ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 07, pp.54139-54142, July, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

EFFECT OF VACUUM ASSISTED CLOSURE ON WOUND HEALING

***,[1]Dr. Kanav Mahajan, [2]Dr. Mohinder Singh Chib and [3]Dr. Sanjeev Gupta**

[1]Junior Resident Post Graduate Department of Orthopedics GMC Jammu
[2]Senior Resident Post Graduate Department of Orthopedics GMC Jammu
[3]Associate Professor Post Graduate Department of Orthopedics GMC Jammu

ARTICLE INFO

Article History:
Received 21st April, 2017
Received in revised form
09th May, 2017
Accepted 05th June, 2017
Published online 26th July, 2017

Key words:
VAC, Wound healing.

ABSTRACT

Vacuum Assisted Closure Vacuum assisted closure (VAC) is a relatively new technology with applications in a variety of difficult to manage acute and chronic wounds. It is known by many pseudonyms—TNP (topical negative pressure) SPD (sub-atmospheric pressure) VST (vacuum sealing technique) and SSS (sealed surface wound suction)

Citation: Kanav Mahajan, 2017. "Effect of vacuum assisted closure on wound healing", *International Journal of Current Research*, 9, (07), 54139-54142.

INTRODUCTION

Dressings to treat complex wounds have traditionally been made of cotton gauze which could be soaked with a variety of chemicals including normal saline and sodium hypochlorite solutions. In the1960s, the importance of keeping the wound moist was discovered and a wide array of hydrogels, alginates, and other polymeric and biologically based dressings were developed. More recently, these dressing materials have been combined with antimicrobial compounds such as silver. Most of these products are used based on clinicians experience of ten coupled with invitro and in vivo experimental work and limited noncontrolled clinical trials. For the average clinicians, finding the best dressing for a specific wound can be a daunting task. The VAC was first investigated by Morykwas and Argenta *et al*. in 1997 (Morykwas *et al*., 1997). Their work followed on from studies of negative pressure years previously that had suggested it might improve wound healing. Early work suggested that negative pressure increased blood flows as evidenced by hyperaemia (Greer, 2000). Moryk was and Argenta (Morykwas *et al*., 1997) used a swine model to investigate the effect of negative pressure applied via the VAC on wound healing. The aim of this work was to get the maximum benefit of VAC in the management of acute and chronic wounds without abuse or misuse.

****Corresponding author:*** **Kanav Mahajan,**
Junior Resident Post Graduate Department of Orthopedics GMC Jammu

The VAC Technique

VAC uses medical grade open cell polyurethane ether foam (which is FDA approved for open wounds) as a dressing (Morykwas *et al*., 1997). The pore size is generally 400–600 mm (thought optimal for tissue growth) (Morykwas *et al*., 1997). This foam is cut to fit and closely applied to the selected wounds. An evacuation tube with side ports, which communicate with the reticulated foam, is embedded in it. The aim of the reticulation being that the negative pressure will be applied equally to the entire wound bed. An adhesive drape is then applied over the area with an additional 3–5 cm border of intact skin to provide an intact seal. The evacuation tube is connected to an adjustable vacuum pump and a canister for collection of effluent. The pump can be adjusted in terms of both the timing (intermittent vs. continuous) and magnitude of the vacuum effect. In general an intermittent cycle (5 min on, 2 min off) is employed as this has been shown to be most beneficial (Morykwas *et al*., 1997). Effectively the technique converts an open wound into a controlled and temporarily closed environment.

Mechanisms of action

Wounds generally heal by primary, where edges are brought into close apposition for example by suturing, or secondary intention, where the wound edges are not opposed and a matrix of small blood vessels and connective tissue must be formed in between in order for keratinocytes to migrate across the surface and re-epithelialise the defect. It is a complex, intricate process. The aims of the process can be considered as minimization of blood loss, replacing any deficits with new tissue (granulation) and restoring an intact epithelial barrier as quickly as possible. In order to achieve healing debris must also be removed; any infection controlled and inflammation eventually cleared. The wound then heals with granulation, remodeling of the connective tissue matrix and finally maturation. The rate of healing may be limited by vascular supply and the capacity of the wound to form new capillaries/matrix. Any disruption in the various processes involved—proliferation, angiogenesis, chemotaxis, migration, gene expression, protein production can lead to a chronic wound. When we consider the postulated mechanisms of action of the VAC and how they tie in with our generally accepted ideas surrounding the way wounds heal it is easy to suppose a likely benefit in quicker resolution of wounds.

Removal of oedema/exudate management

Localised oedema can compress the vascular and lymphatic systems in a wound. The VAC removes excess fluid and, therefore, is thought to restore the vascular and lymphatic flow (Banwell and Teotl, 2003). Practically, the next best wound dressing (wet to moist saline dressings) involves labour intensive and potentially hazardous dressing changes. The VAC ensures a closed environment for wounds and, therefore, adheres to universal precautions. They also need to be accessed far less frequently (Banwell and Teotl, 2003). The VAC system allows collection of the removed fluid for analysis. Some centres (Wysocki*et al*., 1993; Yager and Nwomeh, 1999) have shown high levels of proteolytic enzymes in chronic wounds and burns. These enzymes if left in situ could result in matrix degradation and a non-healing wound environment. They have also been demonstrated in the effluent from VAC treated wounds along with cytokines and various acute phase proteins (Gustaffson *et al*., 2002; Buttenschoen *et al*., 2001).

Reduction in levels of bacteria

Infection is known to impede wound healing. Reduced levels of bacteria have been demonstrated in VAC treated wounds. (Morykwas *et al*., 1997) It has also been demonstrated that VAC treated wounds require fewer courses of antibiotics relative to conventionally treated wounds (Gustaffson *et al*., 2002; Buttenschoen *et al*., 2001). This is thought to be due to multiple factors: the positive effect of removing excess wound fluid on local blood and lymphatic flows, greater amounts of oxygen made available for the bacteria killing oxidative bursts and the closed nature of the system. Gouttefangeas *et al*. (2001) measured the cellular content of the foam from the VAC and found high levels of granulocytes, cd4, cd5 and T cells. They supposed the foam to be an attractive habitat for immune cells, partially recruited by a foreign body reaction. However, one recent study of 25 patients undergoing VAC treatment suggests there may be a negative effect on bacterial clearance (Weed *et al*., 2004). In this study, serial cultures showed that bacterial colonization increased significantly during treatment. Though of note, the treatment was beneficial in most cases with rapid wound healing and there were no controls to provide meaningful comparison.

Mechanical stress causing granulation tissue formation and angiogenesis

Ilizarov *et al*. showed that applied mechanical stress to tissues stimulated mitosis and found that new vessels were formed as a result (Ilizarov, 1989; Saxena *et al*., 2004). This and the reduction in oedema could explain changes in blood flow and new vessel formation. However, further theories suggest that the vacuum may directly affect vasomotor tone and vaso-active mediators or simply have a mechanical effect forcing the blood through the area more quickly (Banwell and Teotl, 2003). It is thought that in vivo the external forces applied to cells through the extra cellular matrix are balanced by intracellular cytoskeletal forces with integrins acting as trans-membrane bridges. This balance is thought to be disturbed by application of the VAC leading to release of various intra cellular second messengers which together cause changes in expression of immediate early genes which results in matrix molecule synthesis and, therefore, proliferation (Ilizarov, 1989). The presumption is that this also occurs when the vacuum on the foam is released, causing progressive up-regulation when the vacuum is applied intermittently. This is born out by the finding of more granulation tissue in wounds where the vacuum is applied intermittently (Morykwas *et al*., 1997). Recent work in China (Shi *et al*., 2003) also found that levels of the matrix metalloproteinases 1, 2 and 13 were reduced over time in five chronic wounds treated by the VAC technique. Lower levels of these MMP's would presumably lead to reduced collagen and gelatin breakdown and aid wound healing. A further Chinese group recently examined the protein BcL-2 (Tang *et al*., 2004).They found that expression of this protein was increased in an animal modelon treatment with VAC therapy. This protein helps modulate apoptosis, and the results of the study suggest that the VAC may promote the healing process partly through modulation of apoptosis.

Reverse tissue expansion/skin stretching

The vacuum is thought to encourage migration of keratinocytes across wound defects. A pure in-drawing produced by the vacuum, the so-called 'mechanmechanical creep' effect. This is well seen in abdominal wounds treated by the VAC where a centripetal effect is observed (Fenn and Butler, 2001). It can also be compared to the stretching of tissue produced by tissue expanders used prior to skin grafting procedures.

Complications

When used within recommendations, complications resulting from the use of the VAC are infrequent—but do occur.

1. **Pain:-** Most wounds treated by the VAC are painful by their very nature—for example; burns, pressure sores and infected wounds. However, as with all 'dressings' this pain can be exacerbated by dressings changes. Although VAC treatment carries the advantage of fewer dressings changes, strategies to minimize pain should be employed (Krasner, 2002).
2. **Infection:-** One case report has been published highlighting the occurrence of Toxic Shock Syndrome following VAC treatment (Gwan-Nulla and Casal, 2001). Frank pus within the wound to be treated is a contra-indication to treatment with the VAC. The manufacturers and body of opinion in the literature suggest that the wound bed should be debrided and prepared prior to treatment and any necessary antibiotic treatment commenced. However, the original studies reported enhanced clearance of bacteria in VAC treated subjects (Morykwas *et al*., 1997) and the clinical experience reported by many groups suggests a positive

effect on avoidance of infection. No direct comparison has been made in a clinical trial of infection rates specifically in VAC vs. non-VAC treated patients.

3. **Fluid depletion:-** One recently published case report mentioned two cases of patients at extremes of age (10 months and 82 years) (Barringer*et al*., 2004). Both patients suffered fluid depletion following VAC treatment for skin loss following meningococcal septicaemia and chronic leg ulcers, respectively. In both cases, large amounts of fluid had been lost from the wounds over the course of their treatment. This problem should be considered and managed appropriately where large amounts of effluent are collected by the VAC.

MATERIALS AND METHODS

The study was conducted in the Department of Orthopedics, GMC Jammu from 16th May 2015 to 15th May 2017 on 100 patients with acute and chronic wounds admitted in Govt. Medical College and Associated Hospital. The patients consent was taken for the procedure they underwent. There was no inclusion or exclusion criteria specified in the study.

OBSERVATION AND RESULTS

1) Sex of patient
   - Male 65
   - Female 35
2) Type of wound
   - Chronic Wounds 60
   - Acute Wounds 40
3) Complications-
   - Duration of pain
     1. 24hrs 60 patients
     2. 3days 40 patients
     3. 5days 20 patients
   - Infection
     1. Infection 10 patients
     2. No Infection 90 patients
   - Fluid Depletion –nil

The hospitalization period varied from 1 to 6 weeks. The follow up period was upto 6 months. No mortality was recorded during this study.

Bacterial growth:

Day 4: 40% no growth,
Day 8: 60% no growth
Decrease in Wound size 10% on 4th day, 20% on 8th day, 40% on 12 day, 60% on 16 day

DISCUSSION

Healing is an intricate, interdependent process that involves complex interactions between cells, the cellular microenvironment, biochemical mediators, and extracellular matrix molecules that usually results in a functional restoration of the injured tissue (Clarke and Henson, 1988; Cohen *et al*., 1992). The goals of wound healing are to minimize blood loss, replace any defect with new tissue (granulation tissue followed by scar tissue), and restore an intact epithelial barrier as rapidly as possible. The rate of wound healing is limited by the available vascular supply and the rate of formation of newcapillaries and matrix molecules (Hunt, 1991). These events are heavily influenced by locally acting growth factors that affect various processes including proliferation, angiogenesis, chemotaxis and migration, gene expression, proteinases, and protein production (Clarke and Henson, 1988). Disruption of any of these factors may adversely affect the healing process, resulting in a chronic or nonhealing wound. Blood flow increases and bacterial colonization of wound tissues decreases following the application of subatmospheric pressure to wounds. Any increase in circulation and oxygenation to compromised or damaged tissue enhances the resistance to infection (Whitby and Ferguson, 1992). Successful, spontaneous healing and healing following surgical intervention are correlated with tissue bacterial counts of less than 105 organisms per gram of tissue (Hunt, 1988). Higher levels uniformly interfere in wound healing. Increase in local tissue oxygen levels reduce or eliminate the growth of anaerobic organisms, which have been correlated to decreased healing rates (Kucan *et al*., 1981; Seiler *et al*., 1979). Additionally, the increased flow should make greater amounts of oxygen available to neutrophils for the oxidative bursts that kill bacteria (Daltrey *et al*., 1981). Our study showed that in VAC group after day 4, there were 40% of patients who had no bacterial growth, and on day 8 there were 60% of patients who had no bacterial growth. From our own experience, the VAC is a promising new technology in the field of wound healing. With multiple applications in a variety of wounds including those that can prove difficult to heal: pressure sores, amputation sites, skin grafts, lower limb ulceration, sternotomy wounds, burns and abdominal wounds. Broadly speaking, the applications are for both acute and chronic wounds, salvage procedures or as an adjuvant therapy to improve results of various surgical procedures. The indications for the VAC as chronic open wounds (diabetic ulcers and stage 3–4 pressure sores) acute and traumatic wounds, flaps and grafts, sub-acute and dehisced wounds as well as partial thickness wounds. They advise that non-enteric and unexplored fistulas and wounds containing necrotic tissue and eschar should not be treated with the VAC. Its use is also contraindicated on wounds containing untreated osteomyelitis or malignancy (due to the effect on cellular proliferation). The scientific basis for the VAC has been tested rigorously and the theories surrounding its' mechanisms of actions explored. Centres around the world continue to explore various applications of the VAC and to attempt to improve the way it is used.

However, the VAC is costly and there is a need for larger randomised controlled trials to prove the effectiveness of the technique in the various patient groups suggested by the current anecdotal evidence.

REFERENCES

Banwell PE, Teotl L. 2003. Topical negative pressure (TNP): the evolution of a novel wound therapy. *J Wound Care*, 12(1):28–30.

Barringer CB, Gorse SJ, Burge TS. 2004. The VAC dressing—a cautionary tale. *Br J Plast Surg*., 57(5):482.

Buttenschoen K *et al*. 2001. The influence of vacuum assisted closure on inflammatory tissue reactions in the post operative course of ankle fractures. *Foot Ankle Surg*., 7(3):165–173

Clarke R. A. F. and P. M. Henson, Eds., 1988. The Molecular and Cellular Biology of Wound Repair, PlenumPress, NewYork, NY, USA.

Cohen, K., R. F. Diegelmann and W. J. Lindblad, 1992. Wound Healing: Biochemical and Clinical Aspects, WB Saunders, Philadelphia, Pa, USA.

Fenn CH, Butler PE. 2001. Abdominoplasty wound healing complications: assisted closure using foam suction dressing. *Br J Plast Surg*., 54:348–351.

Gouttefangeas C, Eberle M, Ruck P. 2001. Functional T lymphoctyes infiltrate implanted polyvinyl alcohol foams during surgical wound closure therapy. *Clin Exp Immunol*., 124(3):398–405.

Greer SE. 2000. Whither sub atmospheric dressing? Guest editorial. *Ann Plast Surg*., 45(3):332–334.

Gustaffson R, Johnsson P, Algotsson L. 2002. Vacuum assisted closure therapy guided by C reactive protein level in the treatment of deep sternal wound infection. *J Thorac Cardiovasc Surg*., 123(5):895–900

Gwan-Nulla DN, Casal RS. 2001. Toxic shock syndrome associated with the use of the vacuum assisted closure device. *Ann Plast Surg*., 47(5):552–554.

Hunt, T. K. 1988. "The physiology of wound healing," *Annals of Emergency Medicine*, vol. 17, no. 12, pp. 1265– 1273.

Hunt, T. K. 1991. "Vascular factors govern healing in chronic wounds," in Clinical and Experimental Approach to Dermal and Epidermal Repair: Normal and Chronic Wounds, A. Barbul,M. D. Caldwell, W. H. Eaglstein *et al*., Eds., pp. 1–17, Wiley & Leiss, *Journal of Pathology*, vol. 138, no. 3, pp. 629–646.

Ilizarov GA. 1989. The tension stress effect on the genesis and growth of tissues. Part 1. The influence of stability of fixation and soft tissue preservation. *Clin Orthop*., 238:249–281.

Krasner DL. 2002. Managing wound pain in patients with vacuum assisted closure devices. *Ostomy Wound Manag*., 48(5):38–43.

Kucan, J. O., M. C. Robson, and J. P. Heggers, 1981. "Comparison of silver sulfadiazine, povidone-iodine and physiologic saline in the treatment of chronic pressure ulcers," *Journal of the American Geriatrics Society*, vol. 29, no. 5, pp. 232–235.

Morykwas MJ *et al*. 1997. Vacuum assisted closure: a new method for wound control and treatment: animal studies and basic foundation. *Ann Plast Surg*., 38:553–562.

Saxena V *et al*. 2004. Vacuum-assisted closure: microdeformations of wounds and cell proliferation. *Plastic Reconstr Surg*., 114(5):1086–1096.

Seiler, W. O., H. B. Stahelin, and W. Sonabend, "Einflus aerobe und anaerober keime auf den heilungsverlauf von dekubitalulzera," Schweizerische Medizinische Wochenschrift, vol. 109, pp.

Shi B *et al*. 2003. Effects of vacuum assisted closure (VAC) on the expressions of MMP-1, 2, 13 in human granulation wounds. *Zhongua Zheng Wai Ke Ka Zhi*., 19(4):279–281.

Tang SY *et al*. 2004. Influence of vacuum-assisted closure technique on expression of Bcl-2 and NGF/NGFFmRNA during wound healing. *Zhongua Zheng Wai Ke Ka Zhi*., 20(2):139–142.

Weed T, Ratliff C, Drake DB. 2004. Quantifying bacterial bioburden during negative pressure wound therapy: does the wound VAC enhance bacterial clearance? *Ann Plast Surg*., 52(3):276–27

Whitby D. J. and M.W. J. Ferguson, 1992. "Immunohistological studies of the extracellular matrix and soluble growth factors in fetal and adult wound healing," in FetalWound Healing, N. S. Adzick and M. T. Longaker, Eds., pp. 161–177, Elsevier Science, New York, NY, USA.

Wysocki AB, Staiano-Coico L, Grinnell F. 1993. Wound fluid from chronic leg ulcers contains elevated levels of metalloproteinases MMP-2 and MMP-9. *J Invest Dermatol*., 101:64–68.

Yager DR, Nwomeh BC. 1999. The proteolytic environment of chronic wounds. *Wound Repair Regen*., 76:433– 441

****