A. Qualified professional multidisciplinary team evaluate and document VU patient diagnosis and risk factors for delayed healing or recurrence of VU to guide treatment plan

1. Patient history in context of physical examination including patient’s condition, history of deep vein thrombosis (DVT), pulmonary embolism or malignancy, ulcer treatment history, medical and surgical history and medications: A (Lee et al., 2002; Nelzén & Fransson 2007; Szewczyk et al., 2009)
   a. Post thrombotic syndrome (PTS) or venous insufficiency in superficial and/or deep vein systems and/or the perforating venous system: A (Cushman et al., 2010; Kahn et al., 2005; Szewczyk et al., 2009; Volikova et al., 2009)
   b. Ache, heaviness, pain, itching or tiredness in leg: C (ASPS, 2010; Saedon & Stansby, 2010)
   c. Chronicity of the VU (duration in years ≥ 6 months): A (Kulkarni et al. 2007; Margolis et al., 2000)
   d. History vascular surgery, trauma, repeat intimal venous damage or varicosities: A (Mudge 1988; Nelzén & Fransson 2007; Nicolaides et al., 2000)
   e. Family history venous ulcer: A (Cushman et al., 2010; Bérard et al., 2002)
   f. History of vigorous exercise: C (Berard et al., 2002)
   g. Multiple pregnancy: C (Berard et al., 2002)
   h. Male gender: A (Cushman 2007; Lee et al., 2002; van Rijswijk et al., 1993)
   i. Obesity: A (Cushman et al., 2010; Prandoni & Kahn, 2009)
   j. Increasing age > 50 years: A (Cushman 2007; Cushman et al., 2010; Kulkarni et al., 2007)

2. Physical Exam
   a. CEAP Score: Clinical severity, etiology, anatomy, pathophysiology (CEAP Link: http://www.veithsymposium.org/pdf/avi/4304.pdf): A (Carpentier et al., 2003; Eklöf et al., 2004; Nelzén & Fransson 2007; Vasquez et al., 2010; Volikova et al., 2009)
   b. Lower leg edema: A (Burton, 1993; Duby et al., 1993; Ennis & Menesis, 1995; Kahn et al., 2005; Sampaio et al., 2010)
   c. Growth of hair indicates adequate arterial supply and likely VU healing outcome: B (Grey et al., 2006; Powell, 2010; Sampaio-Santos et al., 2010)
   d. Increased dermal thickness, e.g. >1.985 mm measured using high frequency ultrasound: A (Vesic et al., 2008; Volikova et al., 2009; Xia et al., 2004)
   e. Stasis dermatitis including tan or red-brown skin color (hemosiderin deposits) usually at medial ankle, small erosions, may be open or crusted: A (Alguire et al., 1997; Burton, 1993; Pappas et al., 1997)
   f. Lipodermatosclerosis, (fibrosis of dermis resulting in fibrosing panniculitis of leg with severe, chronic venous insufficiency): A (Geyer et al., 2004; Navarro et al., 2002; Volikova et al., 2009)
   g. Medial lower leg site, with slower healing in ulcers associated with VU location in back calf region: A (McGuirk et al., 2002; Pappas et al., 1997; Szewczyk et al., 2009)
   h. Varicosities: C3 (Alguire et al., 1997; Weiss, 1995)

3. Differential diagnosis to determine cause of ulceration
   a. Ankle Brachial Index (ABI) ratio < 0.8 suggests referral to specialist to assess for arterial disease. (Link forABI professional instructions: http://www.nursingtimes.net/nursing-practice-clinical-research/doppler-assessment-calculating-an-ankle-brachial-pressure-index/205076.article) (Link for ABI interpretation: http://www.healthfair.com/ScreeningsOffered/AnkleBrachialIndex.aspx A (ASPS, 2007; Bjellerup, 2003; Forssgren et al., 2008; Kjaer et al., 2005; McGuirk et al., 2002)

Reference citation: Association for the Advancement of Wound Care (AAWC) Venous Ulcer Guideline. Malvern, Pennsylvania: Association for the Advancement of Wound Care (AAWC). December 2010. Accessible at http://aawconline.org/professional-resources/resources/

Legend: Bold = Evidence Level A. Italics = Level B. Normal = Level C. Underlined = with cost analysis
For Evidence Level Criteria, Please see Page 1 of AAWC Venous Ulcer Guideline Evidence
Venous ulcers can exist in the presence of mixed arterial/venous pathology. C3 (ASPS, 2007; Bonham et al., 2009; Falanga 1997; Kerstein 1996).

**b. Duplex scanning ultrasound to measure blood flow and venous reflux:** A (Ghauri et al., 1998; Kjaer et al., 2005; Lee et al., 2002; Vesic et al., 2008; Yosadharga et al., 2003)

**c. Air plethysmography to monitor hemodynamic flow:** A (Cordts et al., 1992; Garcia-Rinaldi et al., 2002; Perrin, et al., 1999)

**d. Venous refill time (VRT > 20 seconds) measured with a below-knee tourniquet and photoplethysmography to diagnose venous insufficiency and predict VU non-healing:** A (Heit et al., 2001; Kulkarni et al., 2007; Phillips, 1999)

i. **Increased VRT after vein surgery predicts VU healing and non-recurrence:** A (Gohel et al., 2007; Kulkarni, et al., 2007; Phillips, 1999)

**e. Transcutaneous oxygen tension (TCPO2) near VU ≥ 30 mmHg to rule out arterial disease or predict VU healing:** A (Alexanderhouse Group, 1992; Belcaro et al., 2003; Lo et al., 2009; Stacey et al., 1990)

**f. Hypercoagulation, including elevated factor VIII related antigen, von Willebrand factor (VWF), D-dimer and factor V Leiden:** A (Blomgren et al., 2001; Cushman et al., 2010; Fink et al., 2002)

**g. Skin perfusion pressure ≥ 30 mmHg to predict VU and other chronic leg ulcer healing:** A (Adera et al., 1995; Lo et al., 2009; Yamamada et al., 2008)

**h. Elevated temperature > 1.1°C peri-ulcer predicts infection:** A (Woo & Sibbald, 2009; Fierhaller & Sibbald, 2010)

i. Leg ulcers that fail to improve within 6 weeks despite consistent evidence-based treatment may not be caused by venous insufficiency. Consider biopsy for histological diagnosis, or other procedures to identify suspected malignancy, vasculitis, pyoderma gangrenosum, mycobacterial or fungal infection or other atypical etiologies: C2 (Combemale et al., 2007; Schnirring-Judge & Belpedio, 2010; Shelling et al., 2010)

---

**B. Document venous ulcer wound characteristics, monitor and manage ulcer progress**

1. **Document VU progress weekly, or sooner if there is a significant change in ulcer status:** A (Bolton et al., 2004; Kerd et al., 2009; McGuckin et al., 2002)
   a. **Use validated measures that include wound depth (i.e. Bates-Jensen Wound Assessment Tool© or Wound Bed Score)** A (Bolton et al., 2004; Falanga et al., 2006; Keast et al., 2004)

2. **Measure VU area or estimate it from longest length x width to assess healing progress:** A (Kantor & Margolis, 1998; Kantor & Margolis, 2000; McGuckin et al., 2002)
   a. **If wound area has not decreased by at least 40% after 3 weeks of a treatment regimen it is unlikely to heal. Inform care-givers. Re-evaluate diagnosis and/or care plan:** A (Phillips et al., 2000; van Rijswijk et al., 1993; Kerd et al., 2009)
   b. **A multidisciplinary wound team or clinic can improve healing, pain, and other health-related quality of life outcomes, reducing costs of patient care and hospitalization:** A (Gottrup et al., 2001; Marshall et al 2001; Moffat & Franks, 2004; Van Hecke et al., 2008; Vu et al., 2007)
   c. **Community nursing interventions including peer support improve healing, functional ability and health-related quality of life:** A (da Silva et al., 2006; Edwards et al., 2005; Edwards et al., 2009; Forssgren et al., 2008)
   d. Patients who visit the clinic at least once per week experience better and faster VU healing outcomes than those with biweekly visits: C1 (Warriner et al 2010)

3. **If complications, non-adherence to protocol or other issues arise, revise care plan or goals of treatment to address patient concerns:** C (RNAO, 2010; Lorimer et al., 2003)

4. **Validated quality of care VU outcome indicators include healing, recurrence, health-related quality of life and pain:** A (Clarke-Maloney et al., 2005; Edwards et al., 2009; Kjaer et al., 2005)

**C. Patient-oriented care to prevent or heal VU, and prevent VU recurrence. Improve venous return and provide patient and skin care.**

1. **Patient education including cause of skin breakdown, smoking cessation, how and why to use compression and leg elevation:** A (Lorimer et al 2003; McGuckin et al 2002; Van Hecke et al 2009)
2. Lower Leg Elevation is associated with VU healing and reduced recurrence C1 (Abu-Ouw et al., 1994; Collins & Seraj, 2010; Xia et al. 2004)
3. Ambulation or exercise to improve venous hemodynamics or calf muscle pump function C1 (Alexanderhouse Group, 1992; Kerstein, 1996; Padberg et al., 2004)
4. Safe, effective compression options to heal venous ulcers can be cost-effective: A Reduce compression as medically needed based on skin condition, limb shape, neuropathy, and cardiovascular or mixed venous/arterial disease confirmed during diagnostic work-up C3 (Bonham et al., 2009; EWMA 2003; Moffatt CJ et al 1992)
   b. Multi-layer sustained, elastic high-compression bandages, stockings or tubular bandages afford similar VU healing efficacy, better than single layer compression systems. Ideally match compression to patient needs and calf circumference: A (Milic et al. 2010; O’Meara et al 2009; Szewczyk et al 2009)
      i. Two-layer compression improves comfort or quality of life more than 4-layer or short-stretch: A (Jünger et al; 2009; Moffat et al 2008)
      i. High or medium compression elastic stockings derive similar recurrence rates with more consistent use of medium compression. Non-use of elastic compression or stockings is correlated with VU recurrence: A (Nelson & Jones., 2008)
   d. Duke Boot (i.e. Unna Boot + elastic compression and a hydrocolloid primary dressing reduces VU-related pain and is effective for healing most VU): A (Arnold et al, 1994; Lyon et al 1998; Eriksson 1986)
   e. Gradient compression better than uniform compression: C1 (Arnold et al , 1994; Sigel et al, 1975)
   f. Short stretch bandage heals VU more than usual care: A (Brizzio et al 2010; Gould et al 1998; Charles 1991)
   g. Unna boot zinc paste impregnated bandage heals VU more than no compression: A (DePalma et al 1999; Kitka et al., 1988; Rubin et al., 1990
   h. Intermittent pneumatic compression heals VU more than no compression A (Nelson et al 2008; Pekanmaki et al., 1991; Coleridge-Smith et al., 1990). Use with caution if edema occurs centrally.
   k. Standardized manual lymphatic massage: A (Molski et al., 2009; Pereira de Godoy 2008)
5. Manage Peri-wound Skin
   a. Moisturize skin if dry: A (Beitz & van Rijswijk 1999; Finlayson et al., 2010; McGuckin et al., 2002)
   b. Protect skin from chemical or physical trauma: A (Hansson et al 1998; Sayag et al., 1996)
   c. Manage peri-ulcer skin infection, inflammation, edema and circulation. Be alert to sensitization reactions: A (Cameron et al., 2005; Mayrovitz & Larsen, 1994; Neander & Hesse, 2003)

D. Local Wound Care
1. Cleanse VU gently at low pressure (4-15psi) with safe, non-antimicrobial cleanser: A (Chaby, 2010; McGuckin et al., 2002; Romanelli et al., 2010)
2. Debride nonvital tissue to promote chronic VU healing A (Dumville et al., 2009; Mulder et al., 1993 Romanelli, 1997)
   a. Sharp debridement with scalpel, curette or scissors. Evidence cited does not address sterile surgical debridement in the operating room: C2 (Cardinal et al., 2009; Golinko et al., 2009; Williams et al., 2005)
   b. Enzymatic: A (Bergemann et al., 1999; Romanelli, 1997; Westerhof et al., 1987)
   c. Autolytic using hydrogel or hydrocolloid dressing: A (Dumville et al., 2009; Mulder et al., 1993; Romanelli, 1997)
   d. Larval Therapy (bagged or loose) debrides faster, with more pain and similar healing or cost effectiveness to hydrogel: A (Dumville et al., 2009; Wayman et al., 2000)
e. High frequency ultrasound improves healing for 7-8 weeks but not 12 weeks: A (Cullum et al., 2010)
   i. Low frequency ultrasound requires more evidence: C (Breuing et al., 2005; Cullum et al., 2010)
   f. Mechanical debridement can remove VU nonvital tissue and slough. It is less painful and progresses faster with EMLA cream: C (Donati et al., 1994; Lok et al., 1999)

3. Fill deep wounds: C (Beitz & van Rijswijk, 1999)
4. For all patients with a VU including those unlikely to heal, manage wound pain and pain related to VU cleansing and dressing change C2: (Chrisman et al., 2010; Price et al., 2008; Quintanal, 1999)
5. Manage excess exudate and associated odor, improve comfort and reduce dressing change frequency to support cost effective healing: A (Franks et al., 2007; Harding et al., 2001; Lyon et al., 1998)
   a. Alginate: A (Bergemann et al., 1999; Sayag, et al., 1996)
   c. Foam dressings: A (Franks et al., 2007; Gottrup et al., 2008)
   d. Composite dressing: A (Daniels et al., 2002; Jones, 2003; Vanscheidt, et al., 2004)
6. Maintain moist wound environment to support cost effective healing or to reduce venous ulcer pain: A (Chaby et al., 2007; Kerstein et al. 2001)
   a. Hydrocolloid dressings improve pain, healing and costs of care compared to gauze: A (Chaby et al., 2007; Kerstein et al. 2001; Singh et al., 2004)
   b. Hydrogel healing effects compared to saline or enzymatic debridement: A (He et al., 2008; Romanelli, 1997)
   c. Film dressings C1 (Davis et al, 1992)
7. Additional local anaesthetic or analgesic to manage wound-related pain in patients with a VU: A (Briggs et al., 2010)
   a. Ibuprofen-containing foam dressings reduce VU pain more than same non-medicated foam in the first week of use: A (Briggs et al., 2010; Gottrup et al., 2008; Jørgensen et al., 2006; Romanelli et al., 2009)
   b. 5% Eutectic Mixture of Local Anesthetics (EMLA) reduces debridement pain: A (Briggs et al., 2010; Lok et al., 1999)

E. Adjunctive interventions to apply if conservative therapy does not work in 30 days
1. Antimicrobial VU topical care if no healing is seen in 30 days: A (O’Meara et al., 2010)
   a. Consider systemic antibiotic use only on VU with clinical signs of infection: A (O’Meara et al., 2010)
   b. Cadexomer iodine dressings improves healing on clinically infected wounds: A (Hansson, et al., 1998; O’Meara et al., 2010)
   c. Silver-containing foam or collagen/oxidized regenerated cellulose dressings: A (Dimakakos et al., 2009; Jørgensen et al., 2005; Munter et al., 2006)
2. Biologic dressings if no healing is seen in 30 days: C1
   a. Collagen or collagen combinations: C1 (Lanza et al., 2008; Vin, et al., 2002[NS])
   b. Hyaluronic acid or other matrix molecular dressings may decrease VU size or fibrin slough. B (Meaume et al., 2008; Ortonne, 1996; Taddeucci et al., 2004)
3. Skin replacement and grafting options to cover wound if no healing is seen in 30 days, accompanied by proper compression: A (Jones & Nelson, 2007)
   c. Cultured epidermal autografts (autologous keratinocytes): B (Vanscheidt et al., 2007; Liu et al., 2004; Mol et al., 1991)
   d. Allografts: C2 (Beele, et al., 2005; Bolivar-Flores, 1999; Goedkoop et al., 2010; Lingren, et al., 1998: only RCT reported no statistically significant healing effect)
   e. Bi-layered bioengineered skin: A (Falanga et al., 1998; Jones & Nelson, 2007)
4. Biophysical interventions: A
   a. Electrical stimulation: A (Franek et al., 2000; Houghton et al., 2003; Janković & Binić, 2008)
b. Vacuum (Negative Pressure Wound Therapy) has limited evidence for preparing VU for autologous pinch grafting or in VU graft management: C1 (Mendonca, et al 2006; Ubbink et al.,2008; Vuerstaek et al., 2006)
   i. Topical NPWT over skin grafts on chronic wounds may increase the graft quality without significantly affecting the quantity of graft take: C1 (Moisidis et al., 2004)
   ii. Topical NPWT may increase the rate of depth, area or volume reduction of chronic wounds compared to saline gauze. Research is needed on complete healing effects: C1 (Joseph et al., 2000)

c. Warming:  C1  (Robinson & Santilli, 1998; Santill et al., 1999; Von Felbert et al., 2007)

d. Electromagnetic/Radiofrequency (RF) stimulation: A (Kenkre et al., 1996; Ieran et al., 1990; Stiller et al., 1992)

e. Laser, including infrared (IR) stimulation and monochromatic light stimulation: C1 (Flemming & Cullum, 2002b;Gupta et al., 1998)

f. Hyperbaric Oxygen:  C1 (Hammarlund & Sundberg, 1994; Kranke et al., 2004)

g. Ultrasound stimulation:  A (Al-Kurdi et al, 2008; Flemming & Cullum, 2002a; Prescott et al, 1987 ; Taradaj et al., 2008)

h. Whirlpool lacks evidence of efficacy for managing venous insufficiency:  C2 (McCulloch & Boyd, 1992)

5. Systemic pharmacological agents for VU healing or to prevent VU recurrence: A (Coleridge-Smith, 2005; Jull et al., 2006)
   a. Defibrotide C1 (Jull et al., 2006)
   b. Pentoxifylline: A(Collins & Seraj 2010; Falanga et al., 1999; Jull et al., 2006; Nelson et al., 2007 )
   c. Diosminhesperidin (Dafion 500 mg micronized purified flavonoid fraction):  A(Coleridge-Smith, 2005)
   d. Oxerutins (Horsechestnut seed extract) do not improve VU healing or recurrence: A (Leach et al., 2006; Wright et al., 1991)
   e. Stanozolol to reduce pain, edema or symptoms of venous insufficiency:  B (Stacey et al., 1990;Vesić et al., 2008)
   f. Acetylsalicylic acid/ aspirin: C1 (Layton et al., 1994)
   g. Solcoseryl (topical + systemic):  C1 (Biland et al., 1985)
   h. Iloprost infused intravenously: C1 (Ferrara et al. 2007)

6. Topical interventions for VU healing or recurrence: A (Gethin & Cowman, 2008; 2009)
   a. Platelet derived growth factor has shown no significant effects on VU: A (Wieman., 2003)
   b. Cultured allogeneic keratinocyte lysate: C1 (Harding et al., 2005)
   c. Manukah honey improves healing and pain in larger, longer-duration, sloughy VU compared to hydrogel: A (Gethin & Cowman, 2008; Gethin & Cowman, 2009)
   d. Mimosa extract 5% gel compared to hydrogel: C1 (Rivera-Arce et al., 2007)
   e. Amelogenin extra-cellular matrix protein: C1 (Romanelli et al., 2008)
   f. Thymosin-beta-4 has not yet significantly affected VU healing: C1 (Guarnera et al., 2010)

7. Corrective vascular surgery to improve vein function: A (Gloviczki et al., 2009)
   a. Minimally invasive subfascial endoscopic perforating vein surgery (SEPS) plus compression and wound care to heal VU and reduce VU recurrence compared to compression alone: A (Gohel et al., 2007; Van Gent et al., 2006; Wright, 2009)
      i. SEPS results in lower incidence of wound infections, shorter hospital stays and lower VU recurrence rates than classic open vein surgery: A (Gloviczki et al., 2009; Luebke et al. 2009)
   b. Classic open vein surgery (Linton procedure: high ligation, division, and stripping of the saphenous vein) may be needed for some patients, reducing VU recurrence compared to compression alone: A (Barwell et al., 2004; O’Donnell, 2010)
   c. Thermal laser great or small saphenous vein coagulation or ablation: A (Gloviczki et al., 2009; Viarengo et al., 2007)
   d. Thermal radiofrequency vein ablation: A (Gloviczki et al., 2009; Puggioni et al., 2005)
   e. Sclerotherapy with compression requires more evidence: C1 (Hamel-Desnos et al., 2010; O’Hare et al., 2010)
   f. Venous valve repair or reconstruction: C2 (Maleti et al., 2006; Perrin et al., 1999)
F. Local evidence-based wound care VU programs, pain management and patient education until healed

1. **Compression, elevation, ambulation post healing to prevent recurrence**: A (McGuckin et al., 2002; Nelson, et al., 2003)

2. **Elastic compression stocking use for 6 months after DVT may prevent PTS skin changes, but effects on VU remain to be clarified**: B (Prandoni & Kahn, 2009; Aschwanden et al., 2008)

G. Palliative care for patients with a VU

1. Establish individualized goals of care consistent with patient and family wishes and medical condition. Communicate to interdisciplinary team: C (Alvarez et al., 2007; Chrisman, 2010)

2. Continue evidence-based principles of VU care outlined in Sections A-E above to the extent acceptable to the patient and family, including correcting underlying pathology, addressing nutrition and other supportive aspects of care, and sensible, nonharmful) local wound management: C (Alvarez et al., 2007)

3. Attend to patient wound-related quality of life concerns including dressing comfort, wound fluid, odor, and bleeding: C (Alvarez et al., 2007; Chrisman, 2010; Price et al., 2008)

4. Maintain individual dignity and provide psychosocial support to reduce isolation: C (Chrisman, 2010)

5. If surgical management is considered, discuss benefits and risks with patient and family in terms of patient’s condition and goals, presence of devitalized tissue, infection potential and underlying pathogenesis, C (Lee et al., 2007)

   a. Healing may not be a realistic goal, but can occur, improving quality of life: C (Chrisman, 2010)
AAWC VENOUS ULCER (VU) ALGORITHM

Details accessible at http://aawconline.org/professional-resources/resources/

HISTORY

QUALIFIED PROFESSIONAL MULTIDISCIPLINARY TEAM

PHYSICAL EXAM


DIFFERENTIAL DIAGNOSIS VU?

NO: Refer to Appropriate Specialist

TREATMENT TO HEAL VU & PREVENT RECURRENCE: Alleviate edema and VU cause. Cleanse ulcer with a safe cleanser. Debride nonvital tissue. Hydrate wound if dry or manage excess exudate. Maintain moist wound environment. Manage pain and odor.

MANAGE PERI-WOUND SKIN: Moisturize and protect the skin. Manage local skin infection, itching, inflammation, edema and circulation. Be alert for sensitization.

PATIENT-ORIENTED ACTIONS TO PREVENT AND HEAL VU: Educate and coach those with venous insufficiency to elevate foot above heart, flex ankles, do tip toe exercises or walk. Apply proper patient-acceptable compression. Address only relevant patient and family goals in palliative care.

YES: Continue same treatment.

VU AREA 40% LESS IN 3 WEEKS?

NO: Check, revise diagnosis / care.

IF NO HEALING IN 30 DAYS CONSIDER ADJUNCTIVE THERAPY:
Topical Antimicrobial or Honey, Biologic Dressings, Bi-layered Bioengineered skin.
Biophysical electrical, ultrasound or radiofrequency stimulation.
Systemic pharmaceutical options with evidence of efficacy.
If conservative treatment fails, consider corrective vascular surgery to improve vein function, such as minimally invasive subfascial endoscopic perforating vein surgery (SEPS) plus evidence-based compression, wound, skin and patient care above.

Above evidence-based program of care until healed. Continue compression, elevation, skin care and exercise after healing to improve venous function, reduce edema and prevent VU recurrence.