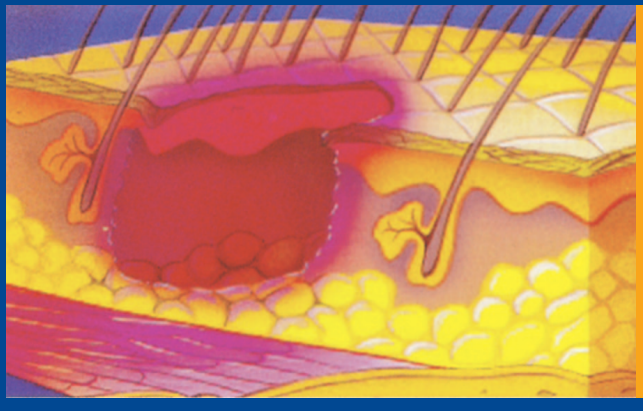
Guidelines for Managing Pressure Ulcers with Negative Pressure Wound Therapy



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ABSTRACT

Pressure ulcers are a serious health issue, leading to clinical, financial, and emotional challenges. Numerous treatment modalities are available to promote wound healing, yet clinicians may be unsure how to incorporate these treatment options into an overall plan of care for the patient with a pressure ulcer. A consensus panel of experienced wound care clinicians convened in July 2004 to review the mechanisms of action and research basis for one such treatment modality: negative pressure wound therapy. After answering key questions about this modality, they developed an algorithm to assist the clinician in making decisions about using negative pressure wound therapy appropriately in patients with Stage III and Stage IV pressure ulcers.

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PRESSURE ULCERS—defined as any lesion caused by unrelieved pressure, resulting in damage of underlying tissue[1]—are acknowledged to be a clinical challenge for both the clinician and the patient. Healing is unpredictable; it often stalls due to such local and systemic factors as bacterial load and infection; edema; pressure; moisture; chronic medical conditions or comorbidities, such as anemia, diabetes mellitus, and renal or hepatic dysfunction; tissue oxygenation; and nutritional status.[2] Because of this, pressure ulcers are considered chronic wounds, defined by Lazarus et al[3] as wounds that have "failed to proceed through an orderly and timely process to produce anatomic and functional integrity, or proceeded through the repair process without establishing a sustained anatomic and functional result."

Technologic advances have given clinicians a myriad of options for managing pressure ulcers, which can lead to improved outcomes of care. The downside, however, is that this product explosion has the potential to cause confusion about which products to use with which wounds and when to discontinue a treatment in favor of another.

In the case of negative pressure wound therapy (NPWT), some clinicians remain unclear as to the best way to use this modality in an overall pressure ulcer treatment strategy. For that reason, a panel of clinicians with expertise in wound management (Table 1) gathered in Chicago in July 2004 to discuss this issue. The panel was charged with (1) evaluating the existing literature base on NPWT and pressure ulcers, (2) evaluating current best practices for pressure ulcer management, (3) developing consensus on guidelines for the use of NPWT in patients with pressure ulcers, and (4) identifying priorities for future research.

For the purposes of this discussion, the term "guideline" was used in the same manner as it was by the Agency for Health Care Policy and Research (AHCPR; now the Agency for Healthcare Research and Quality [AHRQ]) when its series of Clinical

Practice Guidelines was developed.[4] According to the AHCPR, "Guidelines are systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical conditions…The guideline reflects the state of knowledge, current at the time of publication, on effective and appropriate care…The recommendations may not be appropriate for use in all circumstances. Decisions to adopt any particular recommendation must be made by the practitioner in light of available resources and circumstances provided by individual patients."[4]

The definitions of pressure ulcer stages adopted by the AHCPR guideline panel[4] were also followed by the NPWT consensus guideline panel.

INCIDENCE AND PREVALENCE

Although it is accepted that pressure ulcers are a problem to be addressed across care settings, the exact incidence and prevalence are unclear. Reports of pressure ulcer incidence vary widely, from 0.4% to 38% in acute care, from 2.2% to 23.9% in long-term care, and from 0% to 17% in home care, according to a report from the National Pressure Ulcer Advisory Panel (NPUAP).[5] Prevalence rates show the same variability: 10% to 18% in acute care, 2.3% to 28% in long-term care, and 0% to 29% in home care.[5] The numbers should be interpreted cautiously, however, because of discrepancies in methodology. The most accurate current prevalence rates in the acute care setting come from 3 multisite studies reported in 2000 and 2001: 14.8%, 15%, and 15%.[6-8]

FINANCIAL COST OF CARE

Cost is another relative unknown, although again, it is generally accepted to be high. Analyzing data from previously published studies, Beckrich and Aronovitch[9] concluded that 1.6 million pressure ulcers develop in hospitals in the United States each year, with a cost of $2.2 to $3.6 billion. They estimated an incremental cost of $125 to $200 for managing each Stage I or Stage II

Table 1.

NPWT CONSENSUS GUIDELINE PANEL MEMBERS

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pressure ulcer that develops in the hospital; for each Stage III or Stage IV pressure ulcer, they estimated a cost of $14,000 to $23,000.[9] However, higher-stage pressure ulcers rarely heal in the hospital setting, which means these numbers most likely underestimate the true cost to heal Stage III and Stage IV pressure ulcers.[10]

Pompeo[10] examined the relationship between wound burden—defined as a combination of pressure ulcer stage, wound size, and number of wounds—and the cost of care in a long-term acute care facility. He found, not surprisingly, that the higher the wound burden, the higher the cost of care; that is, patients with larger, higher-stage wounds and multiple wounds required more products (wound care supplies, nutritional products, and specialty beds) and more nursing care time.[10] Care for patients with the highest wound burden exceeded $50,000, including longer lengths of stay and higher total daily costs of care.[10]

HUMAN COST OF CARE

The emotional and physical cost to patients and their family caregivers has been explored in studies by Langemo et al[11] and Baharestani.[12] In a descriptive, qualitative, phenomenological study of the lived experience of having a pressure ulcer, Langemo et al[11] interviewed 8 patients who had either a current pressure ulcer or a history of pressure ulcers. Pressure ulcers were found to have a profound impact on the participants' lives, including physical, social, and financial status; change of body image; and loss of independence and control.[11] Those with a Stage IV pressure ulcer and a flap repair and/or those with a spinal cord injury inevitably experienced the grieving process in some form.[11] The researchers observed that individuals who developed a pressure ulcer had to learn a great deal about self-care and prevention of future ulcers, including why healing is often a lengthy process and why nutrition and hydration are vitally important.[11]

Baharestani[12] examined the lived experience of 6 women caring for their frail, elderly husbands who had Stage III or Stage IV pressure ulcers. The women, age 69 or older, had been providing care for their husbands (age 73 or older) at home for 2 to 10 years. In the study,[12] Baharestani described the ongoing struggles experienced by the women in trying to care for their totally dependent husbands, despite grossly limited financial resources, physical abilities, knowledge sources, and support systems. All 6 women had arthritis, plus varying medical problems.[12] They expressed fatigue and pain related to the difficulty in turning, toileting, and transferring their husbands from the bed to the chair, and they described their sorrow at seeing their husbands bedridden and becoming more debilitated.[12] Yet they believed that what they were doing for their husbands was important; none questioned being the primary caregiver.[12] None of the women had any experience in caring for a pressure ulcer, and all lacked even the most basic knowledge of frequent turning, offloading, and moist wound healing. Most of the women learned the hard way, when their husbands were hospitalized for sepsis related to the pressure ulcer or another concomitant condition.[12]

ETIOLOGY OF PRESSURE ULCERS

Pressure ulcers are thought to develop over bony prominences as a result of excessive pressure. This pressure causes ischemia and subsequent necrosis, eventually leading to tissue ulceration. The primary causative factor is pressure. Maklebust defined pressure as "a perpendicular load or force exerted on a unit of area."[13] The amount and the duration of pressure are inversely proportional.[14] Low amounts of pressure over long periods can be just as detrimental to tissue as high amounts of pressure over short periods. The long-held standard of 32 mm Hg as a critical closing pressure is being revisited. Continued research is needed in the areas of capillary perfusion pressure and application of uniform and localized pressure.[15]

Body tissues differ in their ability to tolerate pressure. The skin's blood supply originates in the underlying muscle, which is more sensitive to pressure damage than skin.[16] Tissue tolerance is further compromised by extrinsic and intrinsic factors. Examples of intrinsic factors are moisture, friction, and irritants.[15] Numerous intrinsic factors affect the ability of the skin and supporting structures to respond to pressure and shear forces. Age, spinal cord injury, nutrition, and steroid use are among intrinsic factors believed to affect collagen synthesis and degradation.[17] Other intrinsic factors that affect tissue perfusion are systemic blood pressure, extracorporeal circulation, serum protein, hemoglobin and hematocrit, vascular disease, diabetes mellitus, vasoactive drugs, increased body temperature, and smoking.[17]

How pressure ulcers occur is unclear. One theory holds that they begin from the bone and move outward. Deep tissue injury near the bone occurs first, and it isn't until later, when tissue death continues and reaches the outer layer of the skin (the epidermis), that the skin breaks.[18] The varying critical ischemia time of different tissues supports this theory.

The pressure gradient model has been used to explain how pressure translates into tissue death. External pressure is transmitted from the body surface to the underlying bone, compress-

CLINICAL PEARL #1:

WOUNDS NOT PROGRESSING

To be effective, negative pressure wound therapy should be used at least 22 of 24 hours a day. If a patient's wound is not progressing, take a look at the pump, which keeps a log of how long it has actually been on. Patients have been known to turn off the pump while at home, then turn it back on when returning to see their health care provider. In addition, staff in other departments may turn off the pump for treatment, then neglect to turn it back on when the treatment is done.

ing all of the tissue in between. The greatest pressure occurs over the bone, gradually decreasing to the skin level. Blood vessels, fascia and muscle, subcutaneous fat, and skin are compressed between these 2 counterpressures.[15]

Muscle and subcutaneous fat have a low tolerance for decreased blood flow, making them less resilient to pressure than the skin. Destruction of tissue below skin level is not seen until surface damage is evident.[14] Unless the nervous system is impaired, resulting in loss of sensation, patients normally shift their weight by changing their position when pressure is exerted against the skin for a period of time.[15]

According to the second theory of pressure ulcer formation, pressure ulcers result from skin destruction that occurs at the epidermis and proceeds downward to the deeper tissue. Maklebust and Sieggreen call this theory the "top-to-bottom model."[18] The injury is seen as intact skin with blanchable erythema. This is the less-favored model of pressure ulcer development, given its limited evidence base.[15]

Friction and shear are mechanical forces contributing to pressure ulcer formation. The tissue injury resulting from these forces may look like a superficial skin insult. Although 2 separate phenomena, shear and friction often work together to create tissue ischemia and ulcer development.[15]

Shear is a "mechanical force that acts on an area of skin in a direction parallel to the body's surface. Shear is affected by the amount of pressure exerted, the coefficient of friction between the contacting materials, and the extent to which the body makes contact with the support surface."[4] This is akin to pulling the bones of the pelvis in one direction and the skin in the opposite direction. The deeper fascia slides downward with the bone; the superficial fascia remains attached to the dermis. This injury and the compromise to the blood supply create ischemia and lead to cellular death and tissue necrosis. Shear and friction go hand in hand—one is rarely seen without the other.[15]

Shear injury is not seen at the skin level because it occurs beneath the skin. Elevating the head of the bed increases shear injury in the deep tissue and may account for the large number of sacral ulcers seen in practice.[15]

Unlike shear injury, friction injury is visible. Friction is the "mechanical force exerted when skin is dragged across a coarse surface, such as bed linens."[4] Simply stated, it is 2 surfaces moving across each other. A skin injury caused by friction looks like an abrasion or superficial laceration. Friction can contribute to an injury or stripping of the epidermal layer of the skin, creating an environment conducive to further injury.[15]

An alteration in the coefficient of friction increases the skin's adherence to the outside surface (the bed). Friction then combines with shearing forces, and the ultimate outcome may be a pressure ulcer.[15] Tissue subjected to friction is more susceptible to pressure ulcer damage.[19] These three mechanical forces—pressure, friction, and shear—may act in concert to create tissue damage. Patients at risk for pressure ulcers from friction are older adults, those with uncontrollable movements (eg, spasticity), and those who use braces or appliances that may rub against the skin.[18]

Theories on the etiology of a pressure ulcer need continued research. Those described here may be correct; however, additional research and basic science hold the key to many unanswered questions.[15]

STAGING AND TREATING PRESSURE ULCERS

Once a pressure ulcer has been observed, it is typically staged to reflect the layers of tissue involved. The generally accepted staging system for pressure ulcers was developed by the NPUAP in 1989 and adopted by the panel developing the pressure ulcer treatment guideline for the AHCPR in 1994.[4] In 1998, the NPUAP revised the definition of Stage I pressure ulcers.[20,21] Pressure ulcers are currently staged as follows:

Stage I

An observable pressure-related alteration of intact skin whose indicators, as compared to the adjacent or opposite area on the body, may include changes in 1 or more of the following: skin temperature (warmth or coolness), tissue consistency (firm or boggy feel), and/or sensation (pain, itching). The ulcer appears as a defined area of persistent redness in lightly pigmented skin, whereas in darker skin tones, the ulcer may appear with persistent red, blue, or purple hues.

Stage II

Partial-thickness loss of skin, involving the epidermis, dermis, or both. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage III

Full-thickness skin loss, involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.

Stage IV

Full-thickness skin loss with extensive destruction; tissue necrosis; or damage to muscle, bone, or supporting structures (eg, tendon, joint, or capsule). Undermining and sinus tracts also may be associated with Stage IV pressure ulcers.

Treatment of pressure ulcers involves a strategy that combines the following:

- wound cleansing and debridement to remove devitalized tissue and reduce bacterial burden
- appropriate support surfaces to redistribute pressure
- attention to the patient's nutritional status
- dressings that promote a moist wound healing environment
- appropriate topical, oral, and/or parenteral antibiotic therapy
- use of adjunctive modalities.[15]

During the past decade, technologic advances have con-

tributed to an explosion of products used to manage pressure ulcers. One such treatment modality is NPWT, known as the Vacuum-Assisted Closure® (V.A.C.®) Therapy System (KCI USA, Inc., San Antonio, TX), which is considered to be an adjunctive therapy. NPWT was developed in the 1990s at Wake Forest University, Winston-Salem, NC, by a team led by plastic surgeon Louis Argenta, MD.

The Food and Drug Administration (FDA) has cleared the V.A.C.® Therapy System as a device that helps promote wound healing, through means including drainage and removal of infectious materials or other fluids, under the influence of continuous and/or intermittent negative pressure. The device is indicated for patients with chronic, acute, traumatic, subacute, and dehisced wounds; partial-thickness burns; ulcers (such as diabetic or pressure); flaps; and grafts (Table 2).

Table 2.
GENERAL INDICATIONS FOR NPWT

- Chronic wounds
- Acute wounds
- Traumatic wounds
- Partial-thickness burns
- Dehisced wounds
- Diabetic ulcers
- Pressure ulcers
- Flaps
- Grafts

NPWT: MECHANISMS OF ACTION

The concept of NPWT is based on the principles of physics. The application of controlled subatmospheric pressure causes mechanical stress to tissue and the wound is drawn closed.[22] The degree of pressure to the wounded tissue is small. However, 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 closure and resolution.[23]

Moist wound healing

NPWT applies subatmospheric pressure to the wound bed via a computerized therapy unit attached to an open-cell (reticulated) foam dressing placed in the wound and secured with an adhesive drape. The adhesive drape helps provide a semiocclusive environment that supports moist wound healing, which is the standard for wound care. The vapor-permeable drape facilitates gas exchange, an important consideration when treating wounds infected with anaerobic organisms that would thrive in an occlusive, oxygen-depleted environment.[23] The foam dressing and drape also protect the wound base from environmental contaminants and other bodily fluids and reduce the risk of friction or shear, enhancing the body's ability to heal.[23]

Peripheral edema and circulation

The tissue surrounding a wound is typically characterized by a localized buildup of interstitial (third-space) fluid.[24] 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.[25]

With NPWT, wound fluids are evacuated via a tubing system placed on top of or inside the foam dressing 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.[23] The laser Doppler flow study by Morykwas et al[26] suggests a significant increase in blood flow adjacent to a wound receiving negative pressure, likely 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).[27] These microorganisms consume the nutrients and oxygen that would otherwise be directed toward tissue repair. In addition, they release enzymes that break down protein, 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.[23] As mentioned earlier, circulation is enhanced when interstitial fluid is removed. Any increase in circulation and oxygenation to compromised tissue improves the area's resistance to infection, allowing healing to progress.[24,26,28] In addition, increased blood flow translates to increased delivery of infection-fighting leukocytes.

Granulation tissue

Granulation tissue is a mix of small blood vessels and connective tissue in the wound base. 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.[23] The research by Morykwas et al[26] suggests that granulation tissue formation is enhanced by negative pressure by virtue of interstitial fluid resolution and the resulting increase in circulation.

The science behind NPWT is significant because it enhances the body's reparative mechanisms to promote wound healing.[23] NPWT does not often replace surgical procedures, but may allow a wound to progress to the point that a less-invasive procedure is possible.

SEMINAL PAPERS ON NPWT

A closer look at the seminal papers on NPWT is warranted. Morykwas et al[26] described the scientific basis for subatmospheric pressure in their paper detailing 4 animal studies. Using pig models, they examined the effects of this therapeutic modality on laser Doppler-measured blood flow in the wound and adjacent tissue, rate of granulation tissue formation, clearance of bacteria from infected wounds, and flap survival.

Escalating levels of negative pressure (continuous and intermittent) were applied to assess blood flow in the subcutaneous tissue and muscle around the wound. Morykwas et al[26] found that 125 mm Hg was the optimal level. This pressure setting helped increase the blood flow level by 4 times the baseline. Interestingly, higher pressures—400 mm Hg and above—*inhibited* blood flow.

In the same study,[26] the investigators examined granulation tissue formation by comparing wounds treated with wet-to-moist dressings with those treated with NPWT (either continuous or intermittent suction) plus wet-to-moist dressings. Wounds in the continuous suction group formed 63.3% more granulation tissue over the same period as wounds treated only with wet-to-moist dressings (control group).[26] Those in the intermittent suction group experienced a 103.1% increase in granulation tissue compared with wounds in the control group.[26] Both results were statistically significant.

The wounds were then inoculated with either *Staphylococcus epidermidis* or *S. aureus*. Bacterial levels were initially 10^8 organisms per gram of tissue. No antibiotics were used to treat infection. For the first 3½ days, researchers saw no differences in healing.[26] After that, bacterial levels in wounds treated with NPWT dropped 4-fold (10^4 in the NPWT-treated group, compared with 10^7 to 10^8 in the control group).[26]

Two recent studies have called into question NPWT's role in removing bacteria.[29,30] In a retrospective analysis of 25 patient charts, Weed et al[29] found no consistent effect on reducing the bacterial burden with NPWT; in some cases, bacterial colonization increased. However, this finding did not seem to affect the therapeutic benefits of NPWT. The findings of the Weed et al[29] study may have been affected by the fact that the NPWT dressings for the study patients were changed every 3 to 5 days, not every 12 to 24 hours as recommended in the manufacturer's protocol for infected wounds.[31]

Mouës et al[30] conducted a randomized, controlled trial of 54 patients to study the effect of NPWT on bacterial load. In this trial, the amount of non-fermatative Gram-negative bacilli in wounds treated with NPWT significantly decreased, but *S. aureus* significantly increased.[30] Again, the bacterial burden did not affect the benefit of NPWT on healing. There was a significantly larger reduction in wound surface ($P < .05$) in wounds treated by NPWT when compared with those treated with conventional moist gauze therapy.[30]

Finally, Morykwas et al[26] examined skin flap survival. In the control group (no NPWT), 51.2% of the random flaps survived.[26] In the first of 3 NPWT-treated groups, subjects received NPWT for a period before the flap was elevated; flap survival was 64.8% in this group.[26] A second NPWT-treated group was treated postoperatively with NPWT: The flap was attached on a wound, then NPWT was administered on top of the flap. In this group, flap survival was 67.4%.[26] These results were statistically significant compared with the control group. The best flap survival rates were found in the third NPWT-treated group. Their wounds were pretreated *and* posttreated (NPWT before and after flap elevation), resulting in a 72.2% flap survival rate.[26] This was statistically significant versus either NPWT-treated group and versus the control group.

CLINICAL PEARL #2:

PRESSURE POINTS

The tubing that runs from the foam dressing to the negative pressure wound therapy (NPWT) unit can be a source of pressure ulcers if the clinician is not mindful of where the tubing is positioned. Keep tubing away from bony prominences and creases in the tissue, and monitor patients carefully to ensure that the tubing does not end up in the wrong place when patients are repositioned.

In the same journal issue, Argenta and Morykwas[24] reported on the use of NPWT in humans. They included 300 wounds in their study: 175 chronic wounds, 94 subacute wounds, and 31 acute wounds. NPWT was applied at a subatmospheric pressure of 125 mm Hg (the same pressure found to be most effective in increasing blood flow in the animal studies[26]) and was continued until the wounds were completely closed; could be covered with a split-thickness graft; or had enough healthy, granulating tissue for a flap procedure. In all, 296 wounds responded favorably. This research suggests that NPWT decreases chronic edema by removing interstitial fluid, which may lead to increased localized blood flow, and that the applied forces of NPWT may stimulate granulation tissue formation.[24,26]

NPWT AND WOUND BED PREPARATION

Like other chronic wounds, pressure ulcers fail to heal in a timely manner. Research into why this occurs is now focusing on how the biochemical components of wound healing differ in acute and chronic wounds.[32] There is growing support—based on analyses of wound fluids—for the concept that a chronic wound is "stuck" in the inflammatory phase of healing.[32] Mitogenic cellular activity reportedly decreases in chronic wound fluid, whereas acute wound fluid promotes DNA synthesis.[33-35] For example, DNA synthesis was stimulated when researchers added wound fluids from acute mastectomy wounds to normal skin cell cultures; wound fluids from chronic leg ulcers did not stimulate

DNA synthesis.[36] In addition, adding chronic wound fluid to acute wound fluid has inhibited mitotic activity.[33-36]

Researchers are also looking at cytokines found in wound fluid. One study found lower levels of proinflammatory cytokines (tumor necrosis factor-alpha [TNF-α] and interleukin 1-beta [IL-1β]) in fluid from acute mastectomy wounds than in chronic wound fluid.[36] Harris et al[35] supports this finding: Higher levels of cytokines were found in nonhealing ulcers. Interestingly, cytokine levels decrease when a chronic wound begins to heal.[36]

Chronic wounds have also been shown to have higher levels of matrix metalloproteinase (MMP) than acute wounds.[37-43] This is important because higher levels of MMP and neutrophil elastase activity degrade proteins and the exogenous growth factors needed for wound healing.[44,45] As a result, wound healing may be impaired.

In addition, researchers are interested in the biologic response of cells in chronic wounds. For example, fibroblasts in healing wounds are able to respond to growth factors and divide. This may not be the case with fibroblasts in chronic wounds.[36] The term *senescent* describes cells that have a diminished response to these molecular regulators.

Research into the differences in biologic processes between acute and chronic wounds has resulted in a shift in thinking. Investigators are now talking about wound bed preparation as the key to healing a chronic wound such as a pressure ulcer.[46] This paradigm encompasses a process of removing various burdens that impede healing, including exudate, bacteria, and necrotic/cellular debris.[47] NPWT may be able to play an important role in wound bed preparation, based on the research findings of Morykwas et al,[26] by creating a moist wound healing environment that removes exudate without drying the wound.

NPWT AND PRESSURE ULCERS

Several recent studies have focused specifically on NPWT and its effect on pressure ulcers.[48-51] In addition, numerous case studies[52-55] give a clinical snapshot of this modality in select patients.

Isago et al[48] used NPWT with 10 patients who had Shea Stage IV pressure ulcers, defined as wounds penetrating into the deep fascia, with involvement of bone and muscle (similar to a Stage IV pressure ulcer in the NPUAP staging system[4]). Five patients had sacral lesions, 3 patients had femoral trochanteric pressure ulcers, and 2 patients had ischial pressure ulcers. All patients were paralyzed or bedridden.[48]

Before treatment, the researchers measured the length, width, and depth of the pressure ulcers; this was continued weekly after the initial measurement. The healing index of each pressure ulcer was calculated as initial area of lesion minus final area of lesion divided by time in days.[48]

NPWT was applied for 4 weeks (4 cases), 5 weeks (5 cases), and 7 weeks (1 case). The researchers found that after NPWT treatment, wound area had been reduced an average of 55%, with depth reduced by an average of 61%.[48] In 3 cases, the wounds were small enough (10 cm^2) that the patients were switched to normal saline dressings and the wounds were allowed to heal without operative closure. Wounds in the remaining 7 patients closed to the point that gluteus maximus and posterior thigh flaps could easily be performed for wound closure.[48]

The researchers reported that all patients had poor granulation tissue with edema at the start of treatment. Within 2 weeks of treatment, granulation tissue became firm and red in many cases.[48]

Ford et al[49] enrolled 28 patients with 41 full-thickness pressure ulcers for a minimum of 4 weeks' duration in a randomized trial of NPWT versus wound products marketed by Healthpoint. Twenty-two patients with 35 pressure ulcers completed the 6-week trial. These patients had 17 sacral, 9 ischial, 4 lateral malleolar, 4 calcaneal, and 1 trochanteric pressure ulcers.[49]

In an interim analysis of results, Ford et al[49] reported complete healing in 2 ulcers in the NPWT group and 2 in the Healthpoint group.[49] In each group, 6 wounds underwent flap surgery. Overall, pressure ulcer volume was reduced by a mean of 51.8% with NPWT and 42.1% with the Healthpoint products.[49] Mean reductions in length, width, and depth were 36.9 cm, 40 cm, and 33.6 cm, respectively, for NPWT, compared with 18.7 cm, 19 cm, and 31 cm, respectively, for the Healthpoint products.[49] Mean changes in polymorphonuclear neutrophils, leukocytes, and capillaries were significantly greater in the NPWT group than in the Healthpoint group.[49] In addition, 3 cases of osteomyelitis improved during NPWT treatment compared with no improved cases of osteomyelitis with Healthpoint products.[49]

Based on these findings, the researchers concluded that NPWT appears to be the superior treatment option for reducing inflam-

CLINICAL PEARL #3:

PAINFUL DRESSING CHANGES

Some patients receiving negative pressure wound therapy experience pain during dressing changes. Unless medically contraindicated, 1% lidocaine solution can be introduced down the tubing or injected into the foam dressing before a dressing change; the pump must be set no higher than 50 mm Hg. Clamp the tubing, then wait 15 to 20 minutes before removing the dressing. Similarly, 10 to 30 mL of normal saline solution can be instilled into the tubing and allowed to soak the dressing (the tubing must be unclamped) to permit easier removal of the dressing. Saline can also be injected directly into the foam dressing while 50 mm Hg of pressure is applied to the dressing (the tubing should be clamped as soon as the saline starts to flow into the dressing tubing). Either way, wait 15 to 30 minutes, then remove the dressing.

Table 3.
GENERAL CONTRAINDICATIONS FOR NPWT

- Malignancy in the wound
- Untreated osteomyelitis
- Nonenteric or unexplored fistulas
- Necrotic tissue with eschar present
- Placement over exposed blood vessels or organs

mation at the wound site, and that NPWT shows promise as an adjunctive treatment to systemic antibiotics for osteomyelitis.[49]

Deva et al[50] examined the role of NPWT in difficult-to-heal pressure ulcers in 30 patients in Australia. The patients had been referred to the researchers' tertiary plastic and reconstructive surgical service because their wounds were not considered candidates for reconstructive surgery.[50] All patients had Grade III pressure ulcers. They had been receiving treatment for a mean of 418 days before the plastic and reconstructive surgery referral.[50]

The researchers applied NPWT at pressures of 75 to 125 mm Hg in continuous mode for 48 hours, then switched to intermittent mode.[50] They had 3 main outcome measures: complete wound healing, obliteration of the wound cavity to allow for a surface dressing, and delayed primary wound closure or skin graft placement.[50]

Therapy with NPWT lasted for a mean of 35 days (range, 3 to 124 days), with success in 26 of 30 patients.[50] The researchers found that newer pressure ulcers (those less than 6 weeks old) healed more rapidly than older pressure ulcers.[50]

In a study from Switzerland,[51] 22 patients with pressure ulcers in the pelvic region were randomized to treatment with NPWT or traditional wet-to-dry/wet-to-wet dressings soaked with Ringer's solution. Patients were eligible if they had pressure ulcers deeper than Grade 2, meaning at least penetration into the subcutaneous fat.[51] All patients were paraplegic or tetraplegic.

Following treatment, volume was measured instead of area because the researchers wanted to measure the clinically important reduction in wound size resulting from the newly formed granulation tissue and wound contracture.[51] Their study end point was a 50% decrease in wound volume (all wounds were closed with a flap; therefore, wound closure was not a measured end point).[51]

They found that the time to reach the study end point was virtually identical in both groups: 27 days in the NPWT group and 28 days in the traditional therapy group.[51] The decrease in wound volume was also similar in the 2 groups.[51]

The researchers were surprised by this finding, which contradicted their clinical impression that NPWT allowed for faster wound healing[51] and differed from the findings of Morykwas et al.[26] They postulated that the type of wound they included in the study could have made the difference: They were studying pressure ulcers, not experimentally produced acute wounds.[26,51] Pressure ulcers are slower to regenerate than acute wounds, they said.[51]

Although figures were not cited, the researchers said that a preliminary analysis of costs in their hospital indicated that NPWT was less expensive than traditional dressings if NPWT was used for longer than 2 days.[51] However, no conclusions can be drawn from this.

CLINICAL PEARL #4:

CLEAN VS STERILE TECHNIQUE

The components of the dressing for negative pressure wound therapy (NPWT) are packaged sterile. The consensus panel members agreed that clean technique is acceptable when initiating NPWT or changing the dressing and/or other components. However, they cautioned clinicians to follow their facility's protocols regarding the use of clean vs sterile technique.

EARLY EXPERIENCE OUTSIDE THE UNITED STATES

The seminal studies by Argenta and Morykwas[24] and Morykwas et al[26] were not the first published discussions of the use of negative pressure in wound treatment. Several Russian studies by Zhivotaev[56] and Davydov et al,[57-63] dating back to 1970, describe a "vacuum therapy" for treating various wound types, including postoperative infected wounds of the urinary bladder,[56] suppurative lactational mastitis,[57] acute suppurative diseases of soft tissue and suppurative wounds,[58-61] postoperative wound infection,[62] and wounds in older patients (wound type not specified in the abstract).[63] Unfortunately, these papers are in Russian, with limited English abstracts, making it difficult for English-speaking clinicians to evaluate the study methodology, treatment technique, and treatment outcomes and compare them with papers available in English.

A series of German studies[64-70] describe a technique called vacuum sealing, which is similar to what is known as NPWT in the United States. Again, a language barrier exists. However, the English-language abstracts available on MEDLINE give a clearer indication of study methodology, treatment technique, and treatment outcomes than the Russian papers.

Fleischmann et al[64] were the first to report on vacuum sealing in the German literature. They used this technique to treat soft tissue damage in 15 open fractures and found it efficient for cleansing the wound and triggering proliferation of granulation tissue.[64] No bone infections occurred, although 1 patient developed a soft tissue infection from poor sealing technique that was resolved when the technique was corrected.[64]

Subsequent studies by Fleischmann et al[65-69] describe similar success with the technique in treating dermatofasciotomy of the lower extremity,[65] trauma defects,[66] and acute and chronic wound infections.[67] Two papers by Fleischmann et al,[68,69] pub-

lished in 1998, add another dimension to vacuum sealing: instillation of antiseptics or antibiotics to treat infection. Among the 27 patients, they found only 1 instance of recurrence of infection in a patient with chronic osteomyelitis in 3 to 14 months of follow up.[68,69]

Mullner et al[70] examined the vacuum sealing technique in patients with sacral pressure ulcers, traumatic soft tissue defects, and infected soft tissue defects following rigid stabilization of lower extremity fractures. They, too, found that the technique improved granulation tissue production. Initial wound dimensions decreased in 84% of wounds (38/45) when the vacuum sealing technique was used after irrigation and debridement.[70] Thirty-five wounds closed by granulation, secondary closure, or split-thickness skin grafting.[70]

However, all of the early non-US studies discussed above do not allow an apples-to-apples comparison with studies using the NPWT device currently available in the United States. As Thomas[71] points out, these studies achieved negative pressure by using wall suction devices or surgical vacuum bottles. Those devices can cause problems with appropriate delivery, control, and maintenance of negative pressure.[72] They may also fail to shut off in the event of heavy bleeding.

FDA-CLEARED NPWT DEVICE

An FDA-cleared NPWT device (V.A.C.® Therapy System) consists of a sterile, open-cell foam dressing; a computerized therapy unit that creates negative pressure; and a canister that collects the exudate drawn out of the wound (up to 300 mL; newer versions have larger, 500-mL and 1000-mL capacity canisters).

Tubing connects from the canister to a black or white foam dressing; the dressings are composed of slightly different materials with different porosity. The white polyvinyl alcohol (PVA) foam holds moisture and is hydrophilic (water attracting). The PVA foam has a higher tensile strength than the black polyurethane foam and can be used in tunnels and shallow undermining. The PVA foam is premoistened with sterile water and is nonadherent. The polyurethane foam does not hold moisture and is hydrophobic (water repelling). The polyurethane foam, less dense than PVA foam, is reticulated, which allows for increased distribution of subatmospheric pressure across the wound bed. The polyurethane foam can be used on all types of wounds. The foam dressings come in standard sizes, which are then cut to fit the wound; it is essential for the foam to be in contact with the wound cavity.

The foam dressings and tubing used with NPWT should not be connected to a wall-mounted suction unit. As mentioned earlier, this type of device lacks the control mechanisms needed to provide safe subatmospheric pressure for wound care.[23] Although it is true that these suction units have control panels, they cannot be adequately regulated to deliver the precise amount of subatmospheric pressure needed to provide the benefits of NPWT. The latest NPWT system, for example, features a proprietary pressure sensing technology that measures the pressure delivered to the wound site to help provide controlled wound healing.[73] It also features smart alarms to help ensure patient safety.[73] Wall-mounted suction devices do not have an alarm that sounds when an air leak occurs, which could result in wound bed desiccation. In addition, these devices do not have an alarm to indicate that the canister is full, which increases the risk for exsanguination.

Table 4.

ADDITIONAL RECOMMENDED GUIDELINES FOR NPWT SUCTION MODE

Wound characteristics	Continuous mode	Intermittent mode	Either mode
Difficult dressing application	X		
Flaps	X		
Highly exudating	X		
Meshed grafts	X		
Painful wounds	X		
Tunnels or undermining	X		
Unstable structures	X		
Minimally exudating			X
Large wound			X
Small wound			X
Stalled progress			X
White (PVA) foam			X

Source: V.A.C.® Therapy Clinical Guidelines, KCI USA, San Antonio, TX, October 2004.

When a wall-mounted suction unit is used, there is also a high risk of cross-contamination from the backflow seen in low-volume suction lines.[74,75] Using the same suction device on wound care patients and respiratory patients could provide a route for respiratory tract contamination in patients receiving ventilatory assistance with air-oxygen mixtures or suctioning procedures.[23]

In addition, the collection canisters on most wall-mounted suction units are not designed to provide a closed system. If wound fluid is allowed to stagnate, microorganisms may thrive and cause cross-contamination between patients.[23]

NPWT GUIDELINES

As a result of the consensus panel's discussion, a treatment algorithm (Figure 1) was developed to assist clinicians in making treatment decisions about NPWT use in pressure ulcer management. It should be noted that the very important issue of prevention of pressure ulcers was not evaluated by the consensus panel. The panel members answered the following key questions in developing the algorithm.

Key Questions about NPWT

1. What are the indications for NPWT in patients with pressure ulcers?

In addition to the general indications for NPWT (Table 2), the consensus panel members agreed that NPWT is best used with full-thickness skin defects (ie, Stage III and Stage IV pressure ulcers). The wound should be large enough for adequate contact between the foam dressing and the wound bed and for safe removal of the foam. NPWT can be used with either shallow or deep pressure ulcers. Depth is not the issue; rather, the clinician should consider NPWT if the wound has inadequate or poor granulation tissue and heavy exudate. NPWT can also be used with wounds that have undermining or tunneling.

2. What *wound* characteristics do *not* favor use of NPWT?

In addition to the general contraindications for NPWT (Table 3), the consensus panel agreed on the following wound characteristics that would contraindicate the use of NPWT:

- inadequately prepared wound beds, such as those that need to be debrided or that lack moisture
- wounds that are too small to allow the NPWT foam dressing to come into contact with the wound bed
- freshly debrided wounds without adequate hemostasis
- devitalized wounds with eschar
- wounds with inadequate circulation
- fibrotic wounds
- desiccated wounds.

3. What *patient* characteristics do *not* favor use of NPWT?

Certain precautions should be taken in selecting patients with pressure ulcers who are appropriate candidates for NPWT. The consensus panel agreed that NPWT should not be used with the following patients:

- patients who are unable to adhere to the treatment protocol, such as those who will not consistently offload pressure and those who lack adequate financial or caregiver resources
- patients with untreated malnutrition
- patients who cannot tolerate pain that may be caused by NPWT treatment, even after adjustments have been made (see Question 8)
- patients who have an allergy or tissue intolerance to the adhesive in the drape used to seal the foam dressing
- patients who have conditions that make it impossible to achieve a seal, such as patients with uncontainable incontinence, hyperhidrosis, or certain anatomic characteristics (eg, creases or folds in body tissue)
- patients with bleeding disorders that manifest at the wound level (eg, platelet dysfunction).

The consensus panel members believe that NPWT can be used in palliative wound care if the treatment goals are to relieve pain, manage exudate, and improve hygiene of bedridden patients.

4. What wound size is most appropriate for NPWT use?

The larger the wound, the more beneficial NPWT is compared with other treatments, according to the consensus panel. If the wound meets other criteria, as listed in Question 1, NPWT can still be an appropriate treatment for a smaller wound. The clinician would need to assess cost versus benefit of treatment.

5. How often should the wound be monitored?

The consensus panel agreed with the Wound, Ostomy and Continence Nurses (WOCN) Society[76] recommendation that pressure ulcers be monitored at every dressing change. In the panel members' experience, dressing changes can be extended up to 72 hours (3 times a week) in select wounds. The manufacturer, however, recommends that dressings be changed every 48 hours, or every 12 to 24 hours in the presence of infection.[31] Panel members said that clinicians should consider changing dressings more often if the patient has a heavy bioburden (ie, a grossly infected wound with obvious cellulitis or necrotizing fasciitis). Dressing change intervals are ultimately the clinician's choice.

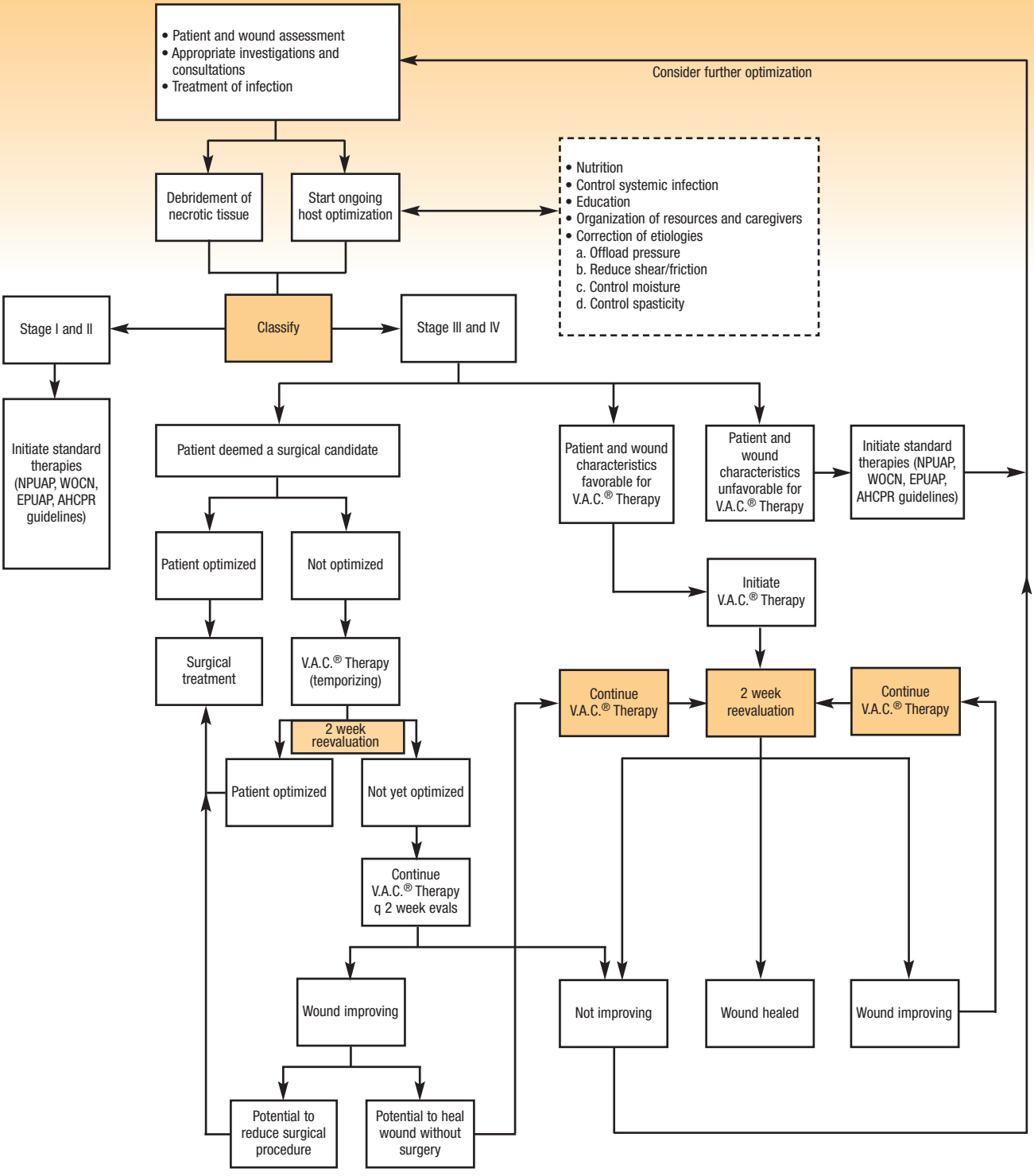
CLINICAL PEARL #5:

BRIDGING

If a patient has 2 pressure ulcers that are close together, they can be treated with the same negative pressure wound therapy unit by way of a technique called bridging. First, place a piece of the adhesive drape or another skin barrier between the 2 wounds to cover and protect the intact skin. Next, fill both wounds with the foam dressing, then connect them with another piece of foam (hence the term "bridging"). The foam pieces must be in contact with each other to ensure that negative pressure is distributed throughout the bridged dressing. Finally, make sure the tubing is centrally located between the wounds to avoid drawing exudate from one wound across to the other wound.

Figure 1.

MANAGEMENT OF A PATIENT WITH A PRESSURE ULCER USING V.A.C.® THERAPY



6. What are the optimal settings for NPWT use in pressure ulcers?

The target pressure setting for pressure ulcers is 125 mm Hg with the black foam dressing and 125 to 175 mm Hg with the white foam dressing. The recommended setting of 125 mm Hg is based on the findings of Morykwas et al.[26] However, this may not be the best starting pressure setting for all patients. In the case of patient pain, the manufacturer recommends reducing the target setting in 25 mm Hg increments, to a minimum of 75 mm Hg, until pain is relieved.[31] If the patient is older, emaciated, or taking an anticoagulant such as warfarin (Coumadin), the clinician should start at a lower pressure (eg, 75 to 100 mm Hg) and titrate up to 125 mm Hg as tolerated.[31]

Two papers by Isago et al[48,77] suggest that lower pressures can be used effectively. In their study of 10 patients with Shea Stage IV pressure ulcers, Isago et al[48] reduced the NPWT pressure to relieve pain in 3 of the 10 patients. Nevertheless, the study result showed a significantly decreased wound mean area and depth. In a later rat model, Isago et al[77] tested varying degrees of subatmospheric pressure on reduction of wounds. Although improvement was seen with lower settings and the data suggested the greatest efficacy at settings above 100 mm Hg,[77] clinical translation of this data is difficult as dressing changes were performed only once per week.

Previous work has shown that considerable tissue ingrowth into the dressing can occur in 3 to 4 days.[78] The ability to resolve differences between the pressure settings in this study may be decreased due to a reduction of the biomechanical stimulation at the wound surface by the ingrown tissue.

The panel noted that the initial well designed study by Morykwas et al[26] was a larger series that also employed laser Doppler flowmetry to evaluate perfusion changes and healing. There was a peak in a bell-shaped curve of perfusion and healing at 125 mm Hg in that study.[26] It is clear that wound closure is important to clinicians and patients. Pain tolerance should guide the selected pressure setting, with settings increased from 75 mm Hg by 25 mm Hg increments if pain is not an issue or reduced from 125 mm Hg by 25 mm Hg increments if pain is significant.

The consensus panel agreed that a one-size-fits-all approach to the pressure setting is inappropriate; it should be tailored to the individual patient's needs, within evidence-based parameters.

They also agreed that continuous suction mode should be used for the first 48 hours of treatment, then switched to intermittent suction mode for the remainder of therapy (5 minutes on, 2 minutes off).[26,31] Certain patient or wound situations, however, may require the use of continuous mode for longer than 48 hours or even for the duration of therapy (Table 4) for patients who have:

- significant discomfort when the intermittent mode is used
- anatomic issues (eg, creases or folds in the skin) or wounds in difficult areas (eg, perineal or toe wounds) that make it difficult to maintain an airtight seal
- wounds with tunnels or undermined areas because continuous suction helps hold the wound closed
- wounds with heavy drainage after 48 hours; intermittent suction should be delayed until the drainage tapers off.

7. Which foam dressing should be used?

The black foam (polyurethane) dressing is appropriate for stimulating granulation tissue while assisting in wound contraction. The white foam (PVA) dressing is more appropriate for wound areas that are ready for epithelialization, for protection of struc-

Table 5.
RECOMMENDED GUIDELINES FOR NPWT FOAM USE

	Black (polyurethane) foam	White (PVA) foam	Either type of foam
Deep, acute wounds with moderate granulation tissue present	X		
Deep pressure ulcers	X		
Flaps	X		
Extremely painful wounds		X	
Superficial wounds		X	
Tunneling, sinus tracts, or undermining		X	
Deep trauma wounds			X
Wounds that require controlled growth of granulation tissue		X	
Diabetic ulcers			X
Dry wounds			X
Post-graft placement (including bioengineered skin)			X
Shallow chronic ulcers			X

Source: V.A.C.® Therapy Clinical Guidelines, KCI USA, San Antonio, TX, October 2004.

tures, for control of granulation tissue growth into the foam, for tunneled and undermined areas, and for patients who cannot tolerate the black foam due to pain. Table 5 lists the manufacturer's recommended guidelines for foam use.[31] The consensus panel agrees with these guidelines.

8. What should be done if the patient is experiencing pain?

As described in Isago et al,[48] the pressure setting can be adjusted downward to reduce pain. Other strategies endorsed by the consensus panel include:

- switching from the black foam dressing to the white foam dressing
- changing from intermittent to continuous suction mode
- using a skin protection product around the dermal wound margins
- using a nonadherent meshed interface between the wound and the foam dressing
- allowing the patient to assist with dressing changes
- appropriately administering topical anesthetics or systemic analgesics
- moistening the foam dressing before removal.

The consensus panel members also suggested reassessing the frequency of dressing changes to determine if a longer interval between changes is possible, taking into account the manufacturer's recommendations.[31]

9. Does osteomyelitis affect NPWT use?

Untreated osteomyelitis is a contraindication for the use of NPWT. However, it is the consensus panel members' experience that NPWT is appropriate when treatment for osteomyelitis has been initiated.

This is supported by Ford et al.[49] In an interim analysis that compared NPWT with the Healthpoint wound gel products, they found that 3 cases of osteomyelitis improved during NPWT treatment, compared with no improved cases of osteomyelitis with Healthpoint products.[49]

10. What are the treatment end points?

The end points of treatment with NPWT depend on whether the patient is a surgical candidate, according to consensus panel members. If a flap procedure is planned, NPWT may be used to temporize the patient prior to surgery. This allows the clinician to address malnutrition, administer appropriate antibiotics, and stabilize coagulopathy. In the panel members' experience, use of NPWT can allow wound improvement to the point that surgery is not needed or a lesser surgical procedure than originally planned can be performed. If the wound does not progress or deteriorates, the patient will require surgical reconstruction of the pressure ulcer or use of another adjunctive modality. NPWT can also be used to help promote healing of flaps.

If the pressure ulcer is to heal by secondary intention, NPWT can be used until the wound achieves a fully granulated surface, with elimination of tunnels and resolution of undermining. NPWT can also be used to decrease wound volume (depth and surface area) until the wound is relatively superficial and the clinician knows it will achieve stable reepithelialization, possibly with another product.

If the wound deteriorates or fails to progress in 2 to 4 weeks, the consensus panel members recommend that the clinician reassess and determine if NPWT therapy is appropriate. This is consistent with recommendations in the AHCPR pressure ulcer treatment guideline[4] and the WOCN pressure ulcer guideline.[76]

11. What is the duration of NPWT treatment?

The consensus panel members recommend the use of NPWT as long as the wound is progressing toward the above end points with no unfavorable wound or patient characteristics.

CLINICAL PEARL #6:

MAINTAINING A SEAL

It is crucial to maintain the seal over the foam dressing; otherwise negative pressure wound therapy will not be successful due to inadequate delivery of negative pressure. Advice on maintaining that seal includes:

- drying the periwound skin thoroughly after cleansing and before applying the dressing and drape. A skin prep or degreasing agent can help to prepare the skin for the drape.
- framing the wound with a skin barrier. This will improve the seal if the periwound tissue is delicate or in a convoluted area.
- using a thin foam dressing designed for use with NPWT in more shallow wounds or wounds near the perineal area.
- positioning the dressing tubing on flat surfaces, away from the perineal area, bony prominences, and pressure areas.
- securing or anchoring tubing with a piece of drape or tape several centimeters away from the dressing. This prevents it from pulling on the wound area, which can cause leaks.

FUTURE DIRECTIONS

A number of clinical trials, supported by KCI, are currently under way. The intention is to delineate various aspects of wound treatment with NPWT. Panel members believe that 2 aspects are particularly important.

The first aspect is the cost issue. Pressure ulcer treatment is known to be costly, although the exact costs have not been definitively demonstrated. What role can NPWT have in reducing those costs? A health economics audit of NPWT cited studies in diabetic foot ulcers that demonstrated lower costs when compared with saline-moistened gauze.[79] In McCallon et al,[80] wound area decreased an average of 28.4% in the NPWT group versus 9.5% in the control group. Philbeck et al[81] estimated the average annual cost for treating each of 100 diabetic foot ulcers to be $23,066 with NPWT and $27,899 with saline-moistened gauze. That study

assumed that at 20 weeks, wound healing would be higher in the NPWT-treated group (50%, compared with 31% of the control group).[81]

Page et al[82] compared outcomes in patients with open foot wounds with soft tissue defects who were treated with NPWT versus those treated with saline-moistened gauze. They found that risk of complications, subsequent foot surgeries, and hospital readmissions were reduced by 70% or more for patients treated with NPWT.[82] Page et al[82] also said that in their study, lengths of stay during readmissions tended to be shorter and wound cavity filling and wound healing tended to take less time with NPWT, although the differences were not statistically significant.

The panel members knew of only 1 study that evaluated the cost of using NPWT to treat pressure ulcers. Philbeck et al[83] reviewed the records of Medicare patients whose pressure ulcers were treated with NPWT after previous wound therapy had failed. They compare the rate of healing of 43 Stage III and Stage IV trochanter and trunk pressure ulcers with the rate published by Ferrell et al[84] in 1993. In Philbeck et al,[83] these pressure ulcers (which averaged 22 cm^2 in area and were treated with low-air-loss surfaces and NPWT) closed at an average daily rate of 0.23 cm^2. The pressure ulcers in Ferrell et al[84] (which averaged 43 cm^2 in area and were treated with low-air-loss surfaces and saline-soaked gauze) closed at a rate of 0.09 cm^2/day.

The study by Philbeck et al[83] showed that with these closure rates, the pressure ulcers treated with low-air-loss surfaces and NPWT for 97 days would cost $14,546. The pressure ulcers treated with low-air-loss surfaces and saline-soaked gauze would cost $23,465, the study showed.[83] Further research to determine the cost-effectiveness of NPWT in patients with pressure ulcers is being conducted.

The second aspect the panel members believe should be validated is the role of NPWT as an adjunctive therapy. To be effective, pressure ulcer management generally requires a multimodal approach. The current body of literature, coupled with anecdotal reports and clinical experience, suggests that NPWT can be an important part of Stage III and IV pressure ulcer care. Clinical trials in progress should provide a clearer idea of how NPWT fits into the treatment picture for pressure ulcers. ●

CLINICAL PEARL #7:

OTHER TREATMENT MODALITIES

In the opinion of the consensus guideline panel members, negative pressure wound therapy (NPWT) can be very effective when used in association with other treatment modalities. The panel collectively has had broad experience with using antimicrobials, profibroblastic agents, growth factors, enzymatic debriders, nonadherent layers, and skin substitutes and alternatives under the NPWT foam dressing to facilitate wound healing. They caution, however, that combination therapy needs to make sense for both the patient and the wound.

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