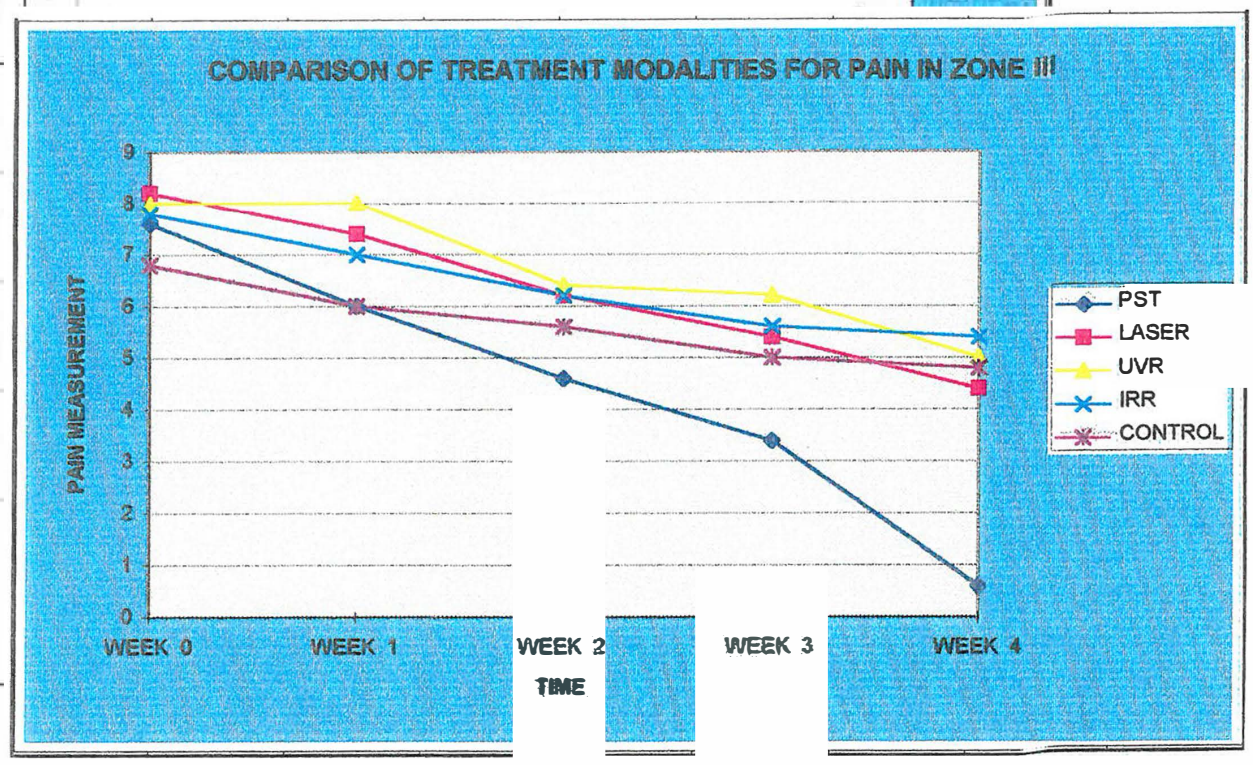


Figure 35 : Graph illustrating mean pain measurement for all groups per week in zone III

Figure 35 shows graphically the pain measurement for the different treatment groups in zone III.

Analysis of pain measurement as recorded on NRS for zone III is as follows :-
The dependent variable of pain measurement at week 0 week 1, week 2 and week 3 showed no significance between the treatment groups. There was a significant difference between the groups at week 4 ($p = 0.0003$).

Repeated measures MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect was significant for zone I ($p = 0.0001$). MANOVA test criteria and F approximations for the hypothesis of no TIME * TREATMENT effect was not significant ($p = 0.1082$). Repeated measures ANOVA tests of hypotheses for between subjects effects was not significant ($p = 0.2065$).

There was a significant change in pain measurement in zone III from week 0 to 1 ($p = 0.0136$), week 0 to 2 ($p = 0.0001$), week 0 to 3 ($p = 0.0001$) and week 0 to 4 ($p = 0.0001$). Significant differences were found between the groups at week 3 ($p = 0.0211$) and week 4 ($p = 0.0019$), but no significant difference was found between the groups at week 1 and week 2.

Pairwise comparisons using least significant differences for zone III yielded the following for the different treatment groups :
Pain measurement at week 0 and week 1 was not significantly different for the different treatment groups. The PST treatment group was significantly better than the other groups at week 2, week 3 and week 4.

Table 15 : Mean pain measurement and standard deviation for all groups in zone IV

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV
PST	8.6	1.34	6.6	1.14	5.0	1.58	3.2	1.09	1.4	1.14
LASER	8.0	1.22	7.6	1.67	6.6	1.34	6.0	2.45	4.2	1.30
UVR	8.0	2.12	7.2	1.92	5.8	1.30	5.6	0.89	5.4	0.55
IRR	6.8	1.64	6.0	2.45	5.6	1.82	5.0	2.83	4.8	2.59
CONTROL	8.2	1.48	7.8	1.48	6.8	1.48	6.2	1.79	5.4	0.89

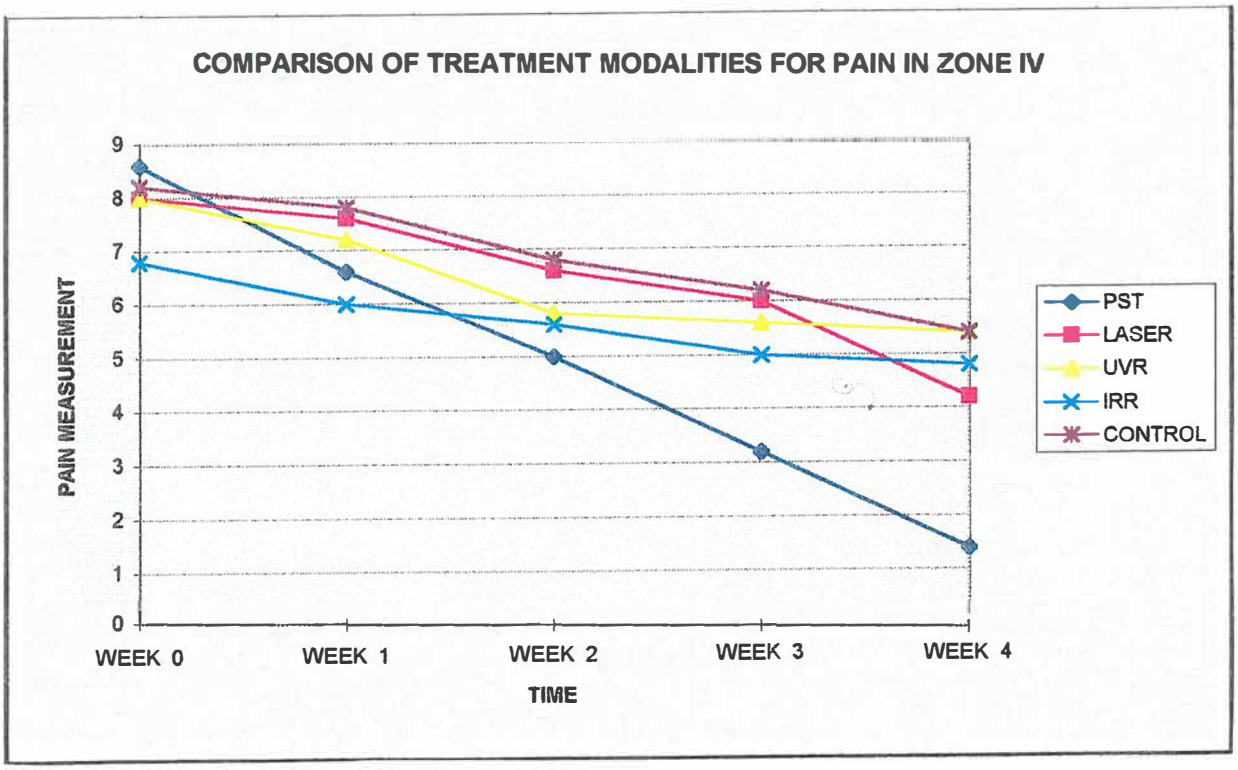


Figure 36 : Graph illustrating the mean pain measurement per week in zone IV

Graphical illustration on the average pain measurement of the different treatment groups in zone IV is shown in figure 36.

Analysis of pain measurement as recorded on NRS for zone IV is as follows :-
The dependent variable of pain measurement at week 0, week 1, week 2 and week 3 showed no significance between the treatment groups. There was a significant difference between the groups at week 4 ($p = 0.0017$).

Repeated measures MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect was significant for zone I ($p = 0.0001$). MANOVA test criteria and F approximations for the hypothesis of no TIME * TREATMENT effect was significant ($p = 0.0030$). Repeated measures ANOVA tests of hypotheses for between subjects effects was not significant ($p = 0.3022$).

There was a significant change in pain measurement in zone IV from week 0 to 1 ($p = 0.0014$), week 0 to 2 ($p = 0.0001$), week 0 to 3 ($p = 0.0001$) and week 0 to 4 ($p = 0.0001$). Significant differences were found between the groups at week 2 ($p = 0.0443$), week 3 ($p = 0.0008$) and week 4 ($p = 0.0001$). No significant difference was found between the groups at week 1.

Pairwise comparisons using least significant differences for zone IV yielded the following for the different treatment groups :
Pain measurement at week 0, week 1 and week 2 was not significantly different for the different treatment groups. The PST treatment group was significantly better than the other groups at week 3 and week 4.

Table 16 : Mean pain measurement and standard deviation for all groups in zone V

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV
PST	8.8	1.09	6.6	1.52	5.2	2.05	3.6	1.34	2.0	1.58
LASER	9.6	0.55	9.6	0.55	7.0	0.50	6.8	0.44	6.4	0.55
UVR	7.0	1.00	6.4	1.34	5.6	1.52	4.4	0.89	4.6	0.55
IRR	8.0	2.12	7.6	2.88	6.8	1.64	5.4	2.07	4.2	1.30
CONTROL	9.8	0.68	9.6	0.55	7.6	0.55	7.2	0.84	6.8	0.45

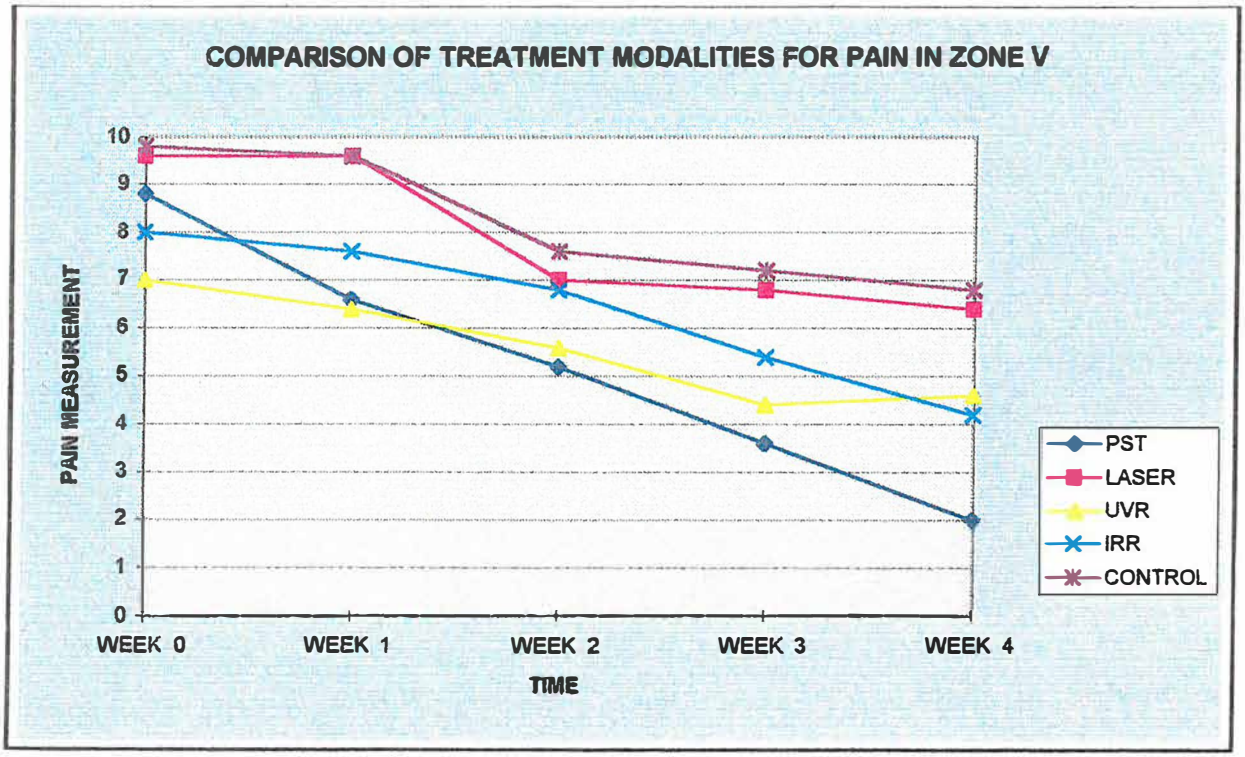


Figure 37 : Graph illustrating mean pain measurement per week for all groups in zone V

Figure 37 shows graphical representation of the pain measurement of all groups in zone V.

Analysis of pain measurement as recorded on NRS for zone V is as follows :-
The dependent variable of pain measurement showed statistical difference between the different treatment groups for all weeks, namely, week 0 ($p = 0.0120$), week 1 ($p = 0.0077$), week 2 ($p = 0.0354$), week 3 ($p = 0.0005$) and week 4 ($p = 0.0005$).

Repeated measures MANOVA test criteria and exact F statistics for the hypothesis of no TIME effect was significant for zone I ($p = 0.0001$). MANOVA test criteria and F approximations for the hypothesis of no TIME * TREATMENT effect was significant ($p = 0.0001$). Repeated measures ANOVA tests of hypotheses for between subjects effects was significant ($p = 0.0009$).

There was a significant change in pain measurement in zone V from week 0 to 1 ($p = 0.0001$), week 0 to 2 ($p = 0.0001$), week 0 to 3 ($p = 0.0001$) and week 0 to 4 ($p = 0.0001$). Significant differences were found between the groups at week 1 ($p = 0.0001$), week 2 ($p = 0.0511$), week 3 ($p = 0.0001$) and week 4 ($p = 0.0001$).

Pairwise comparisons using least significant differences for zone V yielded pain measurement in the UVR treatment group to be significantly better than the other groups at week 0 and week 1. The PST treatment group was significantly better than the other groups at week 3 and week 4.

Table 17 : Mean pain measurement for all groups in all zones

TREATMENT GROUP	WEEK 0		WEEK 1		WEEK 2		WEEK 3		WEEK 4	
	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV	PAIN	STD. DEV
PST	7.8	0.93	6.2	0.50	4.6	0.55	3.0	0.80	1.4	0.77
LASER	8.2	0.82	7.6	1.91	6.6	0.30	6.0	0.55	4.9	0.87
UVR	7.8	0.55	7.3	0.72	5.7	0.70	5.5	0.66	4.7	0.48
IRR	7.5	0.73	7.0	0.87	6.2	0.77	5.4	0.42	4.9	0.52
CONTROL	8.2	1.08	7.6	1.30	6.6	0.75	6.0	0.79	5.7	0.81

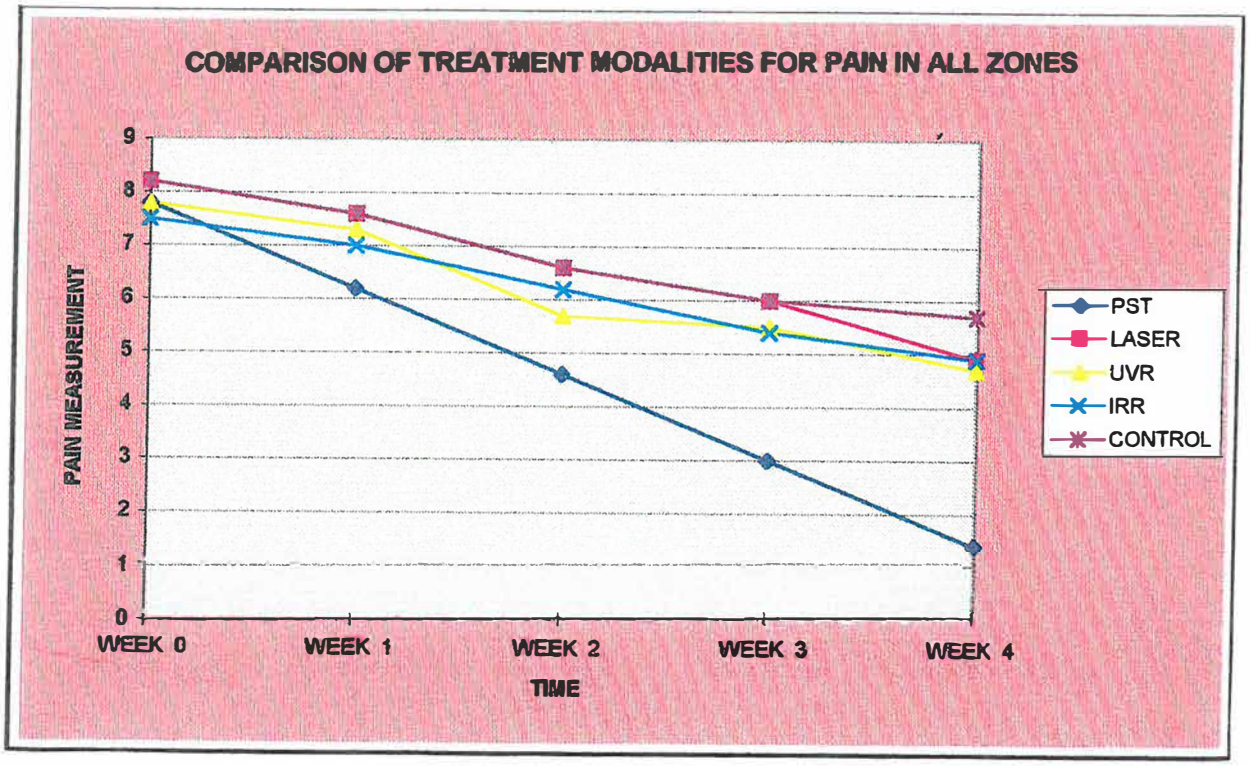


Figure 38 : Graph illustrating mean pain measurement per week for all groups in all zones

The calculation of the means by treatment with the combination of the zones is illustrated in the Figure 38.

Two-way repeated measures analysis of variance testing for differences between treatments, while controlling for zones yielded the following :-
Differences between treatments with regards to pain at week 0 was not significant ($p = 0.4705$). Pain measurement at week 1 ($p = 0.0164$), week 2 ($p = 0.0001$), week 3 ($p = 0.0001$) and week 4 ($p = 0.0001$) showed significant difference between treatments.

MANOVA test criteria for hypothesis of no TIME effect and no TIME * TREATMENT effect was significant ($p = 0.0001$).
MANOVA test criteria for hypothesis of TIME * TREATMENT effect was significant ($p = 0.0001$). MANOVA test criteria and F approximations for the hypothesis of no TIME*ZONE effect yielded no statistical significance. Repeated measures ANOVA for hypotheses of between subjects effects was significant ($p = 0.0001$) for the treatment groups, but not significant for zone.

Repeated measures ANOVA for variables time, treatment and zone yielded the following:-
There were significant differences in pain measurement between week 0 and 1, week 0 and 2, week 0 and 3, and week 0 and 4 ($p = 0.0001$). There were also significant differences between the treatment groups at each week from week 0 to 4. No significant difference was found between the zones.

T tests for least significant difference between the groups was determined. At week 0 there was no significant difference in pain measurement between the treatment groups. At week 1, week 2, week 3 and 4 the PST group differed significantly from the other groups with a lower pain measurement.

CHAPTER 6

6. DISCUSSION

The lore of wound healing has a rich tradition going back many centuries. Tradition has been a serious problem in the clinical management of wounds, as much today as it had been for Ambrose Pare almost 500 years ago when he tried and failed to introduce a degree of consideration for wounded tissue. Open wounds remain a multidisciplinary problem and a clinical challenge (Mulder and Leaper, 1998). The rapid increase in attention focused on wound repair during the past two decades has paralleled an increased demand for clinical trials examining the efficacy of wound care.

6.1 Age distribution

The age range was 20 to 59 years. All wounds included crush injuries to the hand and the age range supports the age of subjects in the workplace and therefore work-related injuries.

6.2 Male : Female ratio

120 of the total number of 125 patients were male patients, and 5 were female patients (120 : 5). This ratio was too small to draw any inferences on wound healing and pain measurement in the male and female patients included in the study.

6.3 Wound healing

Wound healing was measured by cutting out traces of the wound as described in Chapter 4. The raw data obtained may be referred to in Appendix 1.

6.3.1 Traces

The traces for all groups in each of the 5 zones were arranged in series and all wounds showed an overall decrease in wound size over time.

6.3.2 Photographs

Photographs were taken of all groups as described in Chapter 4. On visual examination, the photographs taken of wounds in the different zones in each group showed a decrease in wound size over the consecutive weeks.

6.3.3 Wound healing in the different zones

6.3.3.1 Zone I

Following treatment, open wounds in zone I revealed a marked increase in wound healing over the consecutive weeks in all the groups. The PST group showed accelerated wound healing compared to the other groups. The control group showed least wound healing, with the laser, UVR and IRR showing increased wound healing respectively in comparison to PST.

6.3.3.2 Zone II

Wound healing in zone II showed a marked improvement in the consecutive weeks following treatment. The PST and UVR groups yielded greater wound healing compared to the other groups. In order of performance following the PST group, the UVR, laser and IRR groups showed increased wound healing respectively.

6.3.3.3 Zone III

There was accelerated wound healing in this zone from week 1 to week 5 in the different treatment groups. The PST group yielded the most wound healing followed by the laser, UVR and IRR groups. The control group did not perform as well as other groups.

6.3.3.4 Zone IV

In this zone, the PST group showed greatest wound healing compared to the other groups. The laser, UVR, IRR and control groups performed respectively for wound healing over the time period of week 0 to week 4.

6.3.3.5. Zone V

The findings in zone V yielded accelerated wound healing in all groups. Only at week 1 did the UVR group perform best compared to the other groups. The PST group demonstrated the most wound healing in all the other weeks. The PST group was followed by the UVR, laser, IRR and then the control group in order of yielding increased wound healing.

6.3.3.6 All zones

The PST group yielded the best wound healing overall when compared to the other treatment groups in all the zones. As regards wound healing, the rate of healing was achieved in the following decreasing order : PST, laser, UVR, IRR, control.

6.4 Pain measurement

Pain measurement was assessed using the numerical rating scale as described in Chapter 4. The raw data obtained may be referred to in Appendix 2.

6.4.1 Pain measurement in the different zones

6.4.1.1 Zone I

PST provided more pain relief than the other groups in zone I at each of the 4 weeks. Of the other groups, pain control was achieved in the following decreasing order : IRR, laser, UVR, control.

6.4.1.2 Zone II

The PST group experienced less pain than the other treatment groups in zone II. The other groups provided pain relief respectively after PST: UVR, laser, IRR and control.

6.4.1.3 Zone III

The PST treatment group was better than the other treatment groups in zone III. The other treatment groups provided pain relief in order of performance after PST: laser, IRR, UVR, control.

6.4.1.4 Zone IV

The PST group showed better pain relief compared to the other treatment groups which performed in order of increased pain relief: IRR, UVR, laser, then the control group.

6.4.1.5 Zone V

The treatment groups PST, UVR, IRR, laser and the control groups are listed in order of performance in providing pain relief during wound healing.

6.4.1.6 All zones

Pain measurement was lower in the PST group compared to the other treatment groups. This was followed by UVR and IRR (both performed equally), then laser and the control group.

6.5 Comparison of treatment groups

The PST group yielded the best wound healing and provided the most pain relief compared to the other treatment groups. An explanation for this result lies in the principles of the production of PST. PST is postulated to restore cell membrane potential in damaged cells. There are no dangers due to heating of the tissues as there is negligible heat or no heat produced at all. The overall effect is a uniform spread of the electromagnetic field within the tissues of the whole hand. This is achieved by the position of the electrodes and their

size. The penetration depth of the electromagnetic current produced in PST is 2.2 cm. This is the greatest depth of penetration compared to other treatment groups.

Laser is theorised to play a role in the regeneration of cells in damaged tissue. The temperature of the tissues do not exceed 36.5°C. Treatment with the laser probe is directly over the site of pain, and treatment for wound healing is directly over the wound, and laser therapy spans a circumference of 1 cm^2. The maximum penetration is approximately 1 cm.

UVR is invisible radiation beyond the violet end of the electromagnetic spectrum. The major effect of UVR is the bactericidal effect. This effect results in destruction of bacteria, removes slough and promotes repair to stimulate the growth of granulation tissue. The area of irradiation is the size of the applicator and can be made smaller by blocking out the areas of healthy tissue. The depth of penetration is 2 mm (as for non-luminous generators).

The emission of IRR causes vibrations of the molecules. Heat is produced and transmitted as infrared electromagnetic radiation and local cutaneous vasodilatation occurs. IRR spans the entire area exposed, that is, the entire volar aspect of the hand. Penetration of IRR rays is 5-10 mm.

The dressings of paraffin gauze used in the study is in keeping with the maintenance of tissue hydration to promote wound healing. Previous research on this type of dressing reports that this type produced little adherence and minimal bleeding on removal, but there were reports of increased pain. Paraffin gauze protects the wound and provides hydration. The anti-bacterial mechanisms present in any wound are able to continue functioning normally by keeping the wound moist.

All modalities used in the study are generally available in physiotherapy practices and therefore easily accessible for patient care.

6.6 Limitations

The following limitations of this study were determined :-

1) Extent of debridement

Wounds included in the study were intermittently debrided during and after the saline soak. Debridement removes contaminated and devitalised tissue which improves the ability of the wound to resist infection. No debridement was performed under either local or regional anaesthesia.

2) Vascular status

Clinically none of the patients had major or significant vascular injuries. Minor vascular injuries were not excluded from the study. Clinically these injuries are difficult to detect. As intact circulation is conducive to healing, any wounds with vascular compromise is seen to retard the healing process. No sophisticated vascular assessment, for example, Doppler tests, was performed.

3) Completion of healing

All patients were monitored and treated over a period of four weeks. Not all wounds progressed to complete healing within this period.

4) HIV/AIDS status

Patients were not screened for HIV/AIDS. A positive test may have resulted in propensity to wound infection and impaired healing.

5) Pain measurement (NRS)

This scale was chosen against others because of easier explanation and understanding for the patients attending WARC. Patients were of varying educational backgrounds, often with little or no school education. The same researcher assessed the pain measurement at each evaluation/assessment to minimise the degree of subjectivity.

CHAPTER 7

7. CONCLUSION AND RECOMMENDATIONS

7.1 CONCLUSION

We have come a long way from the concept of *pus bonum et laudible* ('laudable pus') in wound management, but there is a long way to go to be able to produce foetal healing without scarring (Leaper and Harding, 1998). Despite great efforts in the study of wound repair, understanding the mechanisms of its control remains far from complete. As late as the early 20th century, poor healing and non-healing wounds were the rule rather than the exception. The addition of aseptic technique and antibiotics, careful observation and supportive care of wounds markedly improved wound management and outcome. As we prepare to enter the 21st century, clinicians and researchers have notions of scarless repair and eradicating the complications of wound healing and accelerating the wound healing process.

Wound care is receiving a much higher profile in health care. In addition to this improved emphasis, the range of clinical treatments continues to increase. Most of the modern wound dressing materials have been developed following Winter's work in the 1960's, when he identified the benefit of a moist environment for healing wounds in the porcine model. Since that time, a wide range of materials have been developed. Although no consistent improvement in healing times has been shown, particularly in chronic wounds, these carry obvious benefits to patients, carers, and healthcare professionals (Leaper and Harding, 1998).

It has to be recognised that wound care does not only require a wound contact material, but agents such as adjuvant therapy are equally important in the overall management of patients with open wounds. Interesting new work on foetal tissue suggests that the changes which occur after birth may be modified not only to assist in healing, but also to control the amount of scar present when a wound has closed.

Integration of the strategies that improve wound prevention, wound treatment and scar formation will occur over the next decade and it is only by such an approach that major improvements in patient care will occur. The other challenge that will require attention in the future is how and who will provide wound care for patients? With the increasing awareness of the importance and complexity of this subject, many individuals who care for patients or who are involved in scientific research should be involved in wound healing in some capacity or another. A multidisciplinary team approach is ideal and needs to become routine.

McCulloch (1998) states in the Journal of Wound Care that "the physiotherapist is a highly respected member of the wound-care team in the USA. While assisting in all aspects of wound care, including debridement and dressing selection and application, the physiotherapist also provides a unique function. The numerous physical agents all have benefits to offer the patient in contributing to healing. The background knowledge possessed by members of this discipline enhances the services of the wound care team. It is perceived that physiotherapists who remain uninvolved in wound care are a major untapped resource with great potential for promoting wound healing."

7.1.1 Traces

The use of wound traces proved to be a quick, easy and cost-effective method of determining the wound size and of assessing the rate of wound healing. By attaching the recording of the traces to the evaluation notes, it was always readily available for comparison of healing.

7.1.2 Photographs

The photographs proved to be an expensive, though easy and quick method of assessing the wound over a period of time. Photographs had to be processed and developed timeously in order for comparison of the same wound to be made at weekly intervals. The photographs were attached to the evaluation notes for comparison of wound healing.

7.1.3 Treatment group performance

The PST group yielded the best rate of wound healing and provided the best pain relief in comparison to the other treatment groups in all the zones.

In addition to the performance of the PST group, PST has the following advantages:-

- PST is available at most physiotherapy practices and hospital departments.
- It is cost-effective in that fewer treatment sessions are required.
- Application is easy and can be administered over bandages. The other modalities require that the wound be exposed for the administration of the treatment modality.
- The treatment is easy and quick to administer, provided the correct technique is used and precautions observed.

The study has proven that the treatment is effective in wound healing and pain relief, and it is the treatment of choice for the acceleration of wound healing and pain relief.

7.2 RECOMMENDATIONS

7.2.1 Patient education

Many patients are overwhelmed by the gravity of a complex hand injury. Recovery of function depends on the psychological as well as physical recuperation. Patient education is crucial. Patient education from the first visit must be oriented toward both current status and future return to a normal way of life. Return to work, whether to the same or to a different job, should be considered early in the course of treatment. Ideally this goal brings a co-operative effort among the patient, his family, employer and medical professionals.

7.2.2 Pain

Pain is difficult to evaluate because it is a subjective symptom. Quantification is essential, using a standard scale such as the numerical rating scale (Buck and Paice, 1994). A variety of instruments for assessing pain is available. Guiffre and associates (1988) suggest that a tool useful for the measurement of pain should meet the following criteria: be easy to understand and use; require little motivation on the part of the

subject; be easily scored; require few quantitative terms; be sensitive to fluctuations in the perception being scaled. Jensen *et al.*,(1986) concluded from a study of six pain intensity instruments that the numerical rating scale is the most practical index.

Intensity of pain may be evaluated in a thorough examination by selection of the appropriate instruments. Effective oedema management is one means of controlling the pain associated with injury.

7.2.3 Wound care

Cleansing open wounds should be a simple process, by irrigating with warm tap water or saline (Angeras *et al,* 1992). A clean dressing technique should be used which focuses on preventing infection. Necrotic and devitalised tissue can be removed by mechanical debridement, and by providing a moist environment, thereby encouraging wound healing. Careful wound management can attempt to control scar formation and can contribute to a more cosmetically acceptable hand with greater mobility and function.

7.2.4 Exercises

It is important to begin exercises before the close of the inflammatory phase of wound healing and before active participation of the fibroblasts in laying the framework for the formation of new collagen. Initiating early controlled motion aids in the alignment and orientation of the newly formed collagen and will ultimately affect the final degree of motion.

7.2.5 Alternative therapeutic approaches

The experimental evidence and implications in the "laying on of hands" is the influence of the healer's hands on a significantly more rapid rate of wound healing in mice and plants (Bugaj, 1982). Individuals within and out of the medical profession who demonstrate knowledge of healing techniques, could be utilised to guide and train other practitioners in the selection and application of various alternative therapeutic approaches and measures. Healing through this ancient art would complement and not replace, our more traditional therapeutic approaches.

7.2.6 Combination treatments

Greater investment in research to evaluate efficacy is required. Combination treatment, for example, IRR and PST should be assessed. This should be done if we are to ensure cost-effective use of resources and optimal healing for all patients. Wounds need to be stratified and selected according to aetiology, duration and mechanism of healing, as determined, for example, by the extent of granulation and re-epithelialisation.

7.2.7 Educational strategies

Healthcare professionals with recognised training in wound care should disseminate their knowledge and skills to others, including patients. Educational strategies should focus on the pathophysiology of wound healing, assessment, normal and abnormal wound healing, dressing selection, health education and criteria for referrals to other health professionals. These should improve quality and health outcomes for patients, and reduce the high cost associated with healing open wounds. Self-fulfilment and psychological upliftment will be achieved with early return to work and activities of daily living.

The escalating cost of man-hours, the psychological, social and financial burden of wounds to patients, their families and society demands redress. The comparative study to determine the effectiveness of the various wound healing modalities in physiotherapy does assist in addressing this problem. Judith A. Bell-Krotoski, President of the American Association of Hand Therapy entitled her address at the AGM held in March 1999 : "The truth is not negotiable : we *do* make a difference."

Professor Thomas Hunt, in his forward to the book, Wounds: Biology and Management, writes : "Someday, will we arrange for our patients to regenerate their feet and lungs? Then, perhaps, we will heal by a beam of intelligent healing energy."

REFERENCES

Alvarez OM. Wound healing. **Dermatology in general medicine**. 3rd Edition. New York: McGraw-Hill, 1987; 321-336.

Angeras HM, Brandberg A, Falk A, Seeman T. Comparison between sterile saline and tap water for the cleansing of acute soft tissue wounds. **Eur J Surg** 1992; **158**: 347-350.

Apley AG. System of Orthopaedics and Fractures. 5th. Edition. Sydney: Butterworth & Co., 1978; 168-188.

Backhouse KM. Functional anatomy of the hand. **Physiotherapy** 1968; **54**: 114-117.

Barclay V, Collier RJ , Jones A. Treatment of various hand injuries by pulsed electromagnetic energy. **Physiotherapy** 1983; **69**: 186-188.

Barker AF, Barlow PS, Porter J. A double-blind clinical trial of low power pulsed shortwave therapy in the treatment of soft tissue injury. **Physiotherapy** 1985; **71**: 500-504.

Barr NR. **The Hand : Principles and techniques of simple splintmaking in rehabilitation**. London: Butterworths, 1978.

Baxter GD. **Therapeutic Lasers : Theory and Practice**. London: Churchill Livingston, 1994.

Beaumont E, Anderson-Dam M. Wound care science at the crossroads. **Am J Nursing**. **Dec 1998**: 16-16D.

Beck WC. How bacteria got here to there: the basics of surgical asepsis. **Infections in Surgery** 1985; **4**: 266-267.

Bell-Krotoski J. The truth is not negotiable: we can make a difference. **J Hand Ther** 1999; **12**: 3-6.

Bentall R, Eckstein HB. 'A trial involving the use of pulsed electro-magnetism therapy on children undergoing orchidopexy'. **Kinderchirurgie** 1975; **17**: 380-389.

Bohannon RW, Pfaller BA. Documentation of wound surface area from tracings of wound parameters: Clinical report on three techniques. **Phys Ther** 1983; **63**: 1622-1624.

Brandaleone H, Papper E. The effect of the local and oral administration of cod liver oil on the rate of wound healing in vitamin A deficient and normal rats. **Ann Surg** 1941; **114**: 791-798.

Braun GS. 'Pulsed electro-magnetism energy: A new therapeutic modality in podiatry'. **Am Podiatry Assoc** 1965; **55**: 700-705.

Brennan SS. Antiseptic toxicity in wounds healing by second intention. **J Hosp Infection** 1986; **8**: 263-267.

Brennan SS, Leaper DJ. The effect of antiseptics on the healing wound: a study using the rabbit ear chamber. **Br J Surg** 1985; **72**: 780-782.

Brunner R, Haina D, Landthaler M. Applications of laser light of low power density: Experimental and clinical investigations. **Curr Probl Dermatol** 1986; **15**: 111.

Bryner P. Extent measurement in localised lower-back pain: a comparison of four methods. **Pain** 1994; **59**: 281- 285.

Buck M, Paice J. Pharmacological management of acute pain in the orthopaedic patient. **Orthopaedic Nursing** 1994; **16**: 14-23.

Bugaj R "Laying on of hands" healing: Experimental evidence and implications. Proceedings of the IXth International Congress of the World Confederation of Physical Therapy, Stockholm, Sweden, 23-28 May 1982, 287-292.

Cameron DA. Experimental acceleration of wound healing. **American Journal of Orthopaedics** 1961; **53**: 336-343.

Carley PJ, Wainapel SF. Electrotherapy for acceleration of wound healing: low intensity direct current. **Arch Phys Med Rehab** 1985; **66**: 443-446.

Chang LD, Buncke G, Slezak S, Buncke HJ. Cigarette smoking, plastic surgery and microsurgery. **J Reconstr Microsurg** 1996; **7**: 467-474.

Colwell JC, Foreman MD, Trotter JP. A comparison of the efficacy and cost-effectiveness of two methods of managing ulcers. **Decubitus** 1993; : 20-27.

Cornwall MW. Zinc iontophoresis to treat ischaemic skin ulcers. **Phys Ther** 1981; **61**: 359-360.

Dyson M, Suckling J. Stimulation of tissue repair by ultrasound: a survey of the mechanisms involved. **Physiotherapy** 1978; **64**: 105-108.

Evans P. The healing process at cellular level: a review. **Physiotherapy** 1980; **66**: 256-258.

Falabella M. Uncommon causes of ulcers. **MEDSURG Nursing** 1998; **25**: 467-479.

Feedar JA, Kloth LC, Gentzkow GD. Chronic dermal ulcer healing enhanced with monophasic pulsed electrical stimulation. **Phys Ther** 1991; **71**: 639-649.

Fenn H. Effects of pulsed electromagnetic energy on experimental haematomas. **Cancer Medical Association Journal** 1969; **100**: 251-254.

Fergusson AG, Logan JC. Leg ulcers: Assessment of response to certain topical medicaments. **Br Med J** 1961; **1**: 871-874.

Fess GE. Clinical validation of hand rehabilitation : evaluating published research. **Rehabilitation of the Hand**. St. Louis, Mo: CV Mosby, 1995; 1900.

Fischer BH. Topical hypercarbic oxygen treatment of pressure sores and skin ulcers. **Lancet** 1969; **II**: 405-409.

Forster A , Palastanga N. **Clayton's Electrotherapy: Theory and Practice**. 9 Edition. Tokyo: Bailliere Tindall, 1990.

Freytes HA, Fernandez B , Fleming WC. Ultraviolet light in the treatment of indolent ulcers. **South Med J** 1965; **58**: 223-226.

Fugill GC. Pressure sores. **Physiotherapy** 1980; **6**: 46.

Gilchrest BA *et al.* Longwave ultraviolet (UVA) induced histologic and biochemical changes in normal skin. **Clin Res** 1982; **30**: 586A.

Gilchrist SA, Hutchinson WE. The bacteriology of chronic venous ulcers treated with occlusive hydrocolloid dressings. **Br J Dermatology** 1990; **121**: 337-344.

Gilliland EL. Bacterial colonisation of leg ulcers and its effect on the success rate of skin grafting. **Ann Surg** 1988; **70**: 105-108.

Gilman TH. Parameter for measurement of wound closure. **Wounds** 1990; **2**: 95-101.

Glanville HJ. Speeding the patient's return to work. **Physiotherapy** 1965; **8**: 131-133.

Golden JH, Broadbent NRT, Nancarrow JD, Marshall T. The effects of Diapulse on the healing of wounds: a double-blind randomised controlled trial in man. **Br J Plast Surg** 1981; **14**: 267-270.

Gorse GJ, Messner RL. Improved pressure sore healing with hydrocolloid dressings. **Arch Dermatol** 1987; **123**: 766-771.

Goujon C, Divol J, Mouoin G Preliminary results of midlaser treatment of chronic ulceration of the legs (Abstract). Proceedings of the International Society for lasers in surgery and medicine, New York, 1985, 78.

Gray H, Pick TP, Holmes T, Howden R, Johnston TB, Whillis J. **Gray's Anatomy**. 36th Edition. Philadelphia: WB Saunders, 1980.

Griffin JW, Tooms RE, Mendius RA, Clifft JK. Efficacy of high voltage pulsed current for healing of pressure ulcers in patients with spinal cord injury. **Phys Ther** 1991; **71**: 433-442.

Guiffre M, Keane A, Hatfield SM, Korevaar W. Patient-controlled analgesia in clinical pain research management. **Orthopaedic nursing** 1988; **16**: 30-36.

Hardy MA. The biology of scar formation. **Phys Ther** 1989; **69**: 1014-1024.

Harkins HN. Wound Healing. **Surgery: Principles and Practice**. 3rd Edition. Philadelphia: Lippincott, 1965; 7-19.

Hatz RA, Niedner D, Vanscheidt W, Westerhof W. **Wound healing and wound management:A guide for Private Practice**. Heidelberg: Springer-Verlag, 1994, 1-16.

Hayne CR. Pulsed high frequency energy - its place in Physiotherapy. **Physiotherapy** 1984; **70**: 459-466.

High AS, High JP. Treatment of infected skin wounds using ultraviolet radiation : an in-vitro study. **Physiotherapy** 1983; **69**: 359-360.

Hinman CC, Winter GD. Effect of air exposure and occlusion on experimental human skin wounds. **Nature** 1963; **200**: 377-379.

Hodges RE, Baker EM, Hood J, Sauberlich HE, March SC. Experimental scurvy in man. **Am J Clin Nutr** 1969; **22**: 535-548.

Hoppenfeld S. Physical examination of the wrist and hand. **Physical Examination of the Spine and Extremities**. Norwalk: Appleton & Lange, 1976; 59-104.

Howes EL. The rate and nature of epithelialisation in wounds with loss of substance. **Surg Gynecol Obstet** 1943; **76**: 738-745.

Hunt TK, Dumphy JE. **Fundamentals of wound management.** New York, Appleton-Century-Crofts, 1979.

Hunter JM, Mackin JE, Callahan AD. **Rehabilitation of the hand : Surgery and Therapy**. 4th Edition. London: Mosby, 1995;

Jensen MP, Karoly P, Braver S. The measurement of clinical pain : A comparison of six methods. **Pain** 1986; **27**: 117-126.

Kahn J. Open wound management with the HeNe cold laser : case report. **J Orthop Sports Phys Ther** 1984; **3**: 203-204.

Kalinowski DP, Brogan MS, Sleeper MD. A practical technique for disinfecting electrical stimulation apparatuses used in wound treatment. **Phys Ther** 1996; **76**: 1340-1347.

Kana JS, Yoshimura Y, Nakajima T. Effects of low-power density laser radiation of healing on open skin wounds in rats. **Arch Surg** 1985; **14**: 278.

Kaplan EG, Weinstock RE. 'Clinical evaluation of Diapulse as adjunctive therapy following foot surgery'. **J Am Podiatry Assoc** 1968; **5**: 218-221.

Klaber MR. Ultraviolet light in Psoriasis. **Physiotherapy** 1980; **66**: 36-38.

Kleinkort JA, Foley RA. Laser acupuncture: Its use in Physical Therapy. **Am J Acupuncture** 1984; **12**: 51.

Kloth LC, Feedar JA. Acceleration of wound healing with high voltage, monophasic, pulsed current. **Phys Ther** 1988; **68**: 503.

Kloth LC, McCulloch JM, Feedar JA. **Wound Healing : Alternatives in Management**. Philadelphia: FA Davis Company, 1990.

Knighton DR, Fiegel VD, Doucette MM, Fylling CP, Cerra FB. The use of topically applied platelet growth factors in chronic non-healing wounds: a review. **Wounds** 1989; 71-77.

Kurzuk-Howard G, Simpson L, Palmieri A. Decubitus ulcer care: a comparative study. **West J Nurs Res** 1985; **1**: 58-79.

Lam TS, Abergel RP, Castel JC, Meker CA, Dwyer RM Biostimulation of human skin fibroblasts by low energy lasers. *In:* Proceedings of the 5th International Congress of laser Medicine and Surgery, Detroit, Michigan, 1983.

Lawrence JC. The bacteriology of burns. **J Hosp Infection** 1985; **6**: 3-7.

Lazarus GS, Cooper DM, Knighton dr. Definitions and guidelines for assessment of wounds and evaluation of healing. **Arch Dermatol** 1994; **130**: 489-493.

Leaper DJ, Harding KG. Wound care stastistics. Wounds: Biology and Management. Oxford. Oxford University Press, 1998; 98-120.

Leaper DJ, Mulder GD. Factors influencing wound healing. **Wounds: Biology and Management**. Oxford: Oxford University Press, 1998; 52-70.

Lehman JF. Therapeutic heat and cold. **Therapeutic Heat**. Baltimore: Williams and Wilkins, 1992.

Low J, Reed A. **Electrotherapy explained : Principles and Practice**. 1st Edition. London: Butterworth Heinemann, 1990; 1-374.

Lowden TG. Severe infections of the hand. **Physiotherapy** 1968; **54**: 128-131.

MacKinnon JL, Cleek PJ. The penetration of ultraviolet light through transparent dressings : a case report. **Phys Ther** 1984; **64**: 204.

Majno G. **The healing hand: Man and Wound in the Ancient World**. Cambridge: Harvard University Press, 1975.

Malone WD. Wound dressing adherence : a clinical comparative study. **Arch Emerg Med** 1987; **4**: 101-105.

Marks J. Prediction of healing time as an aid to the management of open granulating wounds. **Wld J Surg** 1983; **7**: 641-645.

McCullock JM. Intermittent compression for the treatment of chronic stasis ulceration. **Phys Ther** 1981; **61**: 1452-1453.

McGill SN. The effect of pulsed shortwave therapy on lateral ligament sprain of the ankle. **NZ J Physiother** 1989; **16**: 21-24.

Mendoza CB, Postlethwaite RW, Johnson WD. Veterans administration cooperative study of surgery for duodenal ulcer II. Incidence of wound disruption following operation. **Arch Surg** 1970; **101**: 396-398.

Meijer M, Maasen V, van den Bouwhuijsen F. Pulsed and continuous shortwave Therapy. Chap. 3. Netherlands. Enraf-Nonius. 1991.

Mester E, Mester AF, Mester A. The biomedical effects of laser application. **Lasers Surg Med** 1985; **5**: 31-39.

Mester E, Spiry T, Szende B, Tota JG. Effect of laser rays on wound healing. **Am J Surg** 1971; **122**: 532.

Mims CA, Playfair JHL, Roitt IM. **Medical Microbiology**. St Louis: Mosby, 1993.

Naussbaum EL, Blemann I, Mustard B. Comparison of ultrasound/ultraviolet-C and laser for treatment of pressure ulcers in patients with spinal cord injury. **Phys Ther** 1994; **74**: 812-823.

Nissan M, Rockind S, Ralon N. He-Ne laser irradiation delivered transcutaneously: its effect on the sciatic nerve in rats. **Lasers Surg Med** 1986; **6**: 435-438.

Oliver DE. Pulsed electro-magnetic energy - what is it? **Physiotherapy** 1984; **70**: 458-459.

Par D, Moolenaar H. Endolaser 476. Netherlands. Enraf-Nonius. 1994

Parrish AJ. Science of Photomedicine. Plenium Press,1982.

Parry A. The Musculo-skeletal System. **Physiotherapy Assessment**. London: Biddles Ltd, 1980; 53-63.

Pasila M, Visuri T, Sundholm A. 'Pulsating shortwave therapy: Value in treatment of recent ankle and foot sprains'. **Arch Phys Med and Rehab** 1978; **59**: 383-386.

Raji AM. An experimental study of the effects of pulsed electromagnetic field on nerve repair. **J Hand Surg** 1984; **9B**: 105-111.

Ramirez AT, Soroff hs, Schwartz MS. Experimental wound healing in man. **Surg Gynecol Obstet** 1969; **128**: 283-293.

Rose J. The power of the hand. **Physiotherapy** 1968; **54**: 113.

Rothstein JM. Measurement and Clinical Practice : Theory and Practice. **Measurement in Physical Therapy**. New York: Churchhill Livingston, 1985; 1-46.

Saydak SJ. A pilot test of two methods for the treatment of pressure ulcers. **J Enterostomal Ther** 1990; **3**: 139-142.

Schjelderup V, Klinikk BK. The use of laser therapy in acupuncture. **Scandinavian J Acupuncture & Electrotherapy** 1977; **2**: 70-74.

Schwartz DP, DeGood DE, Shutty MS. Direct assessment of beliefs and attitudes of chronic pain patients. **Arch Phys Med Rehab** 1985; **66**: 806-809.

Schwartz M, Doron A, Erlich M. Effects of low energy He-Ne laser irradiation as post-traumatic degeneration of adult rabbit optic nerve. **Lasers Surg Med** 1987; **7**: 497-505.

Scientific Committee of Tokyo Judo Bonesetter Association. Clinical results of Unilaser 401 and their considerations. Japan. 1984.

Scott BO. **Therapeutic Heat and Cold**. Baltimore, Waverly Press Incorporated, 1965.

Sebern MD. Pressure ulcer management in home care: efficacy and cost effectiveness of moisture vapour permeable dressing. **Arch Phys Med Rehabil** 1986; **10**: 726-729.

Shutty MS, DeGood DE , Tuttle DH. Chronic pain patients' beliefs about their pain and treatment outcomes. **Arch Phys Med Rehab** 1990; **71**: 128-132.

Steinberg MD. 'Diapuls therapy in general podiatry practice'. **J Am Podiatry Association** 1964; **54**: 849-852.

Stotts NA. **Impaired wound healing**. Philadelphia: Saunders, 1986; 343-466.

Tempest MN. The treatment of trauma of the hand. **Physiotherapy** 1980; : 118-122.

Thiel H. Low power laser therapy - an introduction and a review of some biological effects. **The American Chiropractor** 1987; : 14.

Thomas S. Wound dressing material-testing and control. **Pharm J** 1982; **228**: 576-578.

Thomas S. **Wound management and dressings**. London: The Pharmaceutical Press, 1990.

Trelles MA, Mayayo A, Mester A, Rigau J. Low power laser therapy. A review of experimental and clinical data. **Scandinavian J Acupuncture & Electrotherapy** 1987; **2**: 117-121.

Trelles M, Mayo E. Bone fracture consolidates faster with low power laser. **Lasers Surg Med** 1981; **7**: 36-45.

United States Department of Health and Human Services. Treatment of pressure ulcers: clinical practice guideline. Public Health Sercice Agency for Health Care Policy and Research; 1994; **15**; 53-81.

Wadsworth H, Chanmugan APP. **Electrophysical Agents in Physiotherapy: Therapeutic and Diagnostic Use**. . Marrickville, NSW: Science Press, 1980;

Wagstaff et al. A pilot study to compare the efficacy of continuous and pulsed magnetic energy on the relief of low back pain. **Physiotherapy** 1986; **72**: 563-566.

Whitaker OC, Warfield CA. The measurement of pain. **Hospital Practice** 1988; **2**: 155-162.

Wilkerson JD, Morrow JR, Disch JG. The reliability and validity of the handheld planimeter for determining kinetic curve parameters. **Res Q Exerc Sport** 1982; **53**: 180-183.

Williams DA, Robinson ME, Geisser ME. Pain beliefs: assessment and utility. **Pain** 1994; **59**: 71-78.

Wilson. Comparison of shortwave diathermy and pulsed electromagnetic energy in treatment of soft tissue injuries. **Physiotherapy** 1974; **60**: 309-310.

Wilson DH. Treatment of soft tissue injuries by pulsed electrical energy. **Br Med J** 1972; **2**: 269-270.

Wilson DH, Jagadeesh P. 'Experimental regeneration in peripheral nerves and the spinal cord in laboratory animals exposed to a pulsed electromagnetic field'. **Paraplegia** 1975; **14**: 12-20.

Winter GD. Formation of the scab and the rate of epithelialisation of superficial wounds in the skin of the young domestic pig. **Nature** 1962; **193**: 293.

Wynn Parry C B. **Rehabilitation of the Hand**. 4th edition. Boston: Butterworths, 1981;

Young S, Bolton P, Dyson M, al et. Macrophage responsiveness to light therapy. **Lasers in Surgery and Medicine** 1989; **9**: 497-505.

Zarro V. Thermal agents in rehabilitation. **Mechanisms of inflammation and repair**. Philadelphia: FA Davis, 1986; 12.

APPENDIX I

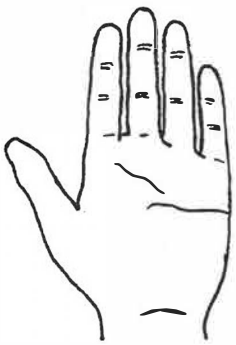
WORKMEN'S ACCIDENT AND REHABILITATION CENTRE
HAND ASSESSMENT CHART

NAME : _____ **ACC. NO** : _____ **CARD NO** :
AGE: _____ **M / F** _____ **Dominant Hand** **R / L**
ADDRESS : **(W)** _____ **Injured Hand** **R / L**
(H) _____
OCCUPATION: _____
DIAGNOSIS: _____
DATE OF INJURY : _____
DATE OF EXAMINATION : _____

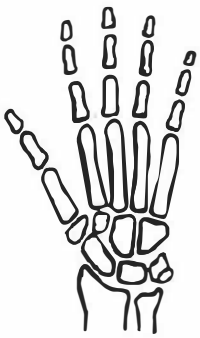
HISTORY : (tick)

Med. Hx.	Where	Cause	Wound Features
Diabetic	home	knife	cut
smoker	work	glass	crush
alcohol	sport	machinery	missile- high velocity
obese	other	explosive	low velocity
nourishment		amputation	other
antibiotics		other	

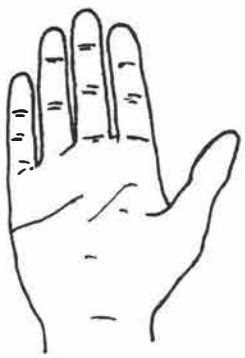
MANAGEMENT : _____
TESTS : (tick) Hb
wound swab
ASSESSMENT : Indicate skin loss, amputations, sensory loss, bone injuries, retained foreign body, lacerations



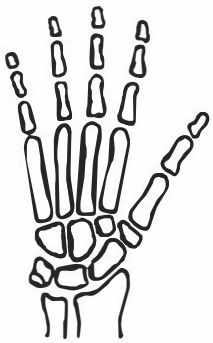
(R) PALM



X-RAY



(L) PALM



X-RAY

WOUND DIMENSIONS : (weekly)

	DATE	DATE	DATE	DATE	DATE	DATE
1. Tegaderm trace (tick)						
2. Measurement (Mettler balance)						
3. Photograph (tick)						
4. Visual inspection						
5. Pain (NRS)						

RANGE OF MOTION :

		DATE		DATE		DATE		DATE		DATE		DATE		DATE	
Passive/Active		P	A	P	A	P	A	P	A	P	A	P	A	P	A
Thumb	MCPJ														
	IPJ														
Index	MCPJ														
	PIPJ														
	DIPJ														
Middle	MCPJ														
	PIPJ														
	DIPJ														
Ring	MCPJ														
	PIPJ														
	DIPJ														
Little	MCPJ														
	PIPJ														
	DIPJ														

MUSCLE STRENGTH :

		DATE	DATE	DATE	DATE	DATE	DATE	DATE	DATE
Thumb	Fpl								
	Fpb								
	AbP								
	AdP								
	EPL								
	EPB								
	Opp								
Pinch	IF								
	LF								
IF	FDP								
	FDS								
MF	FDP								
	FDS								
RF	FDP								
	FDS								
LF	FDP								
	FDS								
Grip strength									

TREATMENT GROUP :

1. **PULSED SHORTWAVE DIATHERMY** (+saline + dressings)
2. **LASER** (+ saline + dressings)
3. **U V R** (+ saline + dressings)
4. **IRR** (+ saline + dressings)
5. **CONTROL** - saline + dressings only.

ASSESSMENT AND TREATMENT NOTES :

DATE: _____

DATE : _____

EVALUATION:

PATIENT COMFORT : PAIN (NRS) 1 2 3 4 5 6 7 8 9 10

TENDERNESS light / deep palpation

QUALITY OF HEALING

- VISUAL (comment) :
- COLOUR MATCH : (pigment)
- THICKNESS OF SCAR :
- TENDERNESS :

APPENDIX 2

MEAN % WOUND HEALING		PST	LASER	UVR	IRR	CONTROL
ZONE I	WEEK 1	100	100	100	100	100
	WEEK 2	66.8	68.1	71.3	69.9	82.7
	WEEK 3	40.2	49.3	41.0	50.4	61.8
	WEEK 4	15.8	27.1	30.4	28.0	40.9
	WEEK 5	6.8	11.5	20.6	14.3	24.9
ZONE II	WEEK 1	100	100	100	100	100
	WEEK 2	50.7	63.1	70.8	79.2	74.0
	WEEK 3	31.9	48.2	37.1	62.0	47.8
	WEEK 4	11.7	30.9	17.9	46.1	32.2
	WEEK 5	3.1	12.2	11.3	30.9	20.1
ZONE III	WEEK 1	100	100	100	100	100
	WEEK 2	63.6	73.3	68.8	73.0	74.7
	WEEK 3	25.3	49.6	56.4	45.6	58.7
	WEEK 4	10.3	20.7	34.5	30.5	37.3
	WEEK 5	2.9	6.7	18.3	16.7	17.9
ZONE IV	WEEK 1	100	100	100	100	100
	WEEK 2	59.2	62.4	74.9	76.7	74.7
	WEEK 3	36.0	49.5	55.7	54.1	54.6
	WEEK 4	18.7	24.9	34.4	37.8	34.9
	WEEK 5	5.4	12.9	23.4	18.3	18.7
ZONE V	WEEK 1	100	100	100	100	100
	WEEK 2	71.1	68.0	64.7	81.7	80.7
	WEEK 3	39.9	35.6	44.4	53.5	60.3
	WEEK 4	21.8	18.3	27.1	40.5	43.6
	WEEK 5	9.4	8.5	16.5	28.8	27.8

APPENDIX 3

MEAN PAIN MEASUREMENT		PST	LASER	UVR	IRR	CONTROL
ZONE I	WEEK 0	6.6	7.6	8.4	6.6	8.2
	WEEK 1	5.4	6.6	8.0	6.2	7.2
	WEEK 2	3.8	6.4	6.2	5.2	6.2
	WEEK 3	1.6	5.6	5.6	5.0	5.8
	WEEK 4	0.8	4.8	4.4	4.6	5.2
ZONE II	WEEK 0	7.2	7.6	7.4	8.2	7.8
	WEEK 1	6.2	6.8	6.8	8.0	7.6
	WEEK 2	4.4	6.6	4.6	7.0	6.8
	WEEK 3	3.2	6.2	5.6	6.0	6.0
	WEEK 4	2.4	4.8	4.2	5.4	6.2
ZONE III	WEEK 0	7.6	8.2	8.0	7.8	6.8
	WEEK 1	6.0	7.4	8.0	7.0	6.0
	WEEK 2	4.6	6.2	6.4	6.2	5.6
	WEEK 3	3.4	5.4	6.2	5.6	5.0
	WEEK 4	0.6	4.4	5.0	5.4	4.8
ZONE IV	WEEK 0	8.6	8.0	8.0	6.8	8.2
	WEEK 1	6.6	7.6	7.2	6.0	7.8
	WEEK 2	5.0	6.6	5.8	5.6	6.8
	WEEK 3	3.2	6.0	5.6	5.0	6.2
	WEEK 4	1.4	4.2	5.4	4.8	5.4
ZONE V	WEEK 0	8.8	9.6	7.0	8.0	9.8
	WEEK 1	6.6	9.6	6.4	7.6	9.6
	WEEK 2	5.2	7.0	5.6	6.8	7.6
	WEEK 3	3.6	6.8	4.4	5.4	7.2
	WEEK 4	2.0	6.4	4.6	4.2	6.8