Use of a Vacuum-Assisted Device in a Neonate With a Giant Omphalocele

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ABSTRACT
Wound healing is a complex process that can be even more challenging in neonatal and pediatric patients. Infants and children have special characteristics such as skin immaturity, a high body surface to weight ratio, sensitivity to pain, increased potential for percutaneous absorption of medication, and an immature immune system that adds to the complexity of treating their wounds. The use of controlled topical negative pressure across a wound surface has been used in adults and children since 1995. Recently, the use of this device has been reported in neonates. This article discusses the normal process of wound healing and describes the use of this device in an infant with a giant omphalocele.
Key Words: giant omphalocele, Vacuum-Assisted Closure, wound healing

The care and treatment of large chronic wounds in neonatal and pediatric Z can be very challenging. Since 1995, Vacuum-Assisted Closure® Therapy (V.A.C.®; Kinetic Concepts, Inc [KCI] San Antonio, Texas) has been a useful therapy in the management of complex wounds in adults and children. Little has been published regarding its use in full-term and premature infants. The V.A.C.® device uses the application of controlled topical negative pressure across a wound surface in a manner that produces rapid wound healing. This case presentation describes the use of this device in a full-term baby with a giant omphalocele who had difficult wound healing and reviews normal wound healing.

Case Presentation

Baby girl IZ was born at 38-week gestation to a 24-year-old, gravida 3, para 1 mother at a local referring hospital. Her mother was negative for hepatitis B and HIV, her rapid plasma reagin test was negative for syphilis, and her blood type was A negative. Her mother received early prenatal care and her pregnancy was complicated by an abnormal 2-hour glucose tolerance test and pregnancy-induced hypertension.

The infant was delivered vaginally under epidural anesthesia and her Apgar scores were 7 at 1 minute and 8 at 5 minutes. She weighed 3320 g. Resuscitation included oral and gastric suctioning, stimulation, and blow by oxygen. A giant omphalocele, measuring 10 × 12 cm, was noted at the time of delivery, which was not diagnosed antenatally. The defect was wrapped with sterile normal saline–soaked gauze and Kerlix (Kendall Brands, now part of Coviden, Mansfield, Massachusetts). A Replogle tube was also passed to decompress her stomach and bowel.

Shortly after delivery, IZ developed increased work of breathing with mild to moderate subcostal retractions and tachypnea because of the large size of the defect that limited her lung expansion, and she was placed into a 40% oxygen hood with good oxygen saturation levels achieved. A blood culture was obtained and a peripheral intravenous catheter was placed for antibiotics (ampicillin and gentamicin) and hydration. She was transported to a regional referral center for surgical repair and ongoing care.

Initially, the pediatric surgeon thought she would require a staged closure that involves placing a silo but a primary repair was achieved on day 1. During surgery, the omphalocele was excised and the liver, spleen, and the majority of bowel were extruded, the sac was completely excised, and the umbilical cord structures were ligated. The defect was closed

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using a Surgisis graft (Cook Medical Products, Bloomington, Indiana) that was placed over the defect attaching circumferentially to the fascia. The skin was then closed over the Surgisis graft. The repair was completed without evidence of significant abdominal compartment syndrome. A Broviac central venous catheter was also placed at the time of surgery. She received perioperative antibiotics, which were continued for 5 days.

Six days postoperatively, a patch of wound opened exposing the Surgisis mesh graft. The antibiotics amikacin and vancomycin were started as well as normal saline wet-to-dry dressing changes 3 times a day. Five days later, on day 11, the wound was malodorous and acetic acid wet-to-dry dressing changes were begun twice a day. On day 14, healing of the wound was improved, the wet-to-dry dressing changes were stopped, and a nonocclusive dressing was placed over the wound that has little granulation tissue over the mesh graft. Silvadene (silver sulfadiazine) was used topically daily beginning on day 17 for increased wound breakdown. During this time, IZ received 2 more courses of antibiotics. Over the next 3 days, progressive wound breakdown developed and the decision was made to consult the wound care team located at the hospital. A wound punch biopsy was done and showed a full-thickness wound with chronic infection of the SurgiSIS graft mesh. Silvadene was stopped and Silvasorb was placed over the wound twice a day.

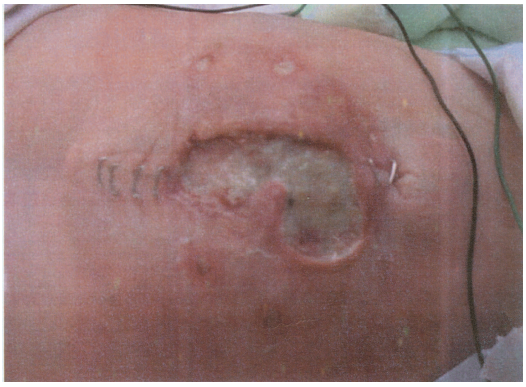
On day 47, IZ was taken to the operating room where the wound was debrided and 90% of the Surgisis graft mesh was excised. It was necessary to leave a portion of the graft mesh in place because the majority of the graft was adherent to the liver that made cautery dissection difficult. The fascial edges were joined by bolster sutures and the defect was closed. Postoperatively, the incision was cleaned with half-strength hydrogen peroxide and triple antibiotic ointment and she received 1-week dose of amikacin and vancomycin. Wound culture specimens that were taken intraoperatively were growing methicillin-sensitive *Staphylococcus aureus* and *Escherichia coli*. One week postoperatively, the skin broke down around the retention sutures. Once again, Silvasorb was used to treat the open wound. Progressive dehiscence developed, and 9 days postdebridement, the open wound measured 5 × 2 cm and contained fascia and Surgisis (Figure 1). At this time, the decision was made to apply a V.A.C.® Therapy device (KCI) (Figure 2).

Giant Omphalocele

Abdominal wall defects in infants have 3 subtypes: omphalocele, gastroschisis, and hernia of the cord. Closure of the fetal abdominal wall depends upon appropriate craniocaudal and lateral infolding of the embryonic disk. Migration and fusion of the cranial, caudal, and lateral folds normally result in an intact umbilical ring by 5-week gestation.[1] Partial or complete arrest of this process results in an omphalocele, with insertion of the umbilical cord onto the central omphalocele sac with a surrounding facial defect. The size of the defect may vary with large defects of the entire midgut as well as the stomach, liver, and spleen. In 50% of cases, the liver, spleen, and ovaries or testes accompany the midgut.[1] The abdominal cavity remains small with the absence of the viscera. One in 5000 babies has an omphalocele that involves the bowel only.[1] Only 1 in 10 000 infants have a giant omphalocele that involves the liver.[1]

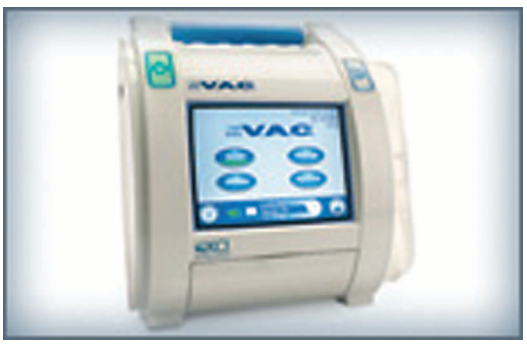
Fetal omphalocele may occur in conjunction with other conditions, such as cardiac or genitourinary

FIGURE 1.



Postoperative wound prior to application of the V.A.C.® therapy. Photograph courtesy of Presbyterian/St. Luke's Medical Center, Denver, Colorado. Reprinted with permission.

FIGURE 2.



Device used for wound vacuum assisted closure therapy, V.A.C.®, Kinetic Concepts, Inc (San Antonio, Texas). Reprinted with permission.

abnormalities, neural tube defects, and the genetic defects trisomy 13 or 18. In addition, omphalocele may be associated with Beckwith–Wiedemann Syndrome or pentalogy of Cantrell.

Normal Wound Healing

Wound healing is an ever-changing and delicate process that at times is taken for granted. The process is an overlapping series of events, beginning with injury to eventual repair. Normally, rapid wound healing occurs in infants and pediatric patients but healing can be delayed by a number of reasons including impaired perfusion, infection, prolonged pressure, poor nutrition, edema, and the wound environment. Neonatal and pediatric wound care requires special expertise and precise management. Infants and children have special characteristics such as skin immaturity, a high body surface to weight ratio, sensitivity to pain, increased potential for percutaneous absorption of medication, and an immature immune system that adds to the complexity of treating their wounds.[2,3] Effective wound management is dependent upon an understanding both of the normal repair process and of factors affecting this process and interventions that can impact the ultimate outcome. There are 4 phases of wound healing.

The first stage includes a vascular response, so within seconds of an injury, blood vessels constrict to stop any bleeding and to limit exposure to bacteria. Platelets cluster together to form a clot, which is the result of conversion of thrombin to fibrinogen and ultimately to fibrin.[4] The second stage involves an inflammatory response, which is the body's first defense system against bacterial invasion. Neutrophils along with macrophages arrive and ingest bacteria. Monocytes, which play a critical role in the healing process, arrive later. They phagocytose bacteria along with damaged tissue, engulfing and destroying microbes present there. Macrophages secrete angiogenesis factor, which stimulates the formation of new blood vessels.[4,5] Ischemic cells release the vasoactive substances bradykinin, histamine, and prostaglandin. Vessels start to dilate, permeability increases, and fluid begins to leak into the wound. Inflammation is a sign of healing but prolonged inflammation due to necrotic, infected tissue or foreign bodies slows the healing process and can lead to chronic wounds.

The proliferative phase is the third stage of healing and involves intense multiplication of cells. Angiogenesis, collagen synthesis, contraction, and epithelialization are part of this phase.[2] Angiogenesis is the growth of new capillaries by which local blood flow for healing is increased. Macrophages secrete lactate and growth factors into the wound, which stimulate fibroblast proliferation. Fibroblasts secrete collagen, which reconstructs connective tissue. Collagen is initially secreted as a gel matrix onto which collagen fibers are deposited, mature, and strengthen. This gel-like substance keeps the wound moist, which facilitates healing.[6] Vitamin C, zinc, oxygen, and iron are required for this process. Granulation occurs when collagen, capillaries, and cells begin to fill the wound space with new connective tissue. Granulation tissue is red and bumpy with a meaty appearance. The wound contracts as myofibroblasts align along the lines of contraction. This is a unified process requiring cell-to-cell and cell-to-matrix communication. The effect of contraction is to decrease the area to be filled in with granulation tissue. Reepithelialization begins as epithelial cells migrate from surrounding skin. Epithelial cells need a viable wound edge and a moist wound surface to migrate across a wound bed.[7] These cells eventually begin to differentiate into various layers of the dermis. The initial scar is bright red, thick, and blanches with pressure.

In the fourth stage, remodeling of the scar continues for about 1 year. Scar tissue regains about two-thirds of its original strength and is never as strong as the normal tissue and never fully retains tensile strength.[4] Wounds slow down or stop their healing process because of numerous factors. Tissue hypoxia is an important cause. Tissue hypoxia decreases resistance of the wound to infection by interfering with phagocytosis. Hypoxia impairs collagen synthesis and increases collagen lysis as well as decreases epithelial proliferation and migration.[8,9] A balanced nutrition is also very important for wound healing. A neonate should optimally be in a good anabolic state with good protein intake. Some centers measure albumin and prealbumin levels and consider them markers of optimal protein intake during their management of chronic wounds.[2] Full formula or breast milk feedings should provide enough protein to optimize wound healing. If oral feedings are not tolerated, total parenteral nutrition with adequate protein, glucose, and fat along with vitamins and trace minerals should be provided.

Wounds that are chronically infected are very slow to heal. Infection prolongs the inflammatory phase of healing, resulting in diminished levels of oxygen in the tissue, with decreased fibroblast production and diminished collagen formation. Many chronic wounds require surgical debridement before they can begin the healing process.

The use of corticosteroids can inhibit the inflammatory response and phagocytosis, interfering with healing. In addition, these medications decrease collagen strength and can inhibit epithelial proliferation.[4] A dry wound bed should be avoided because this leads to slow healing.[4] When a wound is dry, keratinocytes, which are the major cell type of the epidermis, to heal the wound, will need to burrow down to a moist environment in the wound in order to secrete collagenase, which lifts the scab. These

cells later migrate, differentiate, and resurface the wound. A dry wound bed will also interfere with epithelial proliferation and migration. A very important goal of wound healing is to provide a moist wound bed that stimulates the wound into its healing cascade.[4,7]

Infants have multiple developmental considerations that place them at higher risk for skin injury and slower wound healing. During the last trimester of pregnancy, collagen is deposited into the dermis. The dermis of a full-term baby is 60% as thick as that of an adult.[2] This lack of collagen places a neonate at greater risk of becoming edematous, making their skin more susceptible to injury. Differences in skin pH place a neonate at greater risk of skin breakdown. A full-term infant has an alkaline skin surface at birth. Within 4 days, the pH drops to less than 5, creating an "acid mantle." An acidic skin surface protects against bacterial invasion.[8] Premature infants have an immature stratum corneum, which is the outer layer of the dermis, and overall underdeveloped skin structures, and they are at risk for skin disruption and toxicity from topically applied substances. A number of studies involving wound cleansers indicated that several cleansers and disinfectants can destroy or damage fibroblasts and granulation tissue in healing wounds.[8,9] These include Ivory Liqui-Gel, Dial Antibacterial Soap, and Hibiclens. These products were at a 1/100,000 dilution to be considered nontoxic.[9] The skin and wound cleansers, povidone–iodine surgical scrub (Betadine Surgical Scrub) and hydrogen peroxide, were found to be nontoxic to fibroblasts at a 1/1000 dilution.[9] Shur-Clens was noted to be the least toxic to fibroblasts, requiring no dilution to maintain viable cells, with SAF-Clens and saline not far behind. Acetic acid, Biolex, Cara-Klenz, and Puri-Clens had a toxicity index of 10, corresponding to a 1/10 dilution.[9] This was an in vitro study and it is difficult to establish a direct correlation of in vitro findings with in vivo results. There may not be issues with the efficacy of cleansing actions but benefits to tissue repair should be cautiously examined.[9] It is possible that the use of hydrogen peroxide and acetic acid on IZ's wound added to its slow healing, but there were other significant factors including chronic infection that added to nonhealing of her wound. Silvadene, in addition, is not recommended for use in babies younger than 3 months because of concern for absorption of silver.[8]

Principles of Moist Wound Healing

The goal of wound healing is to accomplish all of the principles listed with every wound. These include oxygenation and circulation, removal of necrotic tissue, control of exudates and infection, and provision of a clean, moist, and protective environment. Vacuum-assisted closure V.A.C.® therapy has been used for the treatment of acute and chronic wounds.[10,11] Since its introduction more than a decade ago, it was initially used in adult wound healing and has been applied to the pediatric population. However, its use in neonates has been limited.

V.A.C.® therapy uses the application of controlled topical negative pressure across a wound surface in a manner that produces rapid wound healing.[10,12-14] This negative pressure system creates an environment within the wound bed that resists bacterial growth, encourages capillary growth, and establishes microcirculation. Blood is drawn into the wound bed and brings growth factor, neutrophils, and macrophages to the area. Again, neutrophils are the first responders; they phagocytize bacteria and breakdown fibrin. These cells activate fibroblasts and keratinocytes and attract macrophages to the area. These macrophages engulf large particles such as bacteria, yeast, and drying cells. They clean the wound and secrete cytokines and growth factors.

Cytokines and growth factors attract fibroblasts and endothelial cells, which convert oxygen to superoxide. Superoxide serves as a natural antimicrobial agent, inhibiting infection in the wound. Keratinocytes migrate into the wound bed and begin epithelialization, which, in turn, stimulates secretion of growth factors, cytokine activity, and angiogenesis.[4,14] Negative pressure increases local blood flow and decreases edema, which improves oxygen delivery to the wound bed.[12,14] Slough and loose necrotic material are removed from the wound, cleaning the wound and improving the blood supply. Removing the necrotic tissue decreases bacterial colonization.[12,14]

A good blood supply and a clean wound promote the formation of granulation tissue, which encourages wound closure and ensures that white blood cells are supplied with necessary oxygen while ensuring that aerobic bacteria in the wound bed die. V.A.C.® therapy provides a moist wound environment, which is essential for healing, preventing further necrosis and tissue loss.

V.A.C.® therapy is applied in a manner that was originally described in 1997, with clean wound technique predominantly used.[13] The choice of foam is important. The black reticulated foam (GranuFoam™ Dressing; KCI), is the most common dressing used. It is hydrophobic and does not absorb fluid, but it will stay moist under the occlusive drape. This foam is the most effective at stimulating granulation tissue and wound contraction. This foam is cut to the exact size of the wound. The White Foam™ Dressing (KCI) is an alternative, but it is a denser sponge. It is premoistened and nonadherent. It is more hydrophilic than the Black Foam. It is used more commonly in wounds with exposed tendon, bone, organs, fistulas, or tunnels. It is used to pack tunnels because of its higher tensile strength. A third type of foam,

GranuFoam Silver™ dressing, is also available. It is a Black Foam microbonded with silver that acts as an effective barrier to bacterial penetration and may help to reduce infection. Because the foam is kept constantly moist with the suction force of the pump, care is needed not to overlap the intact skin because maceration of the wound edges can occur. In large wounds, multiple pieces of Black Foam can be used.

The foam is placed into the wound without overlapping the edges and an occlusive drape dressing is placed over the wound extending on to the intact skin to create an airtight seal. A 2-cm hole is cut into the drape by pinching it over the foam. The SensaT.R.A.C.™ (Therapeutic Regulated Accurate Care; KCI) pad is placed directly over the hole in the drape and gentle pressure is applied. Then, the SensaT.R.A.C.™ pad tubing is connected to the canister tubing. The seal from the transparent drape needs to stay intact and occlusive for the therapy to be effective since air leaks are common problems (Figure 3). The V.A.C.® therapy unit is then programmed for a specific amount of suction-negative atmospheric pressure. For infants and children, there are no published recommendations. Typically, the lowest negative pressure of 50 mm Hg is chosen. The negative pressure settings vary from 50 to 200 mm Hg, administered continuously or intermittently. Continuous suction is typically used for the first 48 hours, later adjusted to intermittent suction. Dressing changes are commonly performed at 48-hour intervals or 3 times a week.[22] To prevent granulation tissue from growing into the foam, more frequent dressing changes should be performed.[22] To help minimize patient discomfort during dressing changes, the White nonadherent foam or a nonadherent dressing layer of Adaptic (Johnson & Johnson, New Brunswick, New Jersey) or Mepitel (Molynlycke Health Care, Eddyston, Pennsylvania) or applying a thin layer of a hydrogel to the wound base can be used to line the wound before the Black Foam is placed.[13-15] With wounds with extensive drainage, there is a collection canister that can accurately quantify the drainage.[15,16]

FIGURE 3.

Example of patient with the V.A.C.® therapy device in place. Photograph courtesy of Presbyterian/St. Luke's Medical Center, Denver, Colorado. Reprinted with permission.

Literature Review

Negative pressure therapy has been accepted as a valuable adjunct for wound closure in adults since 1993.[13] Its use was originally reported for the treatment of deep chronic wounds with moderate to high exudate levels such as pressure ulcers, abscesses, and deep wounds secondary to trauma. A retrospective study reported experience in 42 patients from 1999 to 2002 for conditions such as nonhealing sternal, spinal, and lower extremity wounds. The use of the V.A.C.® therapy provided faster wound healing, there were shorter hospital stays, and a reduction in overall cost.[16]

Another study by Mooney et al[17] reviewed 27 pediatric patients with complex wounds, which included open fractures, failed flap closure, abdominal and sterna dehisced wounds, and spinal wound infection. V.A.C.® therapy proved to be advantageous in this group, aiding in closure without need for complex surgical interventions.[17]

A retrospective medical record review of children and infants was conducted to evaluate the effectiveness of V.A.C.® therapy at a large pediatric hospital between January 2003 and 2005.[18] Data were collected on wound type, treatment method and duration, and complications. Sixty-eight patients with 82 wounds were identified. The mean age was 8.5 years and ranged from 7 days to 18 years. Twenty patients (29%), including 8 neonates, were 2 years or younger. Wound types identified were pressure ulcers, extremity wounds, dehisced surgical wounds, open sterna wounds, wounds with fistula, and complex abdominal wall defects. Following the use of negative pressure therapy, 93% of wounds decreased in volume. It was concluded that negative pressure therapy by using the V.A.C.® therapy system can be effectively used to manage a multitude of wounds in children and neonates. No major complications were identified.[18]

Another retrospective medical record review was conducted on 24 neonatal and pediatric patients who had received negative pressure wound therapy for their wounds from 1999 to 2004.[19] Their ages ranged from 14 days to 18 years. The most common wound type was traumatic, with exposed hardware and bone. In a median time of 10 days, 11 wounds were closed by flap, 3 by split-thickness skin graft, 4 secondarily, and 4 primarily. Results were promising. Complete closure was achieved in 22 of 24 patients.[19]

A report in 2006 described V.A.C.® therapy of 3 infants with giant omphalocele from 2002 to 2004.[20] All patients had undergone unsuccessful attempts at closure by using other methods. The first patient was initially treated by staged silo reduction, which disrupted after 21 days. The large mass of the bowel and liver made primary or skin flap closure impossible. V.A.C.® therapy was applied for 45 days. The viscera were subsequently covered with acellular dermal matrix (AlloDerm). The dermal matrix that failed to integrate into the fascial rim was removed. The small remaining defect was covered with split-thickness skin graft at 3 months of age. In the second case, mesh placement was performed 5 months after birth, with subsequent necrosis of the infant's abdominal skin within the immediate postoperative period. The mesh was removed and V.A.C.® therapy was applied for 22 days. The infant subsequently underwent acellular dermal matrix replacement of the fascial defect and full-thickness skin flap closure by tissue expansion. The third case was of a full-term infant with a 6-cm omphalocele that was initially treated by staged silo reduction. After multiple suture line disruptions, the silo was removed and gross-type skin flaps were used to cover the large defect. This procedure was complicated by an enterocutaneous fistula. The mesh was removed and V.A.C.® therapy was applied for 36 days. A healthy granulation bed developed and the V.A.C.® therapy device was allowed for the treatment of the fistula and coverage of the defect. This case series illustrated the challenges faced by pediatric surgeons in the management of giant omphalocele and demonstrates the usefulness of V.A.C® therapy.[20]

In 2005, the V.A.C.® system was used in the care of 2 premature infants, weighing less than 1500 g, with extensive soft tissue defects.[21] The first case involved a former 23-week gestation infant who at 6 weeks of age and 850 g was found to have an omphalomesenteric duct fistula that became infected and ruptured into the abdominal wall. At laparotomy, a 3-cm segment of ileum adjacent to the ruptured omphalomesentric duct was resected. An ileostomy and mucus fistula were placed but the patient had necrosis of the midline musculature and the closure was not accomplished. A bovine pericardial patch was used for temporary closure of the muscle defect, and the overlying necrotic skin was debrided. One week following surgery, V.A.C.® therapy was applied. The White Foam was placed over the defect and a negative pressure of 75 mm Hg was applied. The dressings were changed every 2 to 3 days for 43 days. V.A.C.® therapy was discontinued when the wound was at the level of the skin and the suction device was bigger than the open wound. Later, wet-to-dry dressings were used and the wound was completely epithelialized 14 days after V.A.C.® therapy removal.[21] In the second case, the neonate was a former 27-week gestation infant, born weighing 800 g, who developed a bowel perforation secondary to necrotizing enterocolitis.[21] She received total parenteral nutrition through a percutaneously placed central venous catheter through a saphenous vein that infiltrated, and she developed on day 31 a 7 × 3.5-cm blister on her lower back. There was extensive full-thickness necrosis from the T8/9 region to L5/S1 over the posterior torso and dehiscence of tissues between the paraspinous muscles, involving the spinal laminae and epidural tissue. The dura was exposed but viable. Following intravenous antibiotic administration and debridement, a 7 × 10-cm defect remained on her back. The V.A.C.® therapy system was applied initially by using the White Foam at a negative pressure of 50 mm Hg. Within a week, this was changed to the Black Foam and the negative pressure was increased to 75 mm Hg. The V.A.C.® therapy dressing was changed every 3 to 4 days for 21 days. Mepitel (Molylycke Health Care) was applied to the wound and changed daily until the wound was completely epithelialized 10 days following V.A.C.® therapy removal.[21]

Nursing Implications

When a V.A.C.® therapy device is applied, it is the responsibility of the nurse to maintain its function and settings that are outlined by the wound care team. A team-centered approach should be used and a care plan for the V.A.C.® therapy changes should be instituted at the bedside.

Maintenance of the V.A.C.® therapy system is important and careful assessment is vital to ensure proper negative pressure. Air leaks from under the occlusive dressing are common problems. An air leak can be identified when a hissing sound is heard; smaller leaks may be auscultated with the use of a stethoscope. An air leak would also be suspected when the foam is observed to not being collapsed because the negative pressure has been lost. The pump will also alarm if negative pressure is lost. If an air leak develops, it can be patched with an additional drape.[15]

Pain assessment and treatment should be a priority. Initially, continuous suction is typically used. A pain assessment scale should be put into practice and a pain management plan instituted. Some patients experience pain during the dressing change when the pump is initially turned on and the foam is compressed. Pain medication should be given in 10 to 30 minutes, depending on the route of medication before the dressing change. Most infants and children with moderate wounds tolerate device changes with oral pain medications. Typically, acetaminophen is used for pain control but with larger wounds, parenteral pain medication or conscious intravenous sedation may be needed.[10] If pain or bleeding seems excessive with V.A.C.® therapy dressing changes, one should assess for invasion or adherence of

granulation tissue to the Black Foam. Intermittent suction has been proven to accelerate the growth of granulation tissue faster than continuous suction.[23] Therefore, switching to continuous suction may diminish rapid growth of granulation tissue and dressing changes may be more comfortable. Lining the wound bed with a nonadherent, oil emulsion–type dressing (eg, Adaptic; Johnson & Johnson) or a contact lining (Mepitel; Molynlcke Health Care) may disrupt adherence of the V.A.C. sponge.[23] Decreasing the amount of suction used may also help with pain. More frequent dressing changes may also decrease the growth of granulation tissue into the foam. The manufacturer of the wound V.A.C. device recommends dressing changes every 48 hours for most wounds.[15] Denuded wound margins are noted by older patients and researchers to be a common source of wound pain.[23] To protect intact wound margins under the occlusive V.A.C.® therapy drape, the V.A.C.® therapy sponge should be cut to the exact size of the wound, avoiding overlapping of the sponge on to good skin or to apply a water-soluble skin sealant (3M No Sting Barrier swab; 3M HealthCare, St Paul, Minnesota) as primary prevention. If periwound skin margins break down, one source recommends applying 1-in strips of thin hydrocolloid (Duoderm Thin; Convatec, Princeton, New Jersey) or thin adhesive form (Allevyn; Smith & Nephew, Largo, Florida) to protect the areas that are open before applying the V.A.C.® therapy drape.[23] Include parents in comfort measures during and after dressing changes. Offering an oral sucrose solution may also help with pain.

Close monitoring of fluid loss from the wound into the canister is extremely important, especially in highly exuding wounds or large wounds in relation to patient size. Neonates in particular can lose a significant amount of extracellular fluid from the wound bed and are at risk for dehydration. Accurate measurement is required since fluid replacement may need to be instituted.

Rapid contraction of the wound bed can occur shortly after placing the V.A.C® therapy on an infant with a large abdominal wound at risk for respiratory embarrassment. Care should be taken to follow the neonate's work of breathing and oxygen requirement and to stabilize as needed.

Developmental care issues need to be addressed. Depending upon the site of the wound, neonates may need to lie in a position that is not developmentally supportive. For instance, IZ could lie only on her back because of the limitation of her large wound and the V.A.C.® therapy device. However, positional supports and boundaries were provided to optimize and facilitate appropriate postural alignment. Her mother could not provide kangaroo care. Providing age-appropriate stimulation is important since infants can be spending significant time in their crib while the wound is healing. Range-of-motion exercises should also be considered as indicated. Optimally, a developmental specialist should be a part of the care team.

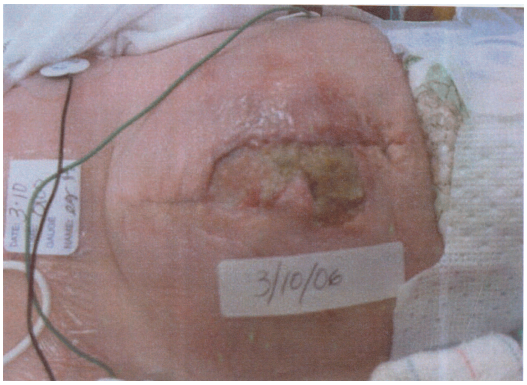
Wound healing can test family members to the limit of their endurance. A family may have to endure multiple surgeries and a prolonged hospitalization when healing is nonexistent or slow. Frustration with the lack of progress in healing is common. Doubts and fears should be treated with respect. Depression can be avoided or improved with good psychological support. Information about wound healing and how a wound V.A.C. system works should be provided.

IZ's Outcome

Three days following the initial V.A.C.® therapy, IZ's wound was showing some granulation tissue and was epithelializing along the wound edges. One week following placement, there was marked improvement; the wound showed healthy granulation tissue. Fibrous tissue was noted to be present as well and this was treated with Accuzyme (DPT Laboratories, Ltd, San Antonio, Texas) during dressing changes to debride this tissue (Figure 4). The wound was assessed and the V.A.C.® therapy device was reapplied every other day. Following 7 weeks of negative pressure wound therapy, the wound was healed enough to stop the V.A.C.® therapy.

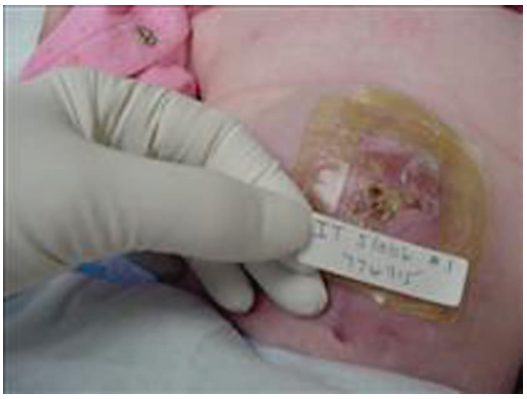
IZ was discharged from the hospital at approximately 5 months of age. In addition to the difficult wound-healing course, IZ had feeding difficulties because of significant gastroesophageal reflux that was related to her large abdominal wall defect. In

FIGURE 4.



Wound after 3 days of V.A.C® therapy. Photograph courtesy of Presbyterian/St. Luke's Medical Center, Denver, Colorado. Reprinted with permission.

FIGURE 5.



Wound after 2 months V.A.C® therapy. Photograph courtesy of Presbyterian/St. Luke's Medical Center, Denver, Colorado. Reprinted with permission.

addition, coordination and stamina were issues for her, which made nippling a challenge. She was discharged from the hospital with an indwelling nasal gastric tube in place. She was not a candidate for surgical treatment of her gastroesophageal reflux because of previous multiple abdominal wall surgeries. She was nippling small volume of formula during the day and was fed the remainder by her mother with gavage feedings. To improve growth, she was discharged home on 27 cal/oz of formula and received continuous-drip nighttime feedings.

Following discharge, she was followed by the Wound Healing Center and pediatric surgeons. One week following discharge, her wound began to break down once again (Figure 5). V.A.C.® therapy was reapplied and her mother was instructed on how to change the V.A.C.® therapy dressings. Three weeks later, the wound was completely healed and V.A.C.® therapy was discontinued. Her wound has remained closed.

Wound healing can be difficult in neonates, particularly those with large wounds or surgical sites. The use of controlled topical negative pressure with systems such as the V.A.C.® therapy system may be helpful in some infants.

References

1. Magnuson DK, Parry RL, Chwals WJ. Abdominal wall defects. In: Martin RJ, Fanaroff AA, Walsh MC, eds. *Fanaroff and Martin's Neonatal-Perinatal Medicine, Diseases of the Fetus and Newborn*. 8th ed. Philadelphia, PA: Mosby-Elsevier; 2006:1380-1386.
2. Lund, CH, Tucker JA. Adhesion and newborn skin. In: Hoath SB, Mailbach HI, eds. *Neonatal Skin Structure and Function*. 2nd ed.; pp. 299-324. New York, NY: Marcel Decker Inc; 2003.
3. Barharestani M, Pope E. Chronic wounds in neonates and children. In: Krasner D, Rodheaver GT, Sibbald RG, eds. *Chronic Wound Care: A Clinical Source Book for Healthcare Professionals*. 4th ed. Malvern, PA: HMP Communications; 2007:673-693.
4. Strodtbeck F. Physiology of wound healing. *Newborn Infant Nurs Rev*. 2001;1:43-52.
5. Clark RAF. Wound repair: overview and general considerations. In: Clark RAF, ed. *The Molecular and Cellular Biology of Wound Repair*. 2nd ed. New York, NY: Plenum Press; 1995:3-50.
6. Flanigan KH. Nutritional aspects of wound healing. *Adv Wound Care*. 1997;10: 48-52.
7. Waldrop J, Doughty D. Wound healing physiology. In: Bryant RA, ed. *Acute and Chronic Wounds. Nursing Management*. 2nd ed. St Louis, MO: Mosby; 1997:413-429.
8. Association of Women's Health, Obstetric and Neonatal Nurses. *Neonatal Skin Care, Evidenced Based Clinical Practice Guidelines*. 2nd ed. Washington DC: AWONN; 2007.
9. Wilson J, Mills J, Prather I, Dimitrijevich SD. A toxicity index of skins and wound cleansers used on in vitro fibroblasts and keratinocytes. *Adv Skin Wound Care*. 2005;18:373-378.
10. Caniano DA, Teich S, Ruth B. Wound management with vacuum-assisted closure: experience in 51 pediatric patients. *J Pediatr Surg*. 2005;40:128-132.
11. Jerome D. Advances in negative pressure wound therapy. *J Wound Ostomy Continence Nurs*. 2007;34:191-194.
12. Smith N. The benefits of VAC therapy in the management of pressure ulcers. *Br J Nurs*. 2004;13:1359-1365.
13. Argenta L, Morykwas M. Vacuum-assisted closure: a new method for wound control and treatment. *Annu Plast Surg*. 38:563-576.
14. Miller M, Glover D. *Wound Management: Theory and Practice*. London, England: The Friary Press; 1999.
15. KCl The Clinical Advantage. *V.A.C. Therapy Clinical Guidelines: A Reference Source for Clinicians*; 2007. Accessed October 2008 from http://www.KCl1.com/KCl1/vacapplicationsvideos
16. Antony S, Terrazas S. A Retrospective study: clinical experience using vacuum assisted closure of the treatment of wounds. *J Natl Med Assoc*. 2004;96:1073-1077.
17. Mooney JF, Argenta LC, Marks MW, et al. Treatment of soft tissue defects in pediatric patients using the VAC system. *Clin Orthop Relat Res*. 2000;376:26-31.
18. McCord SS, Murphy K, Olutoyeo L, Naik-Mathuria B, Hollier L. Negative pressure wound therapy is effective to manage a variety of wounds in infants and children. *J Wound Ostomy Continence Nurs*. 2007;34:573-574.
19. Baharestani M. Use of negative pressure wound therapy in the treatment of neonatal and pediatric wounds: a retrospective examination of clinical outcomes. *Ostomy Wound Manage*. 2007;53(6):75-85.
20. Kilbride K, Cooney D, Custer M. Vacuum-assisted closure: a new method for treating patients with giant omphalocele. *J Pediatr Surg*. 2006;41:212-215.
21. Arca M, Somers K, Derks TE, et al. Use of vacuum assisted closure system in the management of complex wounds in the neonate. *Pediatr Surg Int*. 2005;21:532-535.
22. Bookout K, McCord S, McLane K. Case studies of and infant, a toddler, and an adolescent with a negative pressure wound treatment system. *J Wound Ostomy Continence Nurs*. 2004;31:184-192.
23. Krasner D. Managing wound pain in patients with vacuum assisted closure devices. *Ostomy Wound Manage*. 2002;48(5):38-43.