

DEVICE ASSISTED WOUND BED PREPARATION

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations

For the award of degree of

**M.Ch BRANCH - III
PLASTIC SURGERY**



**INSTITUTE OF RESEARCH AND REHABILITATION OF HAND AND
DEPARTMENT OF PLASTIC SURGERY, STANLEY MEDICAL COLLEGE
AND HOSPITAL CHENNAI – 600001, TAMIL NADU.**

AUGUST - 2009

CERTIFICATE

Dissertation on
Device assisted wound bed preparation

Certified that this dissertation is a bonafide work of Dr. S. ASEER post graduate in MCh Plastic Surgery during 2006-2009 at the department of plastic surgery, Stanley medical college. This study was done under my supervision and guidance.

Prof .T.C.CHANDRAN
Professor and Head
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Stanley Medical College, Chennai - 1
Ethical Committee

CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To

Dr.S.Aseer, PG in M.ch (Plastic Surgery)

Dear Dr.S.Aseer, M.ch (Plastic Surgery)

The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

“A Study on Device Assisted wound bed preparation”

The following members of the ethics committee were present at the meeting held on 24.02.2009 at the Modernised Seminar Hall, Stanley Medical College, Chennai-1 at 12.00 Noon

Dr.C.B.Tharani, Director of Pharmacology,

Madras Medical College, Chennai-3 Chairman of the Ethics Committee

Dr.A.Sundaram, Vice-Principal,

Stanley Medical College, Chennai - 1 Member Secretary of the Ethics Committee

Members

Dr. Jayanthi

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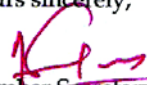
Thiru. A. Senthil Manoharan

Advocate

We approve the project to be conducted in its presented form.

The Institutional Ethics Committee/Independent Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

Yours sincerely,


Member Secretary,
Ethical Committee



ACKNOWLEDGEMENTS

I would like to thank Professor **T.C.CHANDRAN**, M.S, MCh, Prof and Head of the Department, Institute of Research & rehabilitation of hand & Department of Plastic surgery for granting me the opportunity to conduct the study & also guiding me for the study

I would like to thank **Dr. J. MOHANASUNDARAM, M.D.**, the Dean, Stanley Medical College, Chennai -1.

I also thank Prof. **R. Krishnamoorthy**, Reader, IRRH & DPS for his advice.

I also thank assistant Professors of our institute Dr.J.Mohan, Dr.J.Jaganmohan, Dr.N.C.Hariharan, Dr.G.Karthikeyan, Dr.P.Nellaiappar Dr.G.S.Radhakrishnan, Dr.M.Sugumar, Dr.M.Rajkumar, for their guidance & Help.

I thank all my co-residents for their support and help.

I thank the physiotherapist Mrs. K. Geetha for here help.

I thank all my patients, who allowed me, for the study and also for their unlimited cooperation during the study.

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AIM OF THE STUDY

Electrical Stimulation

1. To study the changes which occur in the wound bed following electrical stimulation and comparing with control.
2. To study the effects of electrical stimulation in controlling the bacterial bio burden of chronic wounds.
3. To compare the healing rates of chronic wounds treated with and without electrical stimulation.

NEGATIVE PRESSURE WOUND THERAPY

1. To analyse the efficacy of negative pressure wound therapy in preparing the wound bed of post traumatic wounds.
2. To compare the efficiency of negative pressure wound therapy in wound bed preparation of post traumatic wounds with control, using biochemical parameters.

INTRODUCTION

Preparation of wound bed is an important foundation for successful wound treatment. Understanding complex and dynamic interaction of multiple factors that control the healing process is vital for successful outcome. Science of wound bed preparation comes from technological advances and their application in clinical practice.

This **concept of wound bed preparation** was introduced in United States in late 1990, when certain advanced therapeutic products like growth factor got regulatory approval by FDA. However it was formulated in November-2000 by **European tissue repair society meeting**, held in oxford.

The therapeutic products used by them were.

1. Slow and sustained release iodine hexamer
2. Negative pressure therapy
3. Enzymatic preparation like collagenase

This concept has provided a structured and systemic approach in wound management with better utilization of technological advances of recent past. **The concept of wound bed preparation** was developed to describe the process by which chronic wounds are made suitable for surgical intervention and get benefited from available wound care technology.

Acute wounds also require wound bed preparation to prime the wound surface for healing.

REVIEW OF LITERATURE

OPTIMIZING THE PATIENT FOR WOUND BED PREPARATION

The first step in wound bed preparation is to address the systemic factors and to optimize the patient.

The factors detrimental to wound healing are

1. Aging
2. Smoking
3. Nutrition
4. Obesity
5. Diabetes
6. Medication

AGING

Aged patients have inherent wound healing difficulties that differ from those in younger patients. They heal at a slower rate, and aging causes a decrease in reepithelialization, collagen synthesis and angiogenesis. As the patient ages the response to various stress factors differ with reduced tolerance. The stress may be extrinsic (eg. environmental) or intrinsic (eg, diabetes). The decreased proliferative potential of cells and increased

senescence when compared to younger patient have been implicated to free radical theory of age related changes that occur in cellular level.

SMOKING

Smoking and consuming other tobacco products cause micro vascular constriction, and activation of sympathetic nervous system due to the action of nicotine. Cigarette smoke also contain carbon monoxide which contributes to tissue hypoxias by binding hemoglobin which has higher affinity for oxygen and decrease the delivery of oxygen to peripheral tissue. Patient with history of heavy smoking are at increased risk of fat necrosis, wound infection, and respiration complication. Patient is advised to stop smoking at least 4 week before undergoing surgery. Cessation of smoking is mandatory in treatment of wound healing.

NUTRITION

Although no definitive recommendation, optimizing nutrition is at the fore front of preparing a patient for surgical correction of wound and to ensure proper immune function.

It is important that he/she has a well balanced diet with sufficient protein intake daily vitamin and mineral supplementation. Malnutrition

predisposes a patient to further progression of the wound and detrimental to the treatment process.

Patient who are at particular risk include.

1. Elderly
2. Gastro intestinal disorder.
3. Renal failure
4. Alcoholism
5. Cancer
6. Chronic disease

Average adult nutritional requirement is 25-35 Kcal / kg /day.

In normal metabolic state average person requires 0.17gm of nitrogen or 1gm of protein/kg/day.

In patients with hypoproteinemia angiogenesis and fibroblast proliferation are decreased significantly.

Arginine and glutamine are important Amino acids in wound healing.

Decreased serum protein lead to decreased in amount of tissue oxygen delivered to the tissues. Vitamins are instrumental in wound healing with vitamin A & C carrying the burden of work.

Vitamin A

Vitamin A is important in wound phagocytosis and cell mediated immunity. Maintenance dose supplementation is recommended during

wound healing. **15,000-25,000 IU** of vitamin A supplementation daily, is limited for 4 weeks to avoid toxicity.

Vitamin C

Primary role of vitamin C is that it acts as a co factor in the formation of collagen. It also has antioxidant property which is necessary for tissue growth and repair. Recommended dietary supplementation is **60mg/day**.

Vitamin B

Antistress group of water soluble vitamins are best taken as B-complex. It includes (thiamine B1) riboflavin (B2) niacin (B3) pyridoxine (B6) Biotin, Panthothenic acid, folic acid, cobalamine (B12), choline and inositol.

Zinc

It is important in collagen formation and protein synthesis. Daily requirement is 15-21 mg/day.

Copper

Copper is required for cross linking of collagen and elastin. It is required for the formation of hemoglobin in red blood cells & in bone formation.

OBESITY

A patient is considered overweight if the body mass index is between 25 and 30kg /m². Obesity is defined as BMI greater than 30 kg/m². Obese patient are at greater risk of infection, hematoma, wound dehiscence, flap necrosis. Even with the risk associated with obesity and surgery, it is not advisable to recommend these patients to loose weight in a short period of time before undergoing surgery. However it's prudent to insist the patients about the importance of healthy diet and regular exercise.

DIABETES MELLITUS

Poorly controlled diabetes mellitus impedes wound healing by interfering with fibroblast function, impaired revascularization, and inflammatory cell activity in all the stages of the wound healing process.

Physiological intake of effective insulin combined with stress of surgery and anesthesia causes release of catecholamine, cortisol and glucagon resulting in development of stress hyperglycemia.

Tight glycemic control in patient who have diabetes is a priority. Use of off loading foot wear in neuropathic wounds facilitates healing processes.

MEDICATIONS

Medications such as NSAID, steroids, chemotherapeutic agent are particularly detrimental in wound healing.

NSAID

Asprin, ibuprofen have been implicated in decreased wound healing by decreasing the collagen production.

Asprin has anticoagulant effect, so stopped 7-10 days before surgical procedure.

Steroids

Steroids increases the risk of infection, suppresses the fibroblast proliferation, impairs collagen synthesis and decreases the formation of granulation tissue and extra cellular matrix. If consumed for longer duration, the decreased wound healing effect persist for up to 1 year after administration. Vitamin A can be used to antagonize the effects of corticosteroids.

Chemotherapeutic agents

They interfere with inflammatory response of wound healing. They also decrease fibroblast proliferation, wound contractions and interfere with immunological responses.

So Chemotherapeutic agents are avoided during wound healing. They can be used 10-14 days after the wound heals.

It is imperative that plastic surgeons have an extensive knowledge of the modifiable risk factor affecting wound healing processes and their subsequent complications.

OPTIMIZE THE WOUND

Wound bed preparation offers opportunities for the management of wounds. These range from addressing basic aspects such as management of infection, necrotic tissue and exudates to more complex management

There are four components to wound bed preparation, which address the different pathophysiological abnormalities underlying chronic wounds. Based on the work of the International wound Bed Preparation Advisory Board, an acronym has been formed.

Using the names of the components in the English language; the framework has been named TIME. The TIME framework aims to optimize the wound bed by reducing edema and exudates, reducing the bacterial burden and importantly correcting the abnormalities contributing to impaired healing. This should facilitate the normal endogenous process of wound

healing, provided the underlying intrinsic and extrinsic factors affecting the wound's failure to heal have also been addressed

Evolution of the TIME framework

TIME acronym Term proposed by International wound Bed Preparation Advisory Board,

T = Tissue, non-viable or deficient Tissue management

I = Infection or inflammation control

M = Moisture imbalance or Moisture balance

E = Edge non-advancing or undermined Epithelial (edge) advancement

TISSUE (T)

The presence of necrotic or compromised tissue is common in chronic non-healing wounds, and its removal has many beneficial effects. Non vascularized tissue, provides a good culture medium for bacteria. The necrotic tissues impede the healing process. The senescence cells of the wounds are unresponsive to certain signals in wound healing process.

Unlike chronic wounds, which usually only require debridement once if at all, acute wounds require repeated debridement.

INFLAMMATION AND INFECTION CONTROL (I)

Chronic wounds are often heavily colonized with bacterial organisms. This is due in part to the fact that these wounds remain open for prolonged

periods, but is also related to other factors such as poor blood flow, hypoxia and the underlying disease process. There is little question that clinical infection resulting in failure to heal must be treated aggressively and promptly. Evidence shows that a bacterial burden of 10^6 organisms or more per gram of tissue seriously impairs healing, although the reason for this is poorly understood.

Recently, there has been increasing interest in the possible presence of biofilms in chronic wounds and their role in impaired healing or recurrence. **Biofilms** are bacterial colonies surrounded by a protective coat of polysaccharides; such colonies become more easily resistant to the action of antimicrobials.

However, intensive investigation is needed to determine the role of biofilms in delayed healing of chronic wounds.

MOISTURE BALANCE

Most evidence for moist wound healing was developed in experiments on acute wounds, but the findings were quickly extrapolated to chronic wounds.

One reason for this uncertainty is that this fluid appears to have different properties in acute and chronic wounds. For example, fluid collected from acute wounds will stimulate the in vitro proliferation of fibroblasts, keratinocytes, and endothelial cells. Conversely, fluid from

chronic wounds will block cellular proliferation and angiogenesis and contains excessive amounts of matrix metalloproteinases (MMPs) capable of breaking down critical extracellular matrix proteins, including fibronectin and vitronectin. There is no doubt that some MMPs play a key role in wound healing – for example, interstitial collagenase (MMP-1) is important for keratinocyte production. Maldistribution of other enzymes (MMP-2, MMP-9) impair healing

EPITHELIAL (EDGE ADVANCEMENT)

The healing process involves well-defined phases. However, chronic wounds do not seem to have defined timeframes for healing and fail to progress sequentially through the phases.

For example, it has been stated that diabetic ulcers are ‘stuck’ in the proliferative phase.

Indeed, there is evidence of impaired metabolism of certain matrix proteins including fibronectin, which affects tissue protein accumulation and remodelling in diabetic foot ulcers.

There is increasing evidence that the resident cells of chronic wounds have undergone phenotypic changes that impair their capacity to proliferate and move. The extent to which this is due to senescence is not known, but the response of diabetic ulcer fibroblasts growth factors seems to be impaired.

TISSUE MANAGEMENT (T)

First hurdle in applying the concept of WBP is the confusion that proper wound bed preparation can be achieved solely by debridement. Debridement is definitely an important part of wound bed preparation, however debridement alone is not enough to sustain healing in chronic ulcer.

Debridement in a chronic wound is to think of debridement, as a way to introduce an acute wound in a chronic wound.

Chronic wounds tend to accumulate necrotic burden of senescent cells, corrupt ECM and inflammatory enzymes. They need to be removed continuously without removing healthier tissues. In acute wounds the necrotic eschar or gangrenous tissue removal are critical in controlling life and limb threatening infection.

Surgical debridement

It is the fastest way to debride the wound but it's not selective because it removes viable tissue also.

Mechanical debridement

Mechanical debridement is fast but non-selective and painful it's generally used when there is large amount necrotic tissue.

- They are
1. Wet to dry dressing
 2. Hydrosurgey
 3. Irrigation

Hydro surgery

Hydro surgery is the first technically advanced surgical instrumentation that redefines surgical technique. The tangential hydro-dissection system with versajet has been available since 2003 and is a departure from commonly practiced surgical technique. The Versajet generates a powerful coherent stream of saline (up to 15,000 pounds per square inch) across a platform that moves parallel to the wound surface. The energy of the saline beam ablates the impacted tissues, which get carried away in the effluent by way of the venturi effect. This process creates the ability to tangentially excise almost any wound, with minimal collateral damage to the surrounding healthy tissue. The versajet is as effective in removing bacteria as are the high-powered pulse lavage systems, with less risk for damaging underlying tissue. The versajet has led to a paradigm shift in surgical debridement. Wound can now be debrided centrifugally by starting within the necrotic zone and working toward the periphery where the normal tissue resides. Debridement ends when the healthy tissue is encountered.

Autolytic debridement

Debridement occurs by using the body's own endogenous proteolytic enzymes and phagocyte cell in clearing up necrotic debris. This process is facilitated by the use of moisture retentive dressing.

Chemical debridement

The proteolytic enzymes which digest the necrotic tissues were used to debride the wounds.

Currently two enzymes are used

- 1) Papain urea
- 2) Collagenase

Biological debridement

It involves the use of larva of the green bottle fly (*Lucila serricata*) this is used in patients with larger ulcer with significant necrotic material that are not amenable to surgical debridement. Larvae therapy is effective against methicillin resistant staphylococcus aureus and *B.haemolytic streptococci*.

Maintenance debridement

Maintenance debridement is the debridement done between surgical debridement. Interventions may be made by autolytic, chemical and biological.

BACTERIAL BIO BURDEN AND BIO FILM

The role of bacteria in the healing (or) non healing wounds has been a controversial subject. Although sterility of the wound bed cannot be achieved, control of bacterial burden in terms of bacterial density and pathogenicity is a goal in wound bed preparation.

The important point in discussing the bacterial bio burden in chronic wound is the impact of host response.

Clinical infection

The presence of multiplying bacteria in the body tissue that results in spreading cellular injury as a result of toxin, competitive metabolism and inflammation.

Signs of infection in acute wounds are

Swelling

Erythema

Pain

Signs of infection in chronic wound

Increasing ulcer size

Increased exudates

Dark red and friable granulation tissue

Colonization

Defined as the presence of replicating bacteria and adherent microorganisms without tissue damage.

Critical colonization

It's a novel concept that states that the bacterial burden in chronic wound does not elicit typical signs of infection but delays healing.

That is because the bacteria in the wound do not incite an intense inflammatory response.

Bio film

Bio film are complex communities of bacteria that have evolved ways to communicate with each other through water channels. And have protective extra cellular polysaccharide matrix covering.

Bio film have high resistance to antibiotic.

A critical Bacteria load, the synergistic relationship between microorganisms, presence of specific pathogens are the adverse factors which affect wound healing.

Based on various studies, the presence of 10^6 or more colony forming units per gram of tissue presets delayed wound healing.

It has been also suggested that presence of 4 or more type of organism is a predictor of impaired healing, possibly due to synergistic action of microorganisms.

The presence of B.haemolytic streptococci in the wound delays wound healing. The data on staphylococcus aureus, pseudomonas aeruginosa as major contributor to delayed healing is not been consistent.

Indication of tropical antimicrobial is limited. Systemic antimicrobial (or) topical antiseptics should only be prescribed in patients who are clinically infected or exhibit critical colonization.

The routine use of antibiotics to facilitate wound healing is not supported by evidence. In case of critical colonization topical antiseptics may offer first line treatment.

If the wound fails to improve after initial course of 2 to 3 weeks, systemic antibiotic should be considered.

New topical antiseptics available are:

1. Cadexomer iodine
2. Silver

Both are broad spectrum antimicrobial and effective against bio film. Iodine is useful bacteriostatic and bactericidal agent that is effective against methicillin resistant staphylococcus aureus. 0.9% elemental iodine released on exposure to exudates in wound. Silver has proven effective as an antiseptic with minimal toxicity. Silver exerts its lethal effect even in low concentration, particularly sensitive organisms are susceptible to 6 parts per million.

Topical antimicrobials

1. Mupirocin
2. Silver sulfadiazine
3. Neomycin, bacitracin, polymyxin (Neosporin)

Mupirocin

Mupirocin is a topical antibiotic with a unique mechanism of action. This agent binds to bacterial RNA synthetase and inhibits protein production. It is effective when used in wounds that are colonized or infected with gram positive bacteria, including MRSA.

Silver sulfadiazine

Silver sulfadiazine is a combination of silver and sulfonamide anions in an aqueous cream vehicle. This product is active against gram – positive bacteria, gram-negative bacteria, and yeast. It is used commonly in the management of burn wounds because of its broad spectrum antimicrobial action.

MOISTURE BALANCE DRESSING

The moist wound dressing is beneficial to some non healing wounds although the choice of dressing depends on the characteristics of the wound bed of a patient at the given time.

Further more a systematic review reported that dressing alone will not be able to heal chronic wound.

Nevertheless dressings are important because their use helps patients in their wound management and may prepare the wound bed for other therapies.

Dressing that maintain a moist wound environment are described as being moisture retentive. This property is measured by the moisture vapour transmission rate.

A dressing is moisture retentive when its MVTR is less than $840\text{g/m}^2/24\text{hrs}$.

Films (polyurethane film)	300-800 mvtr	No absorptive capacity.
Hydrocolloid (carboxymethyl cellulose, pectin, gelatin, guar gum)	< 300 mvtr	Not infected wound(or) heavily Exudative wound.
Hydrogel (water polymer humectants)	-	For minimally exuding wounds May be used to moisten gauze Padding.
Absorptive wound filler (calcium alginate)	-	Used under compression bandage Effective in undermined (or) on tunneling wounds. High absorptive capacity.
Foams (polyurethane foam)	-	For highly exuding wounds Not for dry wound.
Gauze	-	For heavily exuding wounds Useful as a secondary dressing to contact layer.

Matrix metalloproteinase

Matrix metalloproteinase's and their inhibitors control degradation and formation of the extra cellular matrix which are an essential step in wound healing. Extra cellular matrix contain growth factors and cytokines MMP'S are zinc dependent group of enzymes. In chronic wounds there is usually higher level of MMP'S 1 and 8 and low level of Timp-1. MMP'S 1,2,8,9 and 14 are elevated in diabetes mellitus. MMP'S - 9 is elevated in venous ulcer.

Attempts to decrease the MMP'S in wound fluid by specialized dressing which specifically binds MPP'S, had no significant difference in wound closure when compared to control. Thus non specific inhibition of MMP'S are not beneficial.

Growth factors

Multiple in-vitro animal studies has show the beneficial effects of growth factors such as PDGF, FGF, GM-CSF, PGDF is approved for use in diabetic foot ulcer. Clinical trails of exogenous application of these growth factors failed to produce expected significant results.

Cellular senescence or near senescence with continued exposure of cells in wound bed to inflammatory cytokines, reactive oxygen species, bacterial toxins results in accumulation of senescent cells. When this cell population reach a critical number, wounds are unlikely to heal even with

optimal care. Current treatment strategies to circumvent this problem are to remove senescent cells and repopulate the wound bed with viable non-senescent fibroblast using tissue engineered skin.

SUMMARY

Prerequisite for effective therapy are that the wound bed has little necrotic burden, manageable bacterial load, minimal inflammation, resident cell that can regenerate needed tissue.

Thus Wound Bed Preparations is the first step in achieving healing of wounds.

ELECTRICAL STIMULATION

Over 25 yrs many controlled research studies have produced evidence that low density electric current leads to augmented and accelerated wound healing.

Increase rate of new bone formation was demonstrated when small direct current was applied in fracture site.

How ever the healing rate of soft tissue wounds particularly superficial open wounds have benefited by addition of electrical stimulation to the treatment regime.

SKIN BATTERY

An trancutaneous potential difference exist in normal human skin known as the skin battery.

The stratum corneum is negatively charged with respect to deeper dermis with an average potential difference of 23 volts.

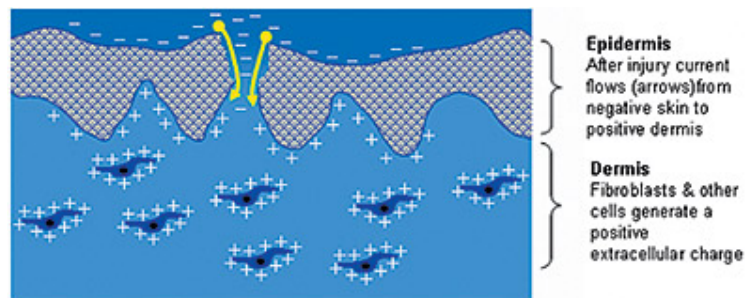
It's believed that these potentials are generated in actively metabolizing basal region of epidermis (stratum basal).

A term called “**current of injury**” is generated between the skin and deeper tissue, when there is a break in the skin.

This current is expected to continue until the skin defect is repaired. Healing of injured tissue is arrested (or) incomplete if the current no longer flow adequately.

The rationale for applying the electrical stimulation is that it mimics the natural current of injury and it will jump start (or) accelerate the wound healing process.

Figure 1: The current of injury.



Types of current:

Several types of current used for wound healing

Direct current

Pulsed current

Alternate current

Direct Current:-

Low intensity direct clinically 20-100 u amp at voltage <8volt are used.

The cathode is kept over the wound

The anode is placed in peri wound region

The reversed polarity is used in certain regions.

Pulsed Current:-

The pulsed current generally used are two sub types.

Pulsed Direct current

High voltage pulsed current

PDC generally used to for ES is 30-40 mn (generated by 6-12 volts battery) at 28pulse / second.

HPVC

In HPVC 100 - 250 V at 80 - 100 pulse per sec (HZ) was used.

Alternate current:-

The commonly used two sub types are

- a) TENS devices
- b) Biphasic current

Tens

Generated square wave pulse at 80-90 HZ with 0.1 – 0.2 ms pulse width.

Biphasic Current

In biphasic current 50 to 100 volts, frequency of 100 hertz with pulse width of 150 to 200 nm are used.

Pre Requisite for Electrical Stimulation

The wounds should be shallow.

Moist environment of the wound maintains the optimal bio electric changes.

The wounds need to have adequate vascularity.

The wound should not have active osteomyelitic lesion.

Contra indication for electrical stimulation

Placement of electrodes tangential to cardiac pacemaker

Placement of electrodes along the region of phrenic nerve

Presence of malignancy

Placement of electrodes over bone especially with osteomyelitis

Placement of electrode over topical ointments/creams containing metal ions.

Effects of Electrical stimulus

Inflammatory phase

Initiate the wound repair by its effect of an current of injury.

Increased blood flow

Promote phagocytosis

Enhances tissue oxygenation

Reduces edema perhaps from reduced micro vascular leakage.

Attracts and stimulates fibroblast and epithelial cell

Stimulates DNA synthesis

Controls infection (proven bacteriocidal effect)

Solubilizes blood products including necrotic tissue.

Proliferation Phase

Stimulates fibroblast & epithelial cells.

Stimulate DNA and protein synthesis

Increases ATP generation.

Improves membrane transport

Produces better collagen matrix organization

Stimulate wound contraction

Epithelization Phase

Stimulate epidermal cell reproduction and migration

Produces smoother and thinner scar

INDICATION OF ELECTRICAL STIMULATION

Decubitus ulcer (stage II-IV)

Diabetic ulcer

Venous ulcer

Traumatic ulcer (non healing)

NEGATIVE PRESSURE WOUND THERAPY

Negative pressure wound therapy (NPWT) was developed in the 1990s by researchers at Wake Forest University School of Medicine, Winston-Salem, NC. The concept was based on the mechanics of physics. The application of controlled sub atmospheric pressure causes mechanical stress to tissues. Mitosis is stimulated, new vessels are formed, and the wound is drawn closed. The degree of pressure to the wounded tissue is small, but when all areas of the wound work together in an effort to close toward the center point, the effect of negative pressure becomes impressive and results in quicker healing and resolution.

THE SCIENCE BEHIND NEGATIVE PRESSURE WOUND THERAPY

Moist wound healing

NPWT applies sub atmospheric pressure, or suction, to the wound bed via an vacuum therapy unit attached to an open-cell foam sponge that is placed in the wound and secured with an adhesive drape. The adhesive drape helps to provide a semi occlusive environment that supports moist wound healing, which has been the standard for wound care since the mid-1980s. The drape is vapour permeable to facilitate gas exchange, an important consideration when treating wounds infected with anaerobic organisms that would thrive in an occlusive, oxygen-depleted environment. The foam and

drape also protect the wound base from environmental contaminants and reduce the risk of friction or shear, enhancing the body's ability to heal.

Peripheral Edema and Circulation

The tissue surrounding a wound is typically characterized by a localized buildup of interstitial (third-space) fluid. This fluid mechanically compromises the circulatory and lymphatic systems, impeding oxygen and nutrient delivery to the tissue and supporting inhibitory factors and bacterial growth. Stagnant wound fluid has been shown to contain elements that delay wound healing by suppressing proliferation.

With NPWT, wound fluids are evacuated via a tubing system placed in or on the foam at one end and connected to a disposable canister housed in the therapy unit on the opposite end. Removing this stagnant fluid allows circulation and disposal of cellular waste via the lymphatic system. Laser Doppler flow studies have shown a significant increase in blood flow adjacent to a wound receiving negative pressure as a result of decreased peripheral edema.

Bacterial colonization

When microorganisms invade tissue, infection is present (defined as greater than 10^5 organisms per gram of tissue). These microorganisms consume the nutrients and oxygen that would otherwise be directed toward

tissue repair. They also release enzymes that break down protein, which is an important component in wound repair. Reducing the bacterial load of a wound improves its healing capacity because the body can then concentrate on healing rather than on fighting invasion from bacteria, viruses, or yeast. NPWT can accomplish this by reducing the amount of stagnant infected fluid in the wound. As mentioned earlier, circulation is enhanced when interstitial fluid is removed. Any increase in circulation and oxygenation to compromised tissue improves the resistance to infection, allowing healing to progress.

Granulation Tissue

Granulation tissue is a mixture of small blood vessels and connective tissue in the base of a wound. This base forms a nutrient-rich matrix that can support the migration of epidermal cells across the wound bed. A well-granulated wound provides an optimal bed for epidermal migration and for skin grafts as the newly formed capillaries incorporate the transplanted skin. Studies have shown that granulation tissue formation is enhanced by negative pressure by virtue of interstitial fluid resolution and the resulting increase in circulation.

Controlled negative pressure assists in wound healing by

Providing a moist, protected environment

Reducing peripheral edema around the wound

Stimulating circulation to the wound bed

Decreasing bacterial colonization

Increasing the rate of granulation tissue formation and epithelialization.

The science behind NPWT is significant because it enhances in vivo reparative mechanisms to promote wound healing. NPWT does not replace surgical procedures, but may allow the wound to progress to the point that a less-invasive procedure is possible.

NPWT has demonstrated positive outcomes in a variety of wounds, but guidelines and safety measures developed using evidence-based research must be diligently maintained to protect the patient. Any therapy has the potential for harm when not used appropriately.

Indications and contraindications for the use of VAC

1. The principal indications for the use of VAC are

- Acute and traumatic wounds
- Sub acute wounds (i.e. dehisced incisions)
- Pressure ulcers
- Chronic open wounds (stasis ulcers and diabetic ulcers)
- Meshed grafts
- Venous stasis ulcers
- Lower extremity diabetic ulcers
- Pressure ulcers
- Lower extremity flaps

2. Contraindications

- Fistulas to organs or body cavities
- Necrotic tissue or eschar
- Osteomyelitis (untreated)
- Malignancy in the wound

Type of Foams used

Granu Foam This black, polyurethane (PU) foam dressing has reticulated (open) pores to help evenly distribute negative pressure across the wound bed, assisting in tissue granulation formation in wounds and aiding wound contraction. It is hydrophobic (or moisture repelling), which enhances exudate removal.

Granu Foam Silver This is an open-celled, reticulated polyurethane foam that has been micro bonded with metallic silver via a proprietary metallization process. During V.A.C. Therapy, exposure of the dressing to wound fluid results in oxidation of metallic silver to ionic silver, allowing the continuous, sustained release of silver ions that act as an effective barrier to bacterial penetration.

White Foam Dressing This white polyvinyl alcohol foam is a dense, open-pore foam with a higher tensile strength for use in tunnels and undermining. It is hydrophilic (or moisture retaining) and is packaged pre-

moistened with sterile water. Its characteristics help to reduce the likelihood of adherence to the wound base, thereby, reducing the need for additional interposing materials. White Foam Dressing may be used to assist in minimizing discomfort, over fresh split thickness skin grafts (STSG), or in situations where hype granulation responses are likely. The higher density of White Foam Dressing requires minimum pressure setting of 125mmHg.

FOAM CHOICE RECOMMENDED

Wound Description	Polyurethane (black foam)	Polyvinyl alcohol (soft foam)	Both	Either
Deep, acute wounds with moderate granulation tissue growth	X		X	
Deep wounds with extremely rapid growth in granulation tissue				
Deep pressure ulcers	X			
Superficial wounds				X
Post graft therapy		X		
Fresh grafts	X			
Compromised flaps	X			
Tunneling/sinus tracts/undermining				X
Diabetic ulcers		X		
Dry wounds	X		X	
Deep trauma wounds				X
Superficial trauma wounds				

SUBATMOSPHERIC PRESSURE RECOMMENDED

Wound Type	Rationale for Use	Initial Cycle	Subsequent Cycles	Target Pressure Polyurethane	Target Pressure Polyvinyl-alcohol	Dressing Change Interval
Acute/traumatic wound	Edema removal, wound contraction, granulation growth, protection from outside contaminants	Continuous for first 48 hours	Intermittent (5 min on/2 min off) for duration of therapy	125 mm Hg	125-175 mm Hg	Every 48 hours (every 12 hours with untreated infection)
Surgical wound dehiscence	Edema removal, wound contraction, granulation growth, protection from outside contaminants	Continuous for first 48 hours	Intermittent (5 min on/2 min off) for duration of therapy	125 mm Hg	125-175 mm Hg	Every 48 hours (every 12 hours with untreated infection)
Meshed graft	Edema removal, adhere graft to wound bed, protect against shearing forces	Continuous	Continuous for duration of therapy	75-125 mm Hg	125 mm Hg; titrate up for more drainage	None; remove dressing after 3-5 days when using either type of foam
Pressure ulcer	Granulation tissue growth, edema removal, wound contraction, moist healing environment, protection from outside contaminants	Continuous	Intermittent (5 min on/2 min off) for duration of therapy	125 mm Hg	125-175 mm Hg; titrate up for more drainage	Every 48 hours (every 12 hours with untreated infection)

Chronic ulcer (diabetic/arterial vascular)	Edema removal, granulation tissue growth, enhance epithelial cell migration, provide moist wound healing, protection from outside contaminants	Continuous	Continuous for duration of therapy	50-75 mm Hg	125-175 mm Hg; titrate up for more drainage	Every 48 hours (every 12 hours with untreated infection)
Fresh flap	Surgical/wound drainage removal underneath sutures, promotes flap adherence to wound base, helps immobilize flap, protects from contaminants	Continuous	Continuous for duration of therapy	125 mm Hg	125-175 mm Hg; titrate up for more drainage	Every 72 hours (every 12 hours with untreated infection)
Compromised flap	Edema removal, granulation tissue growth, adherence of flap	Continuous	Continuous for duration of therapy	125 mm Hg	125-175 mm Hg; titrate up for more drainage	Every 48 hours (every 12 hours with untreated infection)

CLINICAL PROFORMA

PROF T.C.CHANDRAN'S UNIT

STUDY OF WOUND BED PREPARATION

NAME : PS NO :

AGE /SEX : DATE OF ENTRY :

OCCUPATION :

:

DIAGNOSIS :

HISTORY

History : trauma /infection/spontaneous

Duration: weeks/ months/years

Past similar illness:

Risk factor : DM/HTN/PVD/CVI/ lymphoedema / vasculitis

Tobacco / alcohol

Duration

Quantity/day

Treatment history

GENERAL EXAMINATION

Anaemia /copd /chf/

Jaundice /anasarca

LOCAL EXAMINATION

No

Situation

Size

Shape

Edges

Floor

Discharge

Base

Surrounding skin

WOUND BED SCORING
VINCENT FALANGA

	0	1	2
ESCHAR/SLOUGH	>25%	1-25%	0
ECZEMA	Severe	Moderate	Mild
DEPTH	Severely depress	Depressed	Flat
WOUND BED COLOR	Severe	Moderate	None
EDEMA	<50% Pink	50-75%	>75%
RESURFACING EPITHELIUM	<25% Healing edges	25-75%	>75%
EXUDATES AMOUNT	Severe	Moderate	Mild

ARTERIAL EXAMNATION

Pain (claudication/rest pain)
 Paraesthesia
 Temperature
 Skin changes
 Capillary refilling
 Pulsation right left
 Femoral
 Popliteal
 Ant tibial
 Dorsalis paedis
 Post tibial
 Duplex Doppler

VENOUS EXAMNATION

Varicosity(LSV/SSV)
 Hyper pigmentation
 Lipo dermatosclerosis
 Trendelenburg test
 Duplex doppler

LYMPHATICS EXAMNATION

Grade 1
 Grade 2
 Grade 3
 Grade 4

SENSORY EXAMINATION

INVESTIGATION

Heamoglobin

PCV

Blood sugar

RBS

FBS

PPBS

Serum proteins

Serum Albumin / globulin

Others

X ray of local part

Wound biopsy- semi-quantitative bacterial analysis

Biochemical analysis

- tissue collagen
- tissue hydroxyl proline

MATERIAL & METHODS

This study was done in department of plastic surgery, Stanley medical college. The study period was between Jan 2007 and Dec 2008. In this study 2 devices were used as follows & results were analysed.

- 1) Chronic wounds - Electrical stimulation.
- 2) Post traumatic wounds – Negative pressure wound therapy.

CHRONIC WOUNDS - ELECTRICAL STIMULATION

During this study period, 37 patient with chronic superficial wounds registered in our department were included in this study.

Patients with

- 1) Malignant ulcer
- 2) Ischaemic ulcer
- 3) Chronic Osteomyelitis
- 4) Patient with cardiac Pacemaker, were not included for the study.

Thirty Seven Patients with chronic superficial wounds were randomly divided into 2 groups.

- 1) **Group I** - for whom electrical stimulation was given for 3 weeks.
- 2) **Group II** - Regular saline dressing were done for 3 weeks.

None of the group was treated either with antibiotics (or) analgesics.

Out of the 37 patients, two patients had two ulcers and among that one was kept as control and other was kept as study group. Two patients were initially treated as control group for 3 week subsequently these two patients were treated by electrical stimulation for 3 week.

All the patients included in the study were examined as per our protocol. The patients included in the study were evaluated for systemic co-morbid illness like Diabetes mellitus, malnutrition, anemia etc, and treated accordingly.

Detailed clinical examination of the local wounds done as per the clinical Proforma. The response of the wounds for either electrical stimulation or saline dressing were analyzed based on the serial assessment results.

The serial assessment were done initially before the treatment is started. Similar assessment were repeated at the end of week 1, week 2 and week 3 and analysed.

Following clinical parameter were serially assessed

a) clinical examination

- 1) Size & shape
- 2) Quantity of exudates
- 3) Peri wound edema/eczema.
- 4) Wound edge
- 5) Colour of the granulation tissue
- 6) Perception of pain

b) Peri metric analysis

The wound surface area is assessed by tracing the wounds in a transparent sheet.

c) Photographic analysis

Serial photographic analysis of the wounds were done in standard positions. The change in size of the wounds could be assessed by comparing the previous photographs.

d) Bacteriological Analysis

The weekly wound biopsy were done to assess the bacterial bio burden. Bacteriological analysis were done by semi quantitative assay method. The quantitative and qualitative measure were done to identify the organisms and its load in the tissue. The load is expressed in terms of number of colony forming units per gram of tissue.

Group I (ELECTRICAL STIMULATION)

Apart from necessary debridement, the wounds were cleaned with normal saline. Daily Electrical stimulation for the wound is given for 40 min per day for a period of 3 weeks.

Type of Current

The electrical current which we deliver to the local wound is an Biphasic pattern of Alternating current with voltage of 50 volts, frequency of 100 hertz and pulse with of 150 nm/sec. were given. The wave pattern is

Rectangular Biphasic Pattern.

Mode of Delivery

This electrical stimulator has two electrodes, an active electrode and an indifferent electrodes. The polarity of the electrodes keeps changing

ELECTRIAL STIMULATOR



APPLICATION OF ELECTRIAL STIMULATION



alternatively. The frequency of change in polarity depends on the pulse width.

The wound is cleaned and covered with a single layer of moist gauze. An aluminum foil of the same size and shape of the wound is cut and placed over the wound. The active electrodes of the electrical stimulator is fixed to the aluminum foil by alligator clips. The indifferent electrode is placed over a moist gauze and kept over the peri wound skin. The indifferent electrode is kept approximately 5-10 cm apart and secured in place by bandaging. The current is delivered for 40 min/ day

. Following the electrical stimuli for 40 minutes, electrodes were removed and normal saline dressing done for the wounds.

No antibiotic (or) Analgesic are given during this period.

These wounds are examined weekly.

Group II (CONTROL)

This group of patients were treated conservatively by daily saline irrigation and semi occlusive dressing. Debridement was done as and when required. No antibiotic (or) antiseptics used to manage these wounds. These wounds were serially examined at the end of every week.

POST TRAUMATIC WOUNDS – NEGATIVE PRESSURE WOUND THERAPY

During the above study period 20 cases of infected post traumatic wounds were included in the study. All the patients included in the study had evidence of infection and inflammation. These wounds were clinically not fit for definitive surgical management. All these patients had an wound bed score 6 or < 6. These patients are grouped randomly into two groups, group I and II.

Group I (NEGATIVE PRESSURE WOUND THERAPY)

These patients were examined in detail as per our Proforma and graded as per **Falangas Wounds Bed Score**. These wounds were debrided on day 1. After 24 hrs of debridement they are subjected to negative pressure wound therapy

Npwt Application

The wounds were cleaned with normal saline. A foam layer of exact size and shape of the wound is placed over the wound. Then a lateral vented tube is placed over it. Then again a similar foam layer is placed over the tube and secured in place by applying firm bandage. Then wound is covered with a thin membrane bag and water tight closure is achieved. Then the drain tube is connected to the central suction unit and negative pressure

CENTRAL SUCTION UNIT WITH ADJUSTABLE PRESSURE GAUGE



NEGATIVE PRESSURE THERAPY APPLICATION



is maintained gradually from 75 to 125mm Hg. The negative pressure is applied in an continuous mode.

The serial assessment of the wounds were done every 48 hrs to 72 hrs to predict the outcome.

They are

- 1) Clinical evaluation - Falnga's wound Bed scoring system.

WOUND BED SCORING
VINCENT FALANGA

	0	1	2
ESCHAR/SLOUGH	>25%	1-25%	0
ECZEMA	Severe	Moderate	Mild
DEPTH	Severely depress	Depressed	Flat
WOUND BED COLOR	Severe	Moderate	None
EDEMA	<50% Pink	50-75%	>75%
RESURFACING EPITHELIUM	<25% Healing edges	25-75%	>75%
EXUDATES AMOUNT	Severe	Moderate	Mild

- 2) Photography's in standard views.
- 3) Wound biopsy for Biochemical Analysis.
 1. Tissue collagen level.
 2. Tissue Hydroxy proline level.

Subsequent debridement done as and when required. Once the wound bed score has improved > 12 the wounds are considered as fit for definitive surgery and the Negative pressure wound therapy is stopped.

Group II (CONTROL)

These patients similar to group I were post traumatic wounds with a wound bed score of < 6 , who are not fit for definitive surgery. They were clinically assessed by wound bed scoring. Debridement was done on day 1. After 24 hrs of debridement, the wound were inspected and regular dressing done with normal saline. Subsequent debridement done as and when required. The wounds were clinical reassessed every 48 to 72 hrs.

Wound Biopsy was done once in three to four days to estimate the tissue collagen and tissue hydroxyl proline level. The wounds are followed up till the wound bed score increases more than 12.

OBSERVATION & RESULTS

CHRONIC WOUNDS TREATED BY ELECTRICAL STIMULATION

In this study of chronic superficial wounds there were 37 patients. Among the 37 patients, 2 patients had multiple wounds. Two patients who were initially kept as control were later treated by electrical stimulation.

All patients included in the study had wounds in the extremity. All except 2 patients had wounds in the lower extremity.

On analysis of the aetiology of chronic wound in the study diabetes mellitus was most common cause. In the next common category no specific aetiology could be ascertained. Out of the 11 patients who fall in this group 8 patients were habitual smokers.

The various aetiology of chronic ulcers encountered in the study are

Diabetic Ulcer	12
Non Specific Ulcer	11
Venous Ulcer	8
Chronic Ulcer with lymph edema	6
Un stable scar	3
Others	

(Table No-1)

Most of the patients were in the age group between 41-60 yrs. The youngest patient was 23 years of Age and the oldest was 76 years.

Most of the patients with chronic ulcer were males.

Patients who were treated with electrical stimulation had significant decrease in pain, oedema and exudates. The granulation tissue became pink in 16 out of 22 cases at the end of first week. The healing rates of the wounds treated with electrical stimulation were significantly better when compared to the control. The average healing rate of the wound treated by electrical stimulation was 23.04 % / week, and that of the control was 17% per week.

A healing rate of 15% per week is the accepted standard for chronic non-healing wounds. The effective rate of healing even in control in the study can be explained by the fact that all this wounds are superficial.

The healing rate of the wound treated with electrical stimulation ranges between 17.1% - 29.3%. For control the range is from 9.8 to 22.8% (Table No-2).

	Healing rate per week	Average healing rate per week
Electrical stimulation	17.1%-29.3%	23.04%
control	9.8%-22.8%	17.0%

(TableNo-2)

ELECTRICAL VS CONTROL HEALING RATE

ELECTRICAL STIMULATION



ELECTRICAL STIMULATION

ELECTRICAL STIMULATION



ELECTRICAL STIMULATION

DAILY SALINE DRESSING



ELECTRICAL STIMULATION

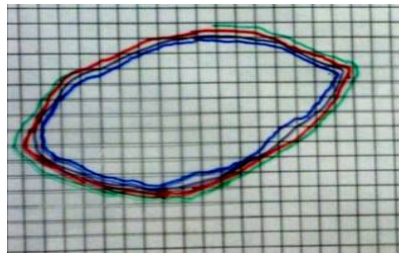
ELECTRICAL VS CONTROL HEALING RATE



CONTROL – DAY 1



CONTROL – 3 WEEKS



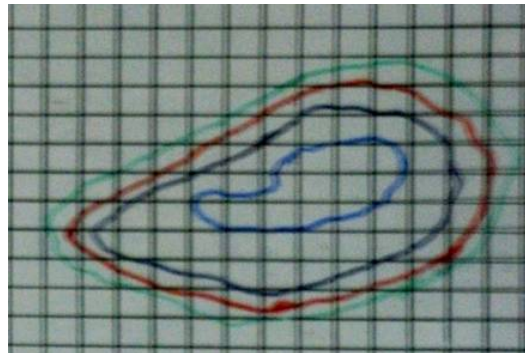
13% DECREASE



ELECTRICAL – DAY 1



ELECTRICAL – 3 WEEKS



25% DECREASE

ELECTRICAL VS CONTROL HEALING RATE



CONTROL - DAY 1



CONTROL - 1 WEEK



CONTROL - 3 WEEKS



HEALED - 6 WEEKS



ELECT - DAY 1



ELECT - 1 WEEK



ELECT - 3 WEEKS



ELECT - POST OP

Bacterial bio burden

Out of the 41 cases of chronic wounds, 58.5% of the cases had significant bacterial count and only 17.07% cases had no growth in culture at the initiation of treatment. The commonest gram-negative organism cultured in the chronic wounds was **pseudomonas aeruginosa** and the commonest gram-positive organism encountered is **staphylococcus aureus**.

The other organisms encountered were klebsiella, Escherichia coli.

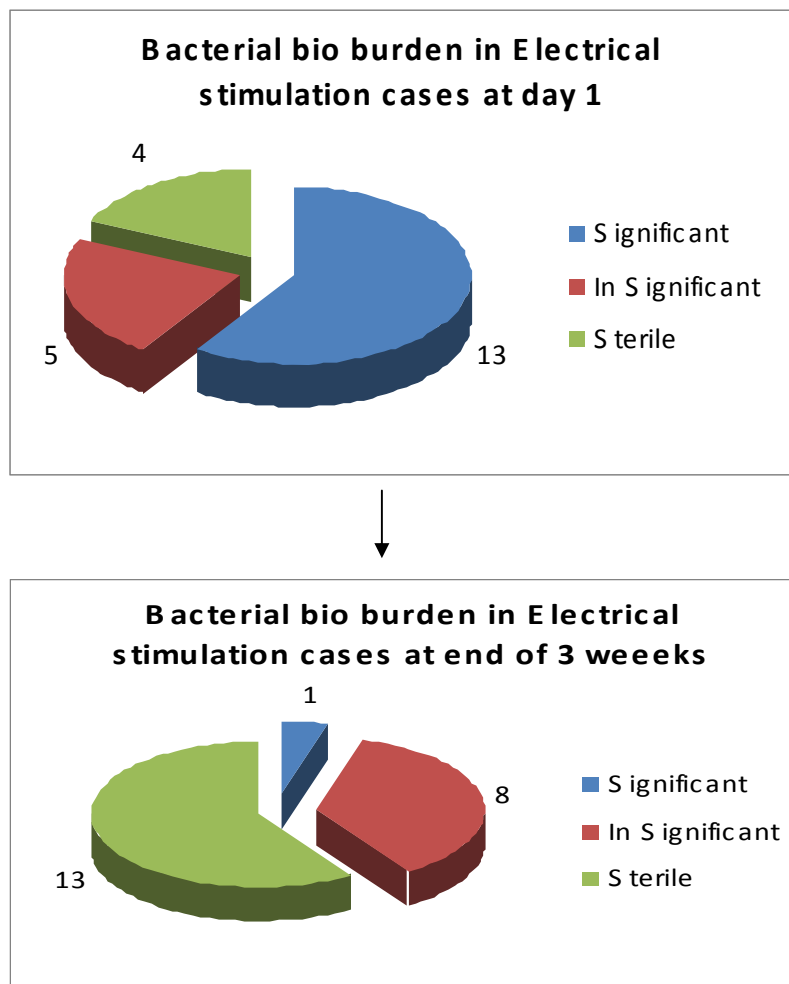


CHART - 1

Out of the 22 patients treated with electrical stimulation 13 patients had significant bacterial count ($>10^5$ cfu/gm of tissue) at the time of initiation of therapy. At the end of 3 weeks, the bacterial count has decreased and only 1 patient had significant bacterial growth.(chart 1)

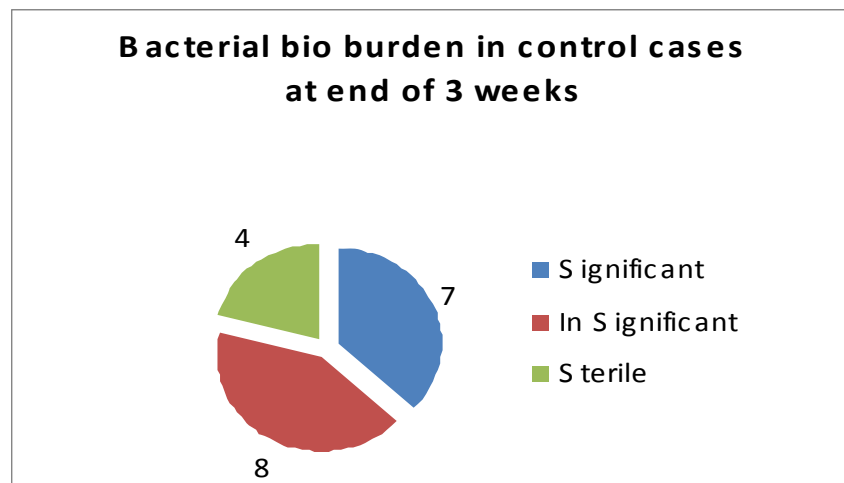
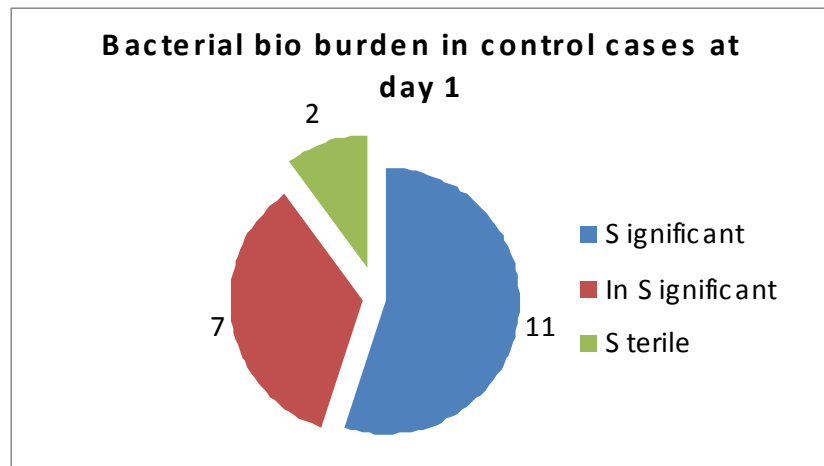


CHART - 2

The 19 cases which were taken as control, were treated only by saline dressing. Of these 11 patients had significant bacterial at the start of therapy. At the end of third week, 7 patients had significant bacterial count.(chart 2)

COMPARISON OF HEALING RATES OF DIFFERENT TYPES OF CHRONIC WOUNDS

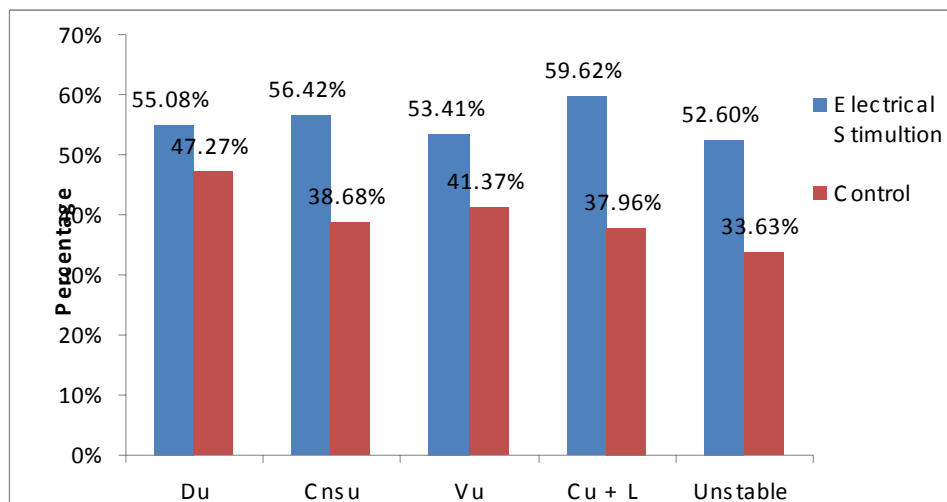


CHART - 3

- Du - Diabetic ulcer
- CNSU - Chronic non-specific ulcer
- Vu - Venous ulcer
- Cu+L - Chronic ulcer with lymphodema
- Unstable - Unstable scar

The healing rate of different type of chronic wounds were analysed and it was found that all types of chronic wounds showed better response with electrical stimulation than control.(chart 3)

POST TRAUMATIC WOUNDS -NEGATIVE PRESSURE WOUND THERAPY

20 patients with post traumatic wounds of more than 1 week duration were included in the study. Out of which, 11 patients underwent negative pressure wound therapy. Remaining 9 patients were treated only by saline dressing and kept as control group. Antibiotic, antiseptics were not used in any of these patients.

All the patients included in the study had wounds in the lower extremity. All patients included in the study had a wound bed score of 6 or < 6, hence they are not amenable for immediate wound closure. The age group of patients included in the study ranges from 20-60 years.

On analysis, we found that the cases treated by negative pressure wound therapy (except two extensively bone exposed cases) required 6.3 days to get converted from average pre treatment score of 4.18 to average post treatment score of 13.09.

The average number of debridements required for cases treated by negative pressure wound therapy was 1.5. (Table – 4).

NEGATIVE PRESSURE WOUND THERAPY TREATED CASE



PRE TREATMENT



DEBRIDEMENT-DAY 1



DAY - 3



DAY - 7



POST OP

NEGATIVE PRESSURE THERAPY - BONE EXPOSED CASE



DAY 1



DAY 3



DAY 7



DAY 14



DAY 17



POST OP

COMPARISON OF NPWT AND CONTROL

	No. of Debridement	Duration of required for WBP (days)
NPT	1.5	6.3
Saline dressing (control)	2.1	12

TABLE - 4

On an average, in cases treated by NPWT there was 2.95 folds increase in collagen content in negative pressure wound therapy treated cases within a period of 6.3 days and 28.4 % decrease in hydroxyl proline. (Table-5) There was a significant decrease in wound and exudate. Granulation tissue increased significantly within a short period.

BIO CHEMICAL PARAMETERS IN NPT AND CONTROL

	Percentage of Folds of increase in collagen	Decrease in percentage of tissue hydroxy proline
NPT	295	28.4
Saline dressing (control)	170	16.7

TABLE - 5

The two extensively bone exposed cases were post traumatic wounds with exposure of entire subcutaneous surface of tibia for wound coverage. These cases required flap cover as they were devoid of periosteum. As we treated these cases over a period of 3 weeks with negative pressure wound therapy, there was an over growth of granulation tissue covering the bone

with a thin layer of separated sequestrum. Thus these two patients could be managed by simpler surgical option of split skin grafting.

POST TRAUMATIC WOUNDS – CONTROL

The cases which were kept as control and treated only by saline dressing required 12 days for wound bed scores to improve from 3.8 to 12.3. (Table-4)

The average number of debridements required for these cases was 2.1 which was far more than the patients treated with negative pressure wound therapy. (Table-4)

On an average there was 1.7 folds increase in collagen in control cases, within a period of 12 days and also 16.5% decrease in hydroxyl proline. (Table-5)

This indicates that the healing process is significantly less in wounds treated without negative pressure wound therapy.

Even on an average period of 12.3 days, the time required for the wound to become fit for wound closure (WBS > 12) there was only 1.7 fold increase in collagen an 16.5% decrease in hydroxy proline.

CONTROL CASES



DAY 1



DAY 4



DAY 8



DAY 15



**FIRST DEBRIDEMENT DONE ON DAY 1
SECOND DEBRIDEMENT DONE ON DAY 5**

CONCLUSION

ELECTRICAL STIMULATION

1. Electrical stimulation improves the local vascularity in 3 -7 days and converts the unhealthy wound bed of chronic superficial wound in to one that is fit for skin cover.
2. Electrical stimulation drastically reduces the bacterial bio-burden to insignificant level over a period of 3 weeks.
3. On an average electrical stimulation increases the healing rate to 23% per week which amounts to 18% more as compared with control over a period 3 weeks.

NEGATIVE PRESSURE WOUND THERAPY

1. The NPT treated wounds are converted from extension phase to transition phase in 6 days which is half the period that is required for control.
2. Bio chemical analysis has shown that cases treated with negative pressure wound therapy had a significant increase in collagen synthesis and increased utilization of hydroxy proline than control, indicating increased production and utilization of components of wound healing.
3. Negative pressure therapy simplifies the surgical management even in cases with extensive exposure of bone. A complex wound which needs a flap cover can be converted in to simple wound which will accept a split skin graft. This is quiet evident in the case shown in photograph.

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