

Optimal use of negative pressure wound therapy in treating pressure ulcers

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ABSTRACT

Pressure ulcers (PrUs) are a challenging health concern for both the clinician and the patient. The exact incidence and prevalence of PrUs varies widely among specific clinical populations, from 0.4% to 38% in acute care, from 2.2% to 24% in long-term care and from 0% to 17% in home care. The economic impact of these wounds is impressive with an estimated cost of \$11 to \$17.2 billion annually in the USA. Guidelines from the National Pressure Ulcer Advisory Panel and European Pressure Ulcer Advisory Panel have provided recommendations for the prevention and treatment of PrUs. Negative pressure wound therapy with reticulated open cell foam (NPWT/ROCF; V.A.C.® Therapy, KCI USA, Inc. San Antonio, TX) has been successfully used for managing PrUs. This review combines expert opinion with scientific evidence to describe the use of NPWT/ROCF in patients with PrUs.

Key words: Negative pressure wound therapy • Pressure ulcer • Wound healing

PRESSURE ULCERS

A pressure ulcer (PrU) is a localised injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure or pressure in combination with shear and/or friction (1–4); more recent research has suggested that microclimate (i.e. moisture and temperature) may also contribute to PrU formation (5). Prolonged pressure causes ischaemia, which leads to tissue necrosis that typically first occurs in the tissue closest to the bone. Ischaemic cell death produces inflammation that results in blood clotting, platelet aggregation, immune complex formation and the accumulation of inflammatory cells. A

number of contributing or confounding factors are also associated with PrUs. Chairbound or bedridden patients are at increased risk for developing PrUs. Additional risk factors for PrU development include advanced age, impaired ability to reposition oneself, friction, decreased sensory perception, impaired nutrition and excessive exposure to moisture (i.e. incontinence, excessive perspiration and wound drainage), which would affect the microclimate environment (1).

PrUs are classified in stages according to the degree of tissue damage (Table 1) based on the guidelines from the National Pressure Ulcer Advisory Panel and European Pressure Ulcer Advisory Panel (1,6).

Treatment of PrUs centres on the following interventions: management of tissue load (i.e. pressure, friction and shearing), nutritional support, ulcer care, and management of bacterial colonisation and infection. Standard care for PrUs depends on the ulcer stage and usually includes pressure relief and skin protection to prevent progression of the ulcer to advanced stages, debridement of necrotic tissue in Stage

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Table 1 NPUAP/EPUAP pressure ulcer stages***Suspected deep tissue injury:**

Purple or maroon localised area of discoloured intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment.

Category/Stage I:

Intact skin with non-blanchable redness of a localised area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding area. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue.

Category/Stage II:

Partial-thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising.

Category/Stage III:

Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle is not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunnelling.

Category/Stage IV:

Full-thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunnelling. Stage IV ulcers can extend into muscle and/or supporting structures (e.g. fascia, tendon or joint capsule) making osteomyelitis possible.

Unstageable:

Full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, grey, green or brown) and/or eschar (tan, brown or black) in the wound bed. Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore stage, cannot be determined.

*Adapted from National Pressure Ulcer Advisory Panel (NPUAP) and European Pressure Advisory Panel (EPUAP) (1,6).

III and Stage IV ulcers, wound cleansing and dressings that promote a moist wound environment (7,8). In addition, advanced therapy options include hydrocolloids (9), alginates (10), hyperbaric oxygen therapy (11) and negative pressure wound therapy using reticulated open cell foam (NPWT/ROCF) (12,13).

USE OF NPWT/ROCF FOR TREATING PrUs

Approximately 40 articles have been published in the literature (based on a proprietary database with daily searches of PubMed and Google Scholar) demonstrating the successful use of NPWT/ROCF for PrUs with a specific focus on Stage III and Stage IV ulcers. The majority of those articles are comprised of retrospective studies or case series, but randomised controlled trials (RCTs) comparing NPWT/ROCF with other wound care treatments have also been published (14).

In the most recent RCT, Wild *et al.* (15) compared NPWT/ROCF to NPWT using Redon bottles (which served as the vacuum source for delivering negative pressure) for treatment of patients with Stage III or Stage IV PrUs. Primary outcomes were relative and

absolute proportion of granulation tissue formation, fibrin and necrosis that were measured using a computer program (Wound Healing Analysing Tool) (16) specifically developed to analyse these tissue types. Patients were randomly assigned to either NPWT/ROCF ($n = 5$) or the Redon drain group ($n = 5$) and followed for a mean of 8.5 days. Results showed a 54% increase in granulation tissue formation with NPWT/ROCF, while the Redon drain group showed a decrease ($P = 0.001$). The NPWT/ROCF group also showed a 27% reduction of fibrin tissue at the wound base compared to a 21.8% increase in the Redon drain group ($P = 0.035$). Necrosis was also reduced in the NPWT/ROCF group but did not reach significance. The authors concluded that NPWT/ROCF was 'more reliable, more efficient and more user-friendly compared with the use of Redon bottles' (15).

In other RCTs comparing NPWT/ROCF management of PrUs to other conventional treatments (e.g. wet-to-dry or wet-to-moist dressings), NPWT/ROCF demonstrated reductions in wound surface area (17), volume (18) and depth (18) as well as improvement in granulation tissue formation (18). More importantly, in a large retrospective

study, Schwien *et al.* (19) demonstrated that PrU patients managed with NPWT/ROCF had significantly fewer hospitalisations than those managed with moist wound healing modalities (35 versus 48% $P < 0.05$), and emergent care for wound-related problems was lower in the NPWT/ROCF group (0 versus 8%; $P = 0.01$). Baharestani *et al.* (20) showed that early NPWT/ROCF initiation for PrU patients was associated with a decrease in total length of home care services; further analysis showed that for each day that NPWT/ROCF initiation was delayed, approximately 1 day was added to the home care length of stay. Further studies are necessary to determine the cost effectiveness of NPWT/ROCF for the treatment of PrUs.

In addition, two consensus panels comprised of experienced wound care physicians and clinicians have convened in the past 10 years to specifically address the use of NPWT/ROCF (V.A.C.[®] Therapy, KCIUSA, Inc., San Antonio, TX) for treating PrUs. Published guidelines as well as an algorithm were developed based on these panels of experts (7,8). The use of an algorithm may provide guidance to clinicians when deciding how to appropriately integrate NPWT/ROCF into their wound treatment management of PrUs.

Wound bed preparation for PrUs

Similar to other chronic wounds, PrUs take longer time to heal than acute wounds. There is a growing support for the concept that chronic wounds stall in the inflammatory phase of healing. Mitogenic cellular activity reportedly decreases in chronic wound fluid, whereas acute wound fluid promotes DNA synthesis. Chronic wounds have also been shown to have higher levels of matrix metalloproteinase (MMP) than acute wounds, which is important because higher levels of MMP and neutrophil elastase activity degrade proteins and exogenous growth factors needed for wound healing (21). As a result, wound healing may be impaired (22).

Research into the differences in biologic processes between acute and chronic wounds has resulted in a shift in thinking. Investigators are now considering wound bed preparation to be the key to healing a chronic wound, such as a PrU (23). This paradigm encompasses a process of removing various burdens that impede healing, including exudate, bacteria and necrotic/cellular debris. NPWT/ROCF

may play an important role in wound bed preparation, because of its accepted mechanisms of action and creation of a moist wound healing environment that removes exudate without drying the wound.

NPWT/ROCF INITIATION CRITERIA AND TREATMENT GOALS

If the patient and wound bed have been optimised, underlying infection managed and wound adequately debrided (Table 2), NPWT/ROCF may be initiated as a first-line therapy for Stage III and Stage IV PrUs (1). NPWT/ROCF may be used to assist in the healing of deep PrUs by stimulating granulation tissue formation and drawing wound edges together (7,8). NPWT/ROCF also effectively manages exudate by draining it away from the wound and reducing oedema at the wound surface. NPWT/ROCF may also play an important role in increasing comfort (e.g. reduction in dressing changes and odour), which in turn may improve patient quality of life and simplify nursing management of these complex wounds. After surgical reconstruction, NPWT/ROCF may be used to manage small dehiscences as well as to improve perfusion of a marginally viable flap.

CONTRAINDICATIONS

There are no specific contraindications with respect to NPWT/ROCF use in PrU patients beyond those presented in the clinical guidelines (24). A comprehensive patient assessment should be performed to determine any factors that may inhibit healing so that they can be addressed during treatment. NPWT/ROCF should not be used as a substitute for debridement, and care should be taken to prevent trauma and/or pressure when placing NPWT/ROCF tubing, particularly over bony prominences (24).

NPWT/ROCF DISCONTINUATION CRITERIA

NPWT/ROCF may be used in the setting of PrUs with a goal of either preparing a wound for surgical closure with a flap or with the plan of complete secondary closure. NPWT/ROCF should be stopped when the PrU and patient have been optimised for surgery or when the

Table 2 Principles for optimising wound healing outcomes for pressure ulcers

Optimise the patient	<ul style="list-style-type: none"> ● Maintain nutritional support ● Control comorbidities such as diabetes, anaemia and pain ● Encourage smoking cessation ● Review and evaluate medications and their effect on wound status ● Manage patient stress ● Evaluate wound for malignancy ● Educate patient and family
Treat the wound pathology	<ul style="list-style-type: none"> ● Improve blood flow and tissue perfusion (e.g. revascularisation) ● Apply compression therapy for oedema and venous insufficiency in the absence of arterial disease ● Use offloading devices and other techniques to optimise: <ul style="list-style-type: none"> ○ Pressure redistribution ○ Positioning ○ Mobility ○ Reduction of friction/shear/pressure ○ Surgical interventions to correct physical deformities
Optimise the healing environment	<ul style="list-style-type: none"> ● Debride the wound ● Treat increased bacterial burden or deep infection <ul style="list-style-type: none"> ○ Osteomyelitis ○ Surrounding cellulitis ● Maintain moisture balance ● Wounds may have dynamic needs, so plan of care should be re-evaluated at frequent intervals

ulcer has healed sufficiently to transition to moist wound healing dressings. A significant reduction in both area and volume of the pressure sore assessed on a weekly basis is a clear continuation criterion (7,8). If less than 30% reduction in volume occurs over a 4-week period, NPWT/ROCF should be stopped and the patient reassessed for optimisation.

TECHNICAL PEARLS

Wound not progressing

Wound should be monitored at every dressing change and should be measured and documented weekly or per facility protocol. PrU treatment regimens should also be evaluated at 2-week intervals or per facility protocol (7,25,26). If the wound does not appear to be progressing, perform a detailed reassessment ensuring that both the patient and wound are still optimised (Table 2).

Pressure points

The tubing that connects the ROCF dressing to the NPWT unit can be the source of PrUs, if the clinician is not mindful of the tubing's position. Keep tubing away from bony prominences and

increases in the tissue, and monitor patients carefully to ensure that the tubing does not end up in a potentially damaging position when patients are repositioned. Also, if the wound is over a bony prominence or in an area where weight bearing may apply additional pressure to the underlying tissue, a pressure-relief surface can be used to optimise patient offloading.

Bridging

Two PrUs that are close together can be treated with the same NPWT unit by creating an ROCF 'bridge' to connect the two wound dressings. The foam pieces must be in contact with each other to ensure that negative pressure is distributed throughout the bridged dressings. An interface material should be placed under the ROCF bridge to protect the skin (24). Finally, make sure the tubing is centrally located between the wounds to avoid drawing exudate from one wound across to the other wound. A customised bridge dressing with built-in ROCF extension (V.A.C.[®] GranuFoam[™] Bridge Dressing, KCI USA Inc.) makes it possible to apply NPWT/ROCF to wounds in difficult anatomical locations (e.g.

sacral PrUs). The ROCF extension makes it possible to position the pressure-sensing pad and tubing on a flat space away from pressure areas.

Maintaining a seal

It is crucial to maintain the seal over the ROCF dressing; otherwise, NPWT will not be successful due to inadequate pressure delivery and will dry out the wound. Tips on maintaining a seal include:

- Dry the periwound skin thoroughly after cleansing and before applying the dressing and drape. A skin preparation or degreasing agent can help to prepare the skin for the drape.
- Frame the wound with a skin barrier. This will improve the seal if the periwound tissue is delicate or in a convoluted area.
- Use a thin foam dressing in shallow wounds or wounds near the perineal area.



Figure 1. Case study 1: (A) Full-thickness skin breakdown on the sacrum. (B) Enzymatic debridement dressings were applied twice daily. (C) Application of negative pressure wound therapy with reticulated open cell foam (NPWT/ROCF). (D) After 8 days of NPWT/ROCF. (E) After 34 days of NPWT/ROCF. (F) Complete wound closure 27 days after discharge.

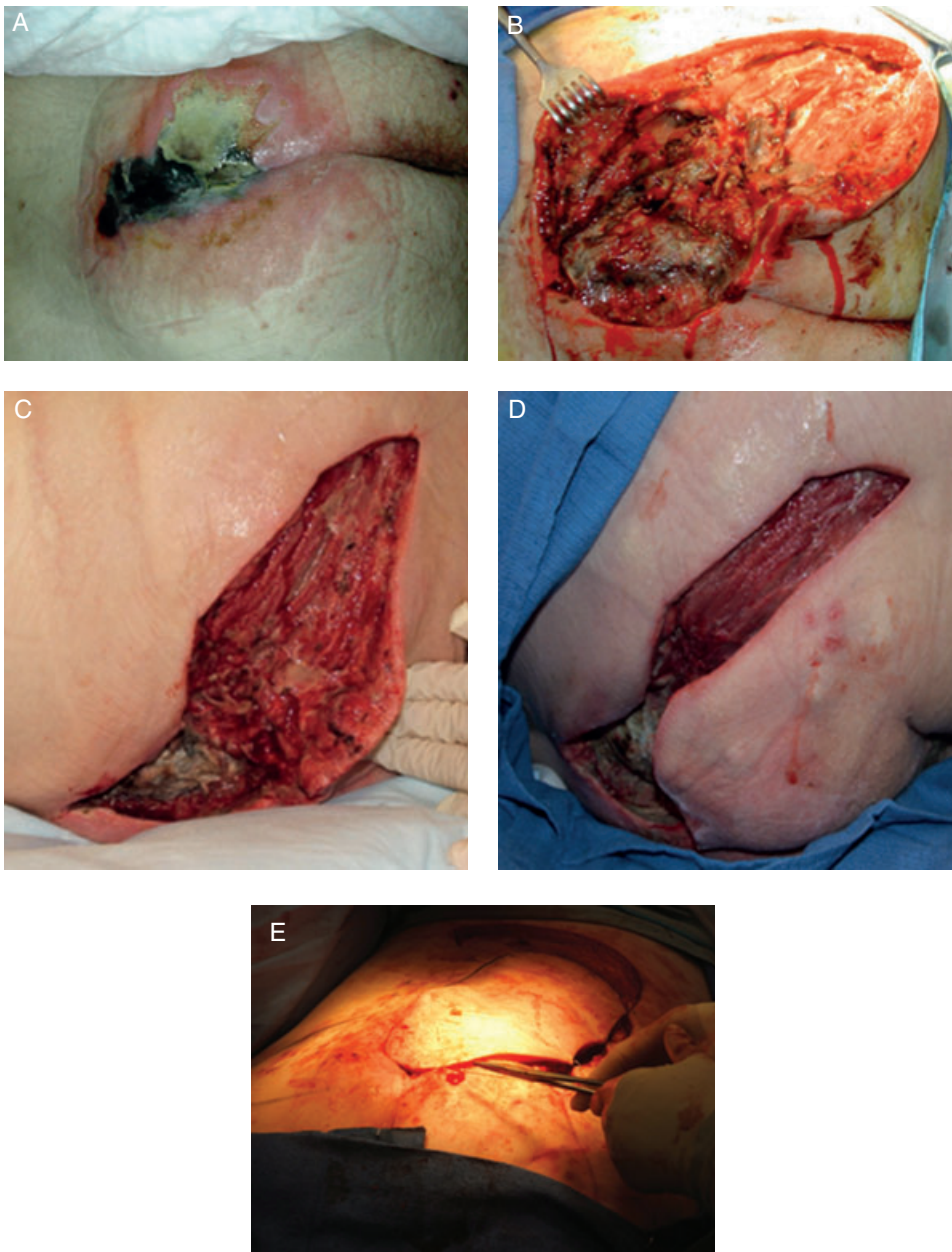


Figure 2. Case study 2: (A) Stage IV sacral ulcer. (B) Post-debridement. (C) After 16 days of negative pressure wound therapy with reticulated open cell foam (NPWT/ROCF). (D) After 28 days of NPWT/ROCF. (E) A fasciocutaneous flap was performed.

- Position the dressing tubing on flat surfaces, away from the perineal area, bony prominences and pressure areas.
- Secure or anchor tubing with a piece of drape or tape several centimetres away from the dressing. This prevents tubing from pulling on the wound area and causing leaks.
- Give particular attention to fluid flow and tubing positioning to allow for optimal flow.
- Avoid placement over bony prominences or within creases in the tissue.

CLINICAL CASES

Case study 1

This patient is a 78-year-old ambulatory woman who fell at home and fractured her humerus and tibia. She developed a full-thickness skin breakdown on the sacrum (Figure 1A). Several factors were addressed:



Figure 3. Case study 3: (A) Sacral pressure ulcer. (B) Wound presented with extensive undermining. (C) Application of negative pressure wound therapy with reticulated open cell foam (NPWT/ROCF). (D) After 3 weeks of NPWT/ROCF. (E) Application of split-thickness skin graft. (F) Successful graft.

fractures, surface and nutrition were optimised. Enzymatic debridement dressings were applied twice daily (Figure 1B). On admission day 6, NPWT/ROCF (-125 mm Hg continuously) was initiated after eschar was reduced (Figure 1C). NPWT/ROCF continued for 34 days with dressing changes three times a week (Figure 1D and E). On day 34, patient was discharged home with an alginate dressing, and wound achieved complete closure 27 days after discharge (Figure 1F).

Case study 2

This patient is a 74-year-old male with a history of diabetes, cardiomyopathy, hypertension and atrial fibrillation who presented 3-weeks post hip replacement. Patient developed a Stage IV sacral ulcer (Figure 2A). Wound was initially debrided (Figure 2B) followed by NPWT/ROCF (-125 mmHg continuously) (Figure 2C). Following 28 days of NPWT/ROCF with dressing changes three times a week (Figure 2D), a fasciocutaneous

flap was performed (Figure 2E), and patient was discharged on postoperative day 3. Patient was an outpatient for 3 of the 4 total weeks of treatment.

Case study 3

This patient is a 70-year-old woman with cerebral infarction suffered from a large sacral PrU (Figure 3A), which presented extensive undermining (Figure 3B). After surgical debridement, NPWT/ROCF was applied (Figure 3C). After 3 weeks of NPWT/ROCF, robust granulation tissue formation was present (Figure 3D). A split-thickness skin graft (STSG) was applied to healthy granulation tissue (Figure 3E), and the STSG adhered successfully (Figure 3F). The wound was resurfaced completely 1 month after STSG.

ECONOMIC VALUE AND FUTURE DIRECTIONS

An organised approach to managing wounds with NPWT/ROCF has been demonstrated to reduce length of hospitalisation and facilitate transition to a home care setting (27). Similar efforts using an algorithmic approach to managing PrUs with NPWT/ROCF have shown equally positive outcomes (28). There is little doubt that NPWT/ROCF can have a positive impact on the quality of life of a PrU patient. To rationalise the seemingly high cost of NPWT/ROCF in our current model of healthcare delivery, more powerful economic arguments for its use are needed. To measure economic benefits of NPWT/ROCF, future cost-effectiveness studies should focus on factors outside of direct therapy costs, such as measures of reduction in complexity and number of surgical procedures/adverse events, reduced nursing time for wound care with less frequent dressings, and a reduction in length of treatment and hospital stay.

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CONFLICTS OF INTEREST

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REFERENCES

- 1 National Pressure Ulcer Advisory Panel and European Pressure Ulcer Advisory Panel. Prevention and treatment of pressure ulcers: clinical practice guideline. Washington, DC: National Pressure Ulcer Advisory Panel, 2009.
- 2 Lyder CH. Pressure ulcer prevention and management. *JAMA* 2003;289:223–6.
- 3 Reddy M, Gill SS, Rochon PA. Preventing pressure ulcers: a systematic review. *JAMA* 2006;296:974–84.
- 4 Bryant R, Nix D. Acute and chronic wounds: current management concepts. 4th edn. St. Louis, MO: Mosby, 2012.
- 5 Baharestani M, Black J, Carville K, Clark M, Cuddigan J, Dealy C, Defloor T, Gefen A, Harding K, Lahmann N, Lubbers M, Lyder C, Ohura T, Orsted H, Ranganathan VK, Reger SI, Romanelli M, Sanada H, Takahashi M. Pressure ulcer prevention: pressure, shear, friction and microclimate in context. A consensus document. *Wounds Int* 2010;26:1–25.
- 6 National Pressure Ulcer Advisory Panel. Pressure ulcer stages revised by NPUAP. www.npuap.org 2007 February 1. URL <http://www.npuap.org/pr2.htm> [accessed on 1 February 2012].
- 7 Gupta S, Baharestani M, Baranoski S, De Leon J, Engel SJ, Mendez-Eastman S, Niezgoda JA, Pompeo MQ. Guidelines for managing pressure ulcers with negative pressure wound therapy. *Adv Skin Wound Care* 2004;17 Suppl 2:1–16.
- 8 Baharestani M, De Leon J, Mendez-Eastman S, Powell G, Weir D, Niezgoda J, Payne W, Nanney LB, Pelham F, Gupta S. Consensus statement: A practical guide for managing pressure ulcers with negative pressure wound therapy utilizing vacuum-assisted closure – understanding the treatment algorithm. *Adv Skin Wound Care* 2008;21 Suppl 1:1–20.
- 9 Heyneman A, Beele H, Vanderwee K, Defloor T. A systematic review of the use of hydrocolloids in the treatment of pressure ulcers. *J Clin Nurs* 2008;17:1164–73.
- 10 Fowler E, Papen JC. Evaluation of an alginate dressing for pressure ulcers. *Decubitus* 1991;4:47–53.
- 11 Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen therapy for chronic wounds. *Cochrane Database Syst Rev* 2004;2:CD004123.
- 12 Mendez-Eastman S. Determining the appropriateness of negative pressure wound therapy for pressure ulcers. *Ostomy Wound Manage* 2004;50 Suppl 4A:13–6.

- 13 Niezgoda JA. Incorporating negative pressure therapy into the management strategy for pressure ulcers. *Ostomy Wound Manage* 2004;50 Suppl 11A:5S–8S.
- 14 An overview of scientific and clinical evidence utilizing KCI Therapeutic Support Surfaces and V.A.C. Therapy for the prevention and treatment of pressure ulcers. Response to AHRQ Request for Information. San Antonio, TX: Kinetic Concepts, Inc., 2012.
- 15 Wild T, Stremitzer S, Budzanowski A, Hoelzenbein T, Ludwig C, Ohrenberger G. Definition of efficiency in vacuum therapy—a randomized controlled trial comparing Redon drains with V.A.C.® Therapy. *Int Wound J* 2008;5:641–7.
- 16 Prinz M, Budzanowski A, Stremitzer S, Hoelzenbein T, Wild T. Objective wound assessment and documentation with WHAT. *Zeitschrift für Pflegewissenschaft* 2005;7:175.
- 17 Moues CM, Vos MC, Van Den Bemd GJ, Stijnen T, Hovius SE. Bacterial load in relation to vacuum-assisted closure wound therapy: a prospective randomized trial. *Wound Repair Regen* 2004;12:11–7.
- 18 Joseph E, Hamori CA, Bergman S, Roaf E, Swann NF, Anastasi GW. A prospective, randomized trial of vacuum-assisted closure versus standard therapy of chronic nonhealing wounds. *Wounds* 2000;12:60–7.
- 19 Schwien T, Gilbert J, Lang C. Pressure ulcer prevalence and the role of negative pressure wound therapy in home health quality outcomes. *Ostomy Wound Manage* 2005;51:47–60.
- 20 Baharestani MM, Houliston-Otto DB, Barnes S. Early versus late initiation of negative pressure wound therapy: examining the impact on home care length of stay. *Ostomy Wound Manage* 2008;54:48–53.
- 21 Baranoski S, Ayello EA. *Wound care essentials: practice principles*. 2nd ed. Philadelphia: Lippincott Williams and Wilkins, 2008.
- 22 Schultz GS, Sibbald RG, Falanga V, Ayello EA, Dowsett C, Harding K, et al. Wound bed preparation: a systematic approach to wound management. *Wound Repair Regen* 2003;11 Suppl 2:S1–S28.
- 23 Falanga V. Wound bed preparation and the role of enzymes: a case for multiple actions of therapeutic agents. *Wounds* 2002;14:47–57.
- 24 V.A.C. Therapy clinical guidelines: a reference source for clinicians. 8-1-2010. San Antonio, TX: Kinetic Concepts, Inc.
- 25 Langemo DK. Quality of life and pressure ulcers: what is the impact? *Wounds* 2005;17:3–7.
- 26 Wilhelmi BJ, Vistnes LM. Surgical treatment of pressure ulcers. *eMedicine*. 2010.
- 27 Wu SC, Armstrong DG. Clinical outcome of diabetic foot ulcers treated with negative pressure wound therapy and the transition from acute care to home care. *Int Wound J* 2008;5 Suppl 2:10–6.
- 28 Gupta S, Gabriel A. Improving pressure ulcer outcomes using an algorithmic approach to negative pressure wound therapy. Presented at the Third Congress of the World Union of Wound Healing Societies, June 4–8, 2008, Toronto, Ontario. PW006. 6-4-2008.