

The Impact of Negative Pressure Wound Therapy on Orthopaedic Infection



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KEYWORDS

- Negative pressure wound therapy • Topical negative pressure • Surgical site infection
- Quorum sensing • Tissue demarcation • Integra • Antibiotic beads • Wound coverage

KEY POINTS

- Topical negative pressure wound therapy (TNP) is used as a dynamic dressing for high-energy wounds following surgical debridement. The author provides support for its utility in this setting as a means of minimizing tissue demarcation (tissue death), the “fuel” of infection.
- TNP is helpful in effecting early wound coverage by eliminating edema and by acting as a dynamic bolster for an applied split skin graft or “artificial skin.”
- TNP has been shown to enhance the survival of random pattern flaps—not infrequently a consequence of the surgical extensions placed on a limb’s transversely directed traumatic wound.
- The use of TNP in the setting of closed surgical wounds has emerged as a safeguard to surgical site infection in certain problematic wound types such as those occurring in the obese patient or those occurring in periarticular fractures of the ankle and tibial plateau.

INTRODUCTION

The author’s personal experience dates from the early 1990s when negative pressure was first introduced by 2 of his colleagues at Wake Forest Medical Center, Lou Argenta, MD and Michael Morykwas, PhD, as a strategy for potentially shortening the hospital stay of patients with decubitus ulcers. The author was one of a small group of clinicians at Wake Forest who gained an early experience with “the VAC” (vacuum-assisted closure, or formerly, the “DecubiVAC”). Given his subspecialty in orthopedic trauma, his proximity to Drs Argenta and Morykwas, and the ready availability of the product, his use of topical negative pressure in wound management quickly became a commonality. He has been a strong proponent of its use in different clinical

settings. Those settings, which are relevant to infection, are elaborated upon in this article.

DEBRIDEMENT

Bernard a raison. La peste est rien. C’est la terrain qui est tout.

(Bernard is right. The germ is nothing. It is the environment which is everything.)

—Louis Pasteur

Ramon Gustilo, MD, teacher, author, and mentor to virtually all orthopedic traumatologists, cited the above in his book entitled, *Orthopaedic Infection* and wrote, “It behooves every surgeon to understand fully the implications of Pasteur’s statement.”¹ Wounded nonviable tissue is the

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fuel of infection and needs to be excised by surgical debridement. For high-energy wounds, the policy of following the initial debridement with a return to the operating room for a "second look" at 36 to 72 hours has become a protocol at many centers, knowing that the tissues in or adjacent to the zone of injury commonly "demarcate" during that span of time.² Tissue "demarcation" is an ill-defined but widely acknowledged concept.

An interesting feature noted in the author's early experience with topical negative pressure wound therapy (TNP) was the fact that when it was used for high-energy wounds following an initial debridement (the author's preference was to set the transmitted negative pressure at -50 mm Hg or -75 mm Hg continuous), there was no or minimal secondary necrosis seen at 36 to 72 hours.³⁻⁵ In other words, there appeared to be something about TNP that prevented the cascade of cellular events that would otherwise proceed to tissue necrosis (or "demarcation") in those patients.

Relevant to these observations was the study by Morykwas and colleagues⁶ on the histology of porcine skin burn wounds, half (on one side of the midline) were managed with saline

dressing changes and half (on the other side of the midline) were managed with TNP. Punch biopsies of the burn wounds were obtained on postburn days 1, 3, 5, and 9 (Fig. 1). The basis for the demise of the tissues managed with saline dressing changes is the lack of dynamic microcirculatory flow through the capillary bed probably due to an engorged interstitium in the tissues. Additional support for this statement is provided by the study of Langfitt and colleagues.⁷ It is now understood that the reason high-energy wounds "demarcate" by the time of the "second look" is the same as was exhibited in the above described porcine burn study of Morykwas.⁶ Given this, one might logically predict that the same phenomenon should apply not only to the tissues at risk in a high-energy extremity wound, a skin burn in a porcine model, but also to any tissue whose initial insult is followed by a second wave of necrosis, such as any infarct with a "reperfusion injury." This line of thought foreshadowed the results of recent studies by Lindstedt and colleagues,⁸ Argenta and colleagues,⁹ and Jordan and colleagues¹⁰ on preservation of blood flow effected by TNP in an ischemic porcine heart

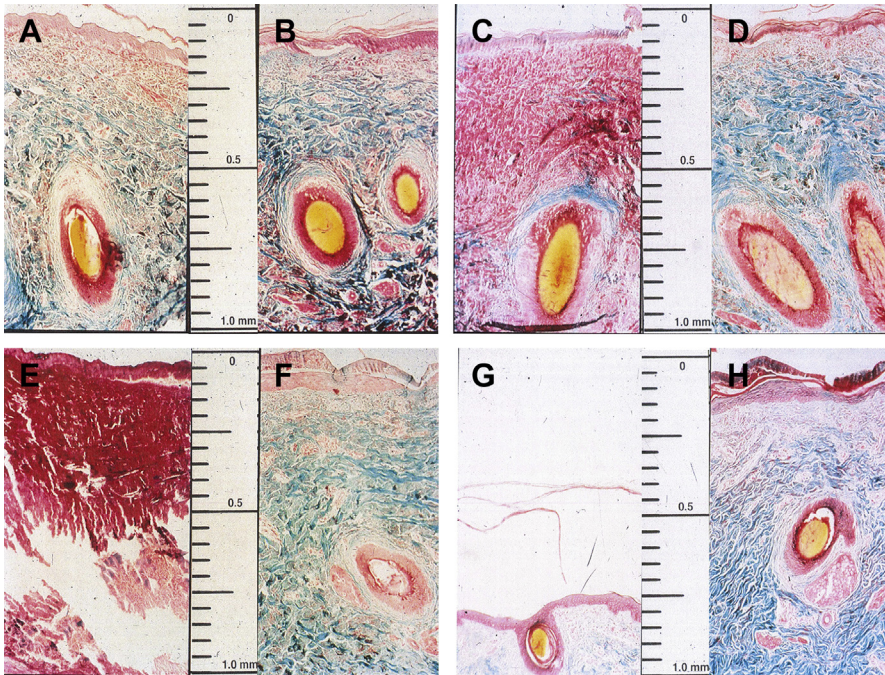


Fig. 1. Punch biopsies of the burn wounds were obtained on postburn days 1, 3, 5, and 9. There were no histologic changes seen on either the saline dressing side (A) or the TNP side (B) at day 1. On day 3, the notable finding on the saline dressing side was a significant zone of coagulation necrosis (C). There were no changes on the TNP side (D). On day 5, there is a clear zone of cleavage between the necrotic zone and the underlying living tissue on the saline dressing side (E). There were no changes on the TNP side (F). On day 9, there was significant sloughing of necrotic tissue with loss of specialized skin elements (G). There were no significant changes on the TNP side (H). From Morykwas MJ, David LR, Schneider AM, et al. Use of subatmospheric pressure to prevent progression of partial-thickness burns in a swine model. *J Burn Care Rehabil* 1999;20(1):15-21; with permission.

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