



1. Overview

- a. In the management of wounds it is imperative to understand principles of wound healing.
- b. Assessment and critical thinking is essential to lower extremity preservation.
- b. Knowledge of the standards of care for (1) diabetic foot ulcers (DFU), (2) chronic venous ulcers, and (3) pressure ulcers, including long term management is vital.

2. Objectives for the workshop

- a. Describe pathophysiology in development of venous ulceration
- b. Differentiate between cellulitis and venous hypertension
- c. Differentiate between lymphedema, lipedema, and venous HTN
- d. List three primary goals of care when managing a patient who presents with a chronic venous ulceration
- e. Describe three standard of care principles in managing a DFU including the “gold standard” for DFU
- f. Understand principles for healing moist wounds
- g. Demonstrate how to perform a proper wound culture and compression wrap

3. Required Readings and Videos

a. Articles

- i. Armstrong, D. G., Boulton, A. J. M., & Bus, S. A. (2017). Diabetic foot ulcers and their recurrence. *New England Journal of Medicine*, 376(24), 2367-2375. https://arizona-primo.hosted.exlibrisgroup.com/permalink/f/1h28lag/TN_nejm10.1056/NEJMr1615439
- ii. Campbell, K. E., Baronoski, S., Gloeckner, M., Holloway, S., Idensohn, P., Langemo, D., & LeBlanc, K. (2018). Skin tears: Prediction, prevention, assessment, and management. *Nurse Prescribing*, 16(12), 600-607.
- iii. Bauer, K., Rock, K., Nazzal, M., Jones, O., & Qu, W. (2016). Pressure ulcers in the United States' inpatient population from 2008 to 2012: Results of a retrospective nationwide study. *Ostomy Wound Management*, 62(11), 30-38.
- iv. Carmel, J. (2011). Venous ulcers. In R. A. Bryant & D. P. Nix (Eds.) *Acute & chronic wounds: Current management concepts* (4th ed.) (pp. 194-212). St. Louis, MO: Elsevier/Mosby.

b. YouTube Videos:

- i. Q&A – Lymphedema for Healthcare Professionals – Lymphatic and Venous Disorders.
 - https://www.youtube.com/watch?v=xGMQCFIXu_4
- ii. Total Contact Cast for Diabetic Foot Ulcers
 - <https://www.youtube.com/watch?v=jHZt0naRnbM>
- iii. Diabetic Foot Wounds and Diabetic Limb Salvage Presentation.
 - <https://www.youtube.com/watch?v=clai-yMq6PE>
- iv. Venous Leg Ulcer
 - <https://www.youtube.com/watch?v=HGtQ609epXk>
- v. Multilayer Compression Wrap for Venous Ulcers
 - <https://www.youtube.com/watch?v=kMpswpqUwY4>

4. Required Procedure Competencies

a. Equipment

- Bandages
- Pink saline bullets
- 4X4 Gauze

- Tape
 - Disposable curettes
 - Culture kits
 - Large bandage scissors
 - Coban Lite (3M product with very specific mmHg when applied) or Profore (a high pressure compression)
- b. Examine the client.
 - i. You will find:
 - Swollen erythematous lower extremities
 - Chronic ulceration
 - c. Assess and document neurovascular status
 - General: gait, footwear
 - Vascular: pedal pulses (not reliable), color (hemosiderin staining), temperature of the limb, capillary refill of the toes
 - Skin/wound assessment: location, peri-wound, wound edge (epibole), exudate, slough, odor
 - d. Choose proper dressing
 - e. Whom to refer to; where and when
 - f. Follow up management for chronic disease

5. During CSI Skills Lab

- a. Prior to arriving, you are expected to have read and watched the above. The skills lab is intended to build upon the above information and allow you to engage in a more patient-centered way.
- b. You will spend sixty minutes at this skills station. This will be divided in the following manner:
 - i. 5 minutes: Short introduction to the skill
 - ii. 5 Minutes: Focused HPI (consider pointing out one student for OLDCARTS) and Basic Exam
 - iii. 45 Minutes: Procedure - Culture – Levine technique; Two-layer compression wrap; Selective debridement of a wound
 - iv. 5 Minutes: Final Report and Preceptor Presentation
- c. Please see the Case Study Worksheet on the next page

Case Study Worksheet: Student Guide

CC: Mrs. A is a 63 year old woman with a non-healing wound on the L lower extremity

| | | |
|----------|---------------------|--|
| O | Onset | |
| L | Location/radiation | |
| D | Duration | |
| C | Character | |
| A | Aggravating factors | |
| R | Relieving factors | |
| T | Timing | |

- Considering these answers, are there any follow up questions you would ask that would not be asked below in the ROS?

ROS: Given the above, which systems will you focus on?

| | |
|----------------------------|--|
| General | |
| HEENT | |
| Respiratory | |
| Cardiovascular | |
| Musculoskeletal | |
| Skin | |
| Endocrine | |
| GI/GU | |
| Genital | |
| GYN (if applicable) | |
| Neuro/Psych | |

Exam:

- How would you document the exam?

Differential Diagnoses:

- List three differentials in their order of likelihood
 1. Probable:
 2. Possible:
 3. Unlikely:

Preceptor Report:

Case Study Worksheet: Instructor's Guide

CC: Mrs. A is a 63 year old woman with a non-healing wound on the L lower extremity

| | | |
|----------|---------------------|------------------------|
| O | Onset | 6 weeks ago |
| L | Location/radiation | Left medial malleolus |
| D | Duration | |
| C | Character | Non-healing, worsening |
| A | Aggravating factors | |
| R | Relieving factors | None |
| T | Timing | Constant |

- Considering these answers, are there any follow up questions you would ask that would not be asked below in the ROS?
 - Have you had a wound like this before?
 - Have you tried to put anything on the wound?

ROS: Given the above, which systems will you focus on?

| | |
|----------------------------|---|
| General | Overall appearance, gait, footwear |
| HEENT | |
| Respiratory | |
| Cardiovascular | (Vascular) Pedal pulses; |
| Musculoskeletal | Swollen erythematous lower extremity, without warmth |
| Skin | Trophic skin changes; Wound assessment – location, peri-wound (temp, color); wound base (quality of tissue slough); wound edges (epibole, odor, drainage) |
| Endocrine | |
| GI/GU | |
| Genital | |
| GYN (if applicable) | |
| Neuro/Psych | Monofilament – check for sensation |

Exam:

- How would you document the exam?

Differential Diagnoses:

- List three differentials in their order of likelihood
 1. Probable: Venous ulceration
 2. Possible: Diabetic ulceration of the lower extremity
 3. Unlikely: Cellulitis

Preceptor Report:

Mrs. A is a 63 year old female who reports to the clinic with a complaint of a non-healing spider bite. She states she noticed the bite about six weeks ago. She's been treating at home with hydrogen peroxide and covering with antibiotic ointment. Lately the drainage has increased and the dressings smell (using sanitary napkins) which has prompted her to make appt. Exam: bilateral LE pitting edema, negative stemmers sign, 3.5 cm by 6 cm ulcer left medial malleolus. Malodor with LE erythema without warmth. Covered with slough and has dry exudate on the peri-wound. The wound is exquisitely painful.

Pressure Ulcers in the United States' Inpatient Population From 2008 to 2012: Results of a Retrospective Nationwide Study

Karen Bauer, NPC, CWS, CHRN; Kathryn Rock, MD; Munier Nazzal, MD, FRCS, FACS, RVT, RVPI, FACCWS; Olivia Jones; and Weikai Qu, MD, PhD

Abstract

Pressure ulcers are common, increase patient morbidity and mortality, and costly for patients, their families, and the health care system. A retrospective study was conducted to evaluate the impact of pressure ulcers on short-term outcomes in United States inpatient populations and to identify patient characteristics associated with having 1 or more pressure ulcers. The US Nationwide Inpatient Sample (NIS) database was analyzed using the International Classification of Disease, 9th Revision, Clinical Modification (ICD-9 CM) diagnosis codes as the screening tool for all inpatient pressure ulcers recorded from 2008 to 2012. Patient demographics and comorbid conditions, as identified by ICD-9 code, were extracted, along with primary outcomes of length of stay (LOS), total hospital charge (TC), in-hospital mortality, and discharge disposition. Continuous variables with normal distribution were expressed in terms of mean and standard deviation. Group comparisons were performed using *t*-test or ANOVA test. Continuous nonnormal distributed variables such as LOS and TC were expressed in terms of median, and nonparametric tests were used to compare the differences between groups. Categorical data were presented in terms of percentages of the number of cases within each group. Chi-squared tests were used to compare categorical data in different groups. For multivariate analysis, linear regressions (for continuous variable) and logistic regression (for categorical variables) were used to analyze the possible risk factors for the investigated outcomes of LOS, TC, in-hospital mortality, and patient disposition. Coefficients were calculated with multivariate regression with all included patients versus patients with pressure ulcers alone. The 5-year average number of admitted patients with at least 1 pressure ulcer was determined to be 670 767 (average overall rate: 1.8%). Statistically significant differences between patients with and without pressure ulcers were observed for median LOS (7 days [mean 11.1 ± 15] compared to 3 days [mean 4.6 ± 6.8]) and median TC (\$36 500 [mean \$72 000 ± \$122 900] compared to \$17 200 [mean \$32 200 ± \$57 500]). The mortality rate in patients with a pressure ulcer was significantly higher than in patients without a pressure ulcer (9.1% versus 1.8%, OR = 5.08, CI: 5.03-5.1, *P* < 0.001). Pressure ulcers were significantly more common in patients who were older or had malnutrition. The results of this study confirm the importance of prevention initiatives to help reduce the negative impact of pressure ulcers on patient outcomes and costs of care.

Keywords: retrospective study, pressure ulcer, wounds and injuries, hospitalization, outcome assessment

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Potential Conflicts of Interest: none disclosed

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Making healing possible

Pressure ulcers are one of the most common health conditions in the United States. The Agency for Healthcare Research & Quality (AHRQ) estimates more than 2.5 million individuals in the US develop pressure ulcers annually. The magnitude of this issue is evident in the fact that it spurred the AHRQ (supported by the Health Services Research and Development Service of the Department of Veterans Affairs and the Boston University School of Public Health) to form a panel of pressure ulcer experts from 6 medical centers to develop a pressure ulcer prevention toolkit to be used in acute care settings with a goal to decrease the incidence of pressure ulcers.¹

As a result of the subsequent increased health care utilization, medical management of pressure ulcers costs the US health care system \$9.1 billion to \$11.6 billion per year.¹ Since 2008, the Centers for Medicare and Medicaid Services (CMS) has discontinued hospital reimbursement for charges related to hospital-acquired conditions, which includes patients who acquire pressure ulcers during admission. A hospital stay involving a pressure ulcer may incur additional annual charges of up to \$700 000.¹ Treatment costs for a Stage 3 pressure ulcer range from \$5900 to \$14 840; treatment of a Stage 4 ulcer may cost between \$18 730 and \$21 410.²

In addition to direct costs, pressure ulcers incur costs in the form of litigation, penalties, and patient costs. More than 17 000 pressure ulcer-related lawsuits (with an average cost of \$250 000) are filed per year.³ Under the Affordable Care Act,³ hospitals also may be penalized up to 1% of their full reimbursement from Medicare if they have high nosocomial infection rates (includes infected pressure ulcers). The cost to the patient who develops a pressure ulcer is of utmost importance. It is estimated that up to 60 000 Americans die each year as a direct result of pressure ulcer-related complications³; a recent white paper² notes pressure ulcers negatively affect a person's quality of life and contribute to substantial psychological stress, pain, loss of work, burden to family, and mortality.

Much of the current focus regarding this public health issue is centered on the importance of prevention. Prevention and management of pressure ulcers require an interdisciplinary approach.¹ As the AHRQ pressure ulcer toolkit exemplifies, many health care systems are implementing improved care plans to deliver coordinated, high-quality care to patients with or at risk of developing pressure ulcers.¹

The purpose of this retrospective descriptive study was to evaluate the impact of pressure ulcers on short-term outcomes in US inpatient populations and identify patient characteristics associated with having 1 or more pressure ulcers.

Methods

Data source. Hospital admissions from 2008 to 2012 listed in the National Inpatient Sample (NIS) database (www.hcup-us.ahrq.gov/nisoverview.jsp) were culled. The NIS is the largest national all-payer hospital inpatient care database in the US and is supported by the Healthcare Cost

Key Points

- The purpose of this study was to evaluate both the rate of pressure ulcers (PUs) among patients in acute care facilities and the effect of having a PU on outcomes and costs of care.
- Using data from the 2008–2012 National Inpatient Sample database, 670 767 patients with at least 1 PU were identified (average rate over the 5-year period was 1.8%).
- The PU rate during that time period was generally stable, and most ulcers (47%) occurred on patients' lower back (lower back/sacral/coccygeal area).
- Compared to patients without a PU, patients with a PU were significantly more likely to have a longer length of stay, higher mortality rate, and higher hospital charges.
- The results of this study confirm PU prevention efforts are needed and warranted.

and Utilization Project (HCUP) of the AHRQ. The NIS contains data from more than 1000 community hospitals in the 47 states that participate in HCUP, which represents more than 95% of the US population. The database estimates a 20% stratified sample population of all nonfederal acute care hospitals throughout the US (excluding long-term care acute hospitals and rehabilitation centers). The NIS includes patients with Medicare and Medicaid, persons who are privately insured, and those who are uninsured. Hospital discharge data are collected annually, and the weighted data represent more than 7 million hospital admissions nationally. All patient and physician identifiers have been removed from this data set. Approval from the institutional review board was not required to conduct this analysis.

Patient selection. Within the HCUP database, patients with pressure ulcers were identified using the International Classification of Disease, 9th Revision, Clinical Modification (ICD-9 CM). Data for all patients having a diagnosis code for pressure ulcer (707.00 through 707.09) during a hospital admission from 2008–2012 were selected with no exclusion criteria. Patient demographics and comorbid conditions were recorded. The comorbidities were calculated with the comorbidity software developed by HCUP (www.hcup-us.ahrq.gov/toolssoftware/comorbidity/comorbidity.jsp#download) based on the study by Elixhauser et al.⁴ In addition, the following risk factors for pressure ulcer development were abstracted based on ICD-9 codes: malnutrition, shock/hypotension, peripheral vascular disease (PVD), incontinence, cerebrovascular disease (CVD), diabetes mellitus, and fractures (vertebral and femur) (see Table 1). Endpoints evaluated were length of stay (LOS, days), total hospital charge (TC),

Table 1. ICD-9 codes used to identify risk factors in the study

| Factor | ICD-9 codes |
|-----------------------------|--|
| Cerebrovascular disease | 430.xx, 431.xx, 432.xx, 433.xx, 434.xx, 435.xx, 436.xx, 437.xx, 438.xx |
| Malnutrition | V85.0x, V85.1x, 260.xx, 261.xx, 262.xx, 263.xx |
| Incontinence | 788.3x, 787.6x |
| Diabetes mellitus | 249.xx, 250.xx |
| Peripheral vascular disease | 440.2x, 440.3x, 440.4x, 443.1x, 443.8x, 443.9x, 444.22, 445.02 |
| Fracture of vertebral/femur | 805.xx, 806.xx, 820.xx, 821.xx, 827.xx, 828.xx |
| Hypotension | 458.xx, 785.5x, 998.0x, 958.4x |

inhospital mortality (Yes/No), and setting to which patient was discharged. The NIS database represents data collected during hospital stay; no postdischarge information was available or analyzed for this study.

Statistical analysis. Continuous variables with normal distribution were expressed in terms of mean and standard deviation. Group comparisons were performed using *t*-test or ANOVA test. Continuous nonnormal distributed variables such as LOS and TC were expressed in terms of median, and nonparametric tests were used to compare the differences between groups. Categorical data were presented in terms of percentages of the number of cases within each group. Chi-squared tests were used to compare categorical data in different groups. For multivariate analysis, linear regressions (for continuous variable) and logistic regression (for categorical variables) were used to analyze the possible risk factors for the investigated outcomes of LOS, TC, in-hospital mortality, and patient disposition. Coefficients were calculated with multivariate regression with all included patients versus patients with pressure ulcers alone. All statistical analysis was performed using IBM's Statistical Package for Social Sciences (SPSS) software version 21 (IBM Corp, Armonk, NY). Type I error rate was set at 0.05.

Results

Patient demographics and prevalence rates. The 5-year average number of admitted patients with 1 or more pressure ulcers in the US from 2008 to 2012 was determined to be 670 767. The total number of annual cases remained stable: 685 526; 678 026; 662 111; 718 550; and 609 620 in the years 2008 to 2012, respectively (see Figure 1). The average overall rate of patients with at least 1 pressure ulcer across all 5 years was 1.8%. Mean overall age of patients with a pressure ulcer was 71.2 ± 16.8 years (male 68 ± 17.4 , female 74.1 ± 15.5) (see Table 2). Patients with a pressure ulcer were significantly older

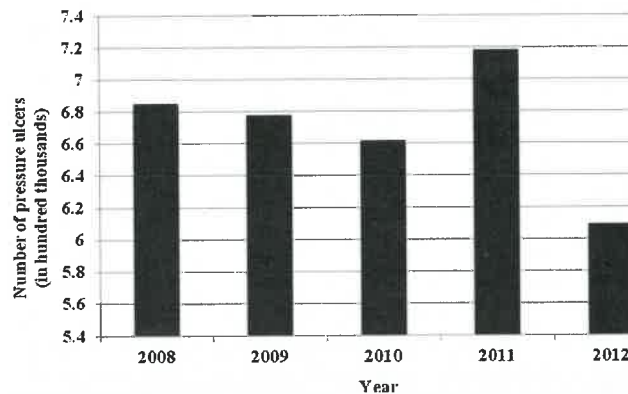


Figure 1. Estimated annual number of hospitalized patients with pressure ulcers in the United States from 2008 to 2012 (National Inpatient Sample).

than persons without pressure ulcers ($P < 0.001$). The rate of patients with pressure ulcers increased with increasing age. Men had a significantly higher rate than women across all age groups ($P < 0.001$), except for admissions in the youngest age group (see Figure 2). The average overall pressure ulcer rate in men (2.0%, $n = 325\,293$) was significantly higher than in women ($n = 351\,110$, 1.6%) (OR: 1.282, 95% CI: 1.276-1.288, $P < 0.001$).

African Americans had a significantly higher rate (2.4%, $n = 119\,113$ out of 4 979 112) compared to all other races ($P < 0.05$). The rate in Caucasians was the second highest (1.8%, $n = 407\,006$ out of 22 621 329) followed by Native American (1.4%), Asian/Pacific Islander (1.3%), Hispanic (1.2%), and others (1.4%). The rate was highest in individuals with Medicare coverage (3.5%, $P < 0.05$). Patients with Medicaid had a rate of 0.8%; privately insured patients (0.6%) and self-pay patients (0.4%) had the lowest rates (see Table 2). Nonelective admissions had a significantly higher rate than elective admissions (1.9% versus 1.1%, $P < 0.001$).

Risk factors for pressure ulcers. Risk factors were identified by ICD-9 codes. The risk factor with the highest association with pressure ulcers was a diagnosis of malnutrition (11.5%, risk ratio [RR] = 8.45, CI: 8.41-8.5, $P < 0.001$). Other factors associated with the presence of pressure ulcers included hypotension (5.5%, RR = 3.56, CI: 3.53-3.58, $P < 0.001$), PVD (5.1%, RR = 3.22, CI: 3.19-3.24, $P < 0.001$), incontinence (5.4%, RR = 3.16, CI: 3.12-3.21, $P < 0.001$), CVD (3.8%, RR = 2.46, CI: 2.44-2.48, $P < 0.001$), diabetes (3.2%, RR = 2.38, CI: 2.37-2.39, $P < 0.001$), and fractures (2.7%, RR = 1.56, CI: 1.54-1.58, $P < 0.001$) (see Table 3).

Pressure ulcer site, stage, and debridement. Among 676 435 pressure ulcer patients, 540 073 (79.8%) had 1 recorded pressure ulcer, 105 383 (15.6%) had 2 pressure ulcers, and 30 979 (4.6%) had more than 2. The most common area for pressure ulcers was the patients' lower back (lower back/sacral/coccygeal areas per ICD-9) (47%); 17% were located on the patients' buttock, 14% on the heel, 9% other locations, and

5% on the hip. The ankle, upper back, elbow, and locations not otherwise specified each accounted for <5%. Of the 540 073 pressure ulcers identified, 79 026 (16%) were Stage 1, 191 308 (38%) were Stage 2, 101 093 (20%) were Stage 3, 97 083 (19%) were Stage 4, and 36 081 (7%) were unstageable according to ICD-9 coding (see Figure 3). The median stage of pressure ulcers was 2 for men, women, and Caucasians; the median stage in African Americans was 3. Persons concurrently suffering from malnutrition had a median stage of 3; persons with hypotension, PVD, incontinence, CVD, diabetes mellitus, and vertebral/femur fractures had a median stage of 2. A total of 71 418 excisional debridements

were performed in 65 582 patients; 5462 patients required multiple procedures.

Impact on patient outcomes.

LOS. The median LOS for individuals with at least 1 pressure ulcer was 7 days (mean 11.1 ± 15), compared to a median of 3 days (mean 4.6 ± 6.8) for patients without a pressure ulcer. Patients were significantly more likely to have a longer LOS (all *P* < 0.001) if they had the following risk factors: weight loss (regression coefficient [coef] = 4.88), paralysis (coef = 3.20), coagulopathy (coef = 2.04), congestive heart failure (CHF) (coef = 1.17), fluid/electrolyte disorder (coef = 1.70), and pulmonary/circulation disease (coef = 2.05).

Cost. Multiple factors contributed to hospital charges. The presence of a pressure ulcer increased costs. The median TC for persons with pressure ulcers was \$36 500 (mean \$72 000 ± \$122 900) compared to persons without pressure ulcers, whose median TC was \$17 200 (mean \$32 200 ± \$57 500). Increased hospital charges were significantly associated (*P* < 0.001) with LOS (coef = 8613), male gender (coef = 4464), African American race (coef = 3483), having private insurance (coef = 7643), or Medicaid beneficiaries (coef = 3729). The following comorbid conditions also significantly affected TCs: pulmonary/circulation disease (coef = 7062), PVD (coef 5887), obesity (coef 4229), hypotension (coef = 2530), and fluid/electrolyte disorders (coef = 3971).

Mortality. Patients with a pressure ulcer had a significantly higher mortality rate than patients without (9.1% versus 1.8%, OR = 5.08, CI: 5.03-5.1, *P* < 0.001); the latter also were more likely to be discharged home (72.5% versus 13.4%, OR = 5.42, CI: 5.39-5.45, *P* < 0.001), whereas patients with pressure ulcers were more likely to be transferred to a skilled nursing facility or intermediate care facility or require home health care (76.9% versus 24.7%, OR = 3.116, CI: 3.112-3.121, *P* < 0.001).

Table 2. Rate of admitted patients with pressure ulcers (PU) by gender, race, and insurance type in United States from 2008 to 2012

| | Total admissions (N) | PU cases (n) | Rate (%) | P value |
|------------------------|----------------------|--------------|----------|---------|
| Gender | | | | |
| Male | 16,366,959 | 325,293 | 2.0 | <0.0001 |
| Female | 22,645,567 | 351,110 | 1.6 | |
| Race | | | | |
| White | 22,621,329 | 407,006 | 1.8 | <0.0001 |
| African American | 4,979,112 | 119,113 | 2.4 | |
| Hispanic | 4,160,270 | 48,212 | 1.2 | |
| Asian/Pacific Islander | 911,360 | 11,914 | 1.3 | |
| Native American | 244,062 | 3,467 | 1.4 | |
| Other | 1,221,971 | 17,019 | 1.4 | |
| Insurance | | | | |
| Medicare | 14,891,815 | 515,233 | 3.5 | <0.0001 |
| Medicaid | 7,860,927 | 65,472 | 0.8 | |
| Private | 12,728,058 | 73,878 | 0.6 | |
| Self-pay | 2,019,929 | 8,343 | 0.4 | |
| No charge | 192,840 | 914 | 0.5 | |
| Other | 1,307,235 | 11,481 | 0.9 | |

Table 3. Pressure ulcer rate by select risk factors

| | With risk factor (ICD-9 code) | | Without risk factor | | P value |
|-----------------------------|-------------------------------|--------------------|---------------------|--------------------|---------|
| | Number | Incidence rate (%) | Number | Incidence rate (%) | |
| Malnutrition | 163,392 | 11.5 | 513,043 | 1.4 | <0.001 |
| Hypotension | 100,887 | 5.5 | 575,548 | 1.5 | <0.001 |
| Peripheral vascular disease | 74,579 | 5.1 | 601,856 | 1.6 | <0.001 |
| Incontinence | 20,787 | 5.4 | 655,648 | 1.7 | <0.001 |
| Cerebrovascular disease | 86,527 | 3.8 | 589,908 | 1.6 | <0.001 |
| Diabetes | 251,163 | 3.2 | 425,272 | 1.4 | <0.001 |
| Fracture | 16,799 | 2.7 | 659,636 | 1.7 | <0.001 |

Discussion

The National Pressure Ulcer Advisory Panel⁵ (NPUAP) defines a pressure ulcer as “localized 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.”

Although the NPUAP recently changed the term *pressure ulcer* to *pressure injury*, the term *pressure ulcer* is used throughout this article to

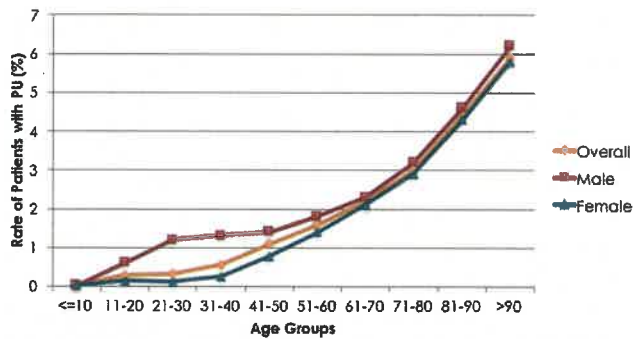


Figure 2. Rate of hospitalized patients with pressure ulcers across all age groups in the United States from 2008 to 2012 (National Inpatient Sample).

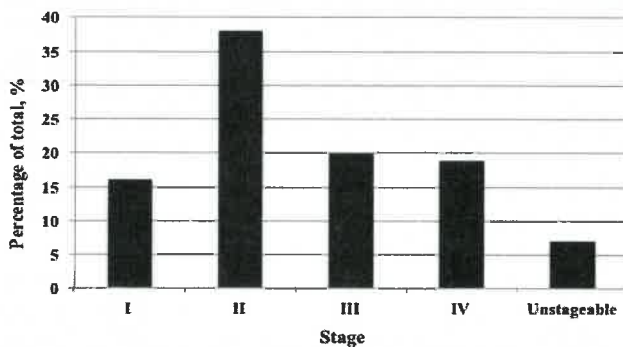


Figure 3. Stage of pressure ulcers in United States' inpatient admissions from 2008 to 2012 (National Inpatient Sample).

maintain consistency with the ICD-9 coding used during the study period.

Pressure ulcers occur in up to 23% of patients in long-term and rehabilitation facilities⁶ and at an incidence of 10% to 41% in ICU patients.^{7,8} The AHRQ² reported nearly 2.5 million individuals are affected by pressure ulcers, and more than 60 000 patients⁹ in the US die each year as a direct result of pressure ulcers. The costs associated with pressure ulcers are considerable. According to a 1996 prospective, year-long study looking at 30 patients and conducted by Xakellis and Franz,¹⁰ the incremental cost per pressure ulcer in the US was \$2731. Per a retrospective review,¹¹ the cost could be as high as \$59 000 if the ulcer was associated with osteomyelitis. Medicaid⁹ estimated each pressure ulcer adds \$43 180 in costs to an individual's hospital stay.

Pressure ulcers also have a significant impact on patient morbidity, mortality, and quality of life.² In their review of pressure ulcers in intensive care patients, Burdette and Kass¹² described pressure ulcers as one of the most expensive and debilitating diseases in the 20th century. In her review of risk factors and the assessment of pressure ulcer risk assessment, Braden¹³ indicated the complexity of the management and

treatment of pressure ulcers can greatly reduce the quality of life, resulting in a worldwide economic dilemma. Similarly, in their retrospective review of the 2003 NIS database, Fogerty et al¹⁴ demonstrated the complexity of pressure ulcer development and the multiplicity of contributing risk factors.

The overall incidence and prevalence of pressure ulcers have been shown in 2 reviews of the literature^{15,16} to remain high in the US despite the supposed improvement in the quality of health care in general and the drastic improvement in understanding these ulcers or the improvements in technologies available for prevention of ulcers specifically. The current study found the annual number of pressure ulcers reported in the US inpatient population was 670 767. Overall, the number of annual cases has remained constant at approximately 600 000 to 700 000 patients during the 5-year period of the study, with a rate of 1.8%. These numbers are lower than the 2.5 million reported by the AHRQ⁹ due to the fact the NIS database only accounts for those individuals with pressure ulcers who were hospitalized. Advances in pressure ulcer management have enabled these patients to receive treatment in outpatient care centers, with the goal of decreasing the duration of hospitalization, readmission rates, additional patient morbidity, and total care costs.⁹

In the opinion of the authors, the observed absence of a decrease in pressure ulcer rates may be attributed to: 1) failure or inadequate application of available prevention strategies during hospital admission or in other patient care environments, or 2) improvements in pressure ulcer assessment and reporting.

Risk factors. Conflict exists as to whether pressure ulcers result from factors largely dependent on caregivers or primarily from factors associated with patient morbidity. It is well established that pressure ulcers continue to be a common health problem, particularly among individuals with physical limitations or persons who are elderly and bedridden.

Age. Perneger et al¹⁷ evaluated the incidence of pressure ulcers (N = 2373) by conducting 3 cross-sectional surveys in a teaching hospital among patients with no pressure ulcer documented on admission. The authors assessed the development of pressure ulcers and the date on which the ulcer was documented and correlated this with diagnosis and reason for admission, among other factors. They found 247 new pressure ulcers occurred during admission (5.7 per 1000 person-days). The risk of pressure ulcer occurrence increased with age (11.2% of patients ages 70 to 79 years versus 34% in patients >90 years). Their results are consistent with the current study where the mean overall age of patients with a pressure ulcer was 71.2 years (68 years in men and 74 years in women), with increasing rates in older age groups. Jaul¹⁸ described pressure ulcers as chronic and healing in the geriatric population; the author indicated the presence of a pressure ulcer constituted a "geriatric syndrome" that was a result of multiple factors: immobility, poor nutrition, aging skin with poor elasticity, and numerous chronic diseases.

The US is experiencing a significant increase in the aging population. By the year 2050, the population 65 years of age and older will nearly double. This creates an urgent need for better prevention strategies and management of pressure ulcers.

Gender. In the current study, the prevalence of pressure ulcers was significantly higher in men (2%) than women (1.6%) (OR: 1.282, 95% CI: 1.276-1.288, $P < 0.001$). Inconsistent conclusions have been drawn as to which gender has a higher predilection for pressure ulcer development. The Waterlow score,¹⁹ developed in 1987 as a tool for pressure ulcer risk assessment, accommodates for research showing women are at a greater risk for development of a pressure ulcer; female gender is assigned a score of 2, where men are assigned a score of 1. This scoring system accounts for gender differences in the observation of predisposition of women to pressure ulcers in individuals who had femoral fractures.²⁰ Similar results were found in a 2-phase epidemiological study ($N = 327$ patients) by Bale et al²¹ that looked specifically at hospice patients. In addition, the authors found decreased pressure ulcer development with the use of a risk assessment tool and subsequent proactive measures such as offloading surfaces. Contrarily, in a prospective cohort study ($N = 258$), Primiano et al²² observed higher rates of pressure ulcers in men. The authors evaluated preoperative, intraoperative, and postoperative risk factors in cases where surgery lasted >3 hours; using bivariate and logistic regression analyses, male gender was found to be predictive of pressure ulcer development. The authors suggested the difference in distribution of adipose tissue in females was protective against pressure ulcer development. The current authors think the higher prevalence among male patients can be attributed to other associated risk factors as opposed to gender predilection. A multicenter cohort study of 3361 patients by Chen et al²³ noted the higher number of spinal cord injuries observed in men could result in more pressure ulcers when compared to women. Gibson et al²⁴ observed similar results in their 2002 qualitative study. The Braden scale²⁵ does not recognize any difference in pressure ulcer risk associated with gender, which is in accordance with other prevalence/incidence research.^{26,27} A review²⁸ of risk assessment found gender difference observed in pressure ulcer development also could be a reflection of the complexity of the overall problem, the multitude of causative events and comorbid conditions, and the lack of a universal risk assessment/classification tool.²⁷

Race/ethnicity. In the US, race/ethnicity, low socioeconomic status, occupation type, and education are consistently related to reduced access to quality health care. Previous studies have reported a significant association between race/ethnicity and pressure ulcer development: a qualitative study by Saladin and Krause²⁹ conducted among post spinal cord injury patients of varying racial-ethnic backgrounds (105 American Indian, 127 Caucasian, 122 Hispanic, 121 African American) found variability in access to treatment may lead

to minority populations experiencing higher pressure ulcer prevalence. A cross-sectional study³⁰ found African American ethnicity was a predictor of pressure ulcer recurrence. However, Furher et al³¹ found no differences in the prevalence of pressure ulcers between African Americans and Caucasians in the general population, although they reported African Americans suffered greater pressure ulcer-related mortality and suffered from more severe, higher-staged ulcers. An age-adjusted descriptive study using matched odds ratio comparisons by Redelings et al³² found African Americans had higher mortality from pressure ulcers than Caucasians. In general, African Americans tend to have less education and higher poverty rates at all ages compared to Caucasians, as shown by the survey analysis of health disparities using the US National Health and Nutrition Survey and follow-up interviews conducted by Farmer and Ferraro.³³ The results were no different in the current study: pressure ulcer incidence rates were higher in African Americans (2.4%, $n = 119$ out of 4 979 112) compared to all the other races ($P < 0.05$). Caucasians had the second highest incidence rate (1.8%, $n = 407$ out of 22 621 329) compared to other minority races such as Native American (1.4%), Asian/Pacific Islander (1.3%), Hispanic (1.2%), and other races (1.4%).

Insurance. In November 2008, the CMS instituted a policy to withhold reimbursement to acute care hospitals for the costs of treating hospital-acquired conditions such as pressure ulcers.³⁴ Despite these policies, pressure ulcer rates were the highest in persons with Medicare coverage (3.5%, $P < 0.05$). The prevalence rates for patients with Medicaid, private insurance, and self-pay patients were 0.8%, 0.6%, and 0.4%, respectively. The high rate of pressure ulcers in Medicare patients may be attributed to the fact that persons eligible for Medicare are 65 years or older, which subsequently places them at higher risk for development of pressure ulcers according to the literature and the current study results.

Comorbidities.

Nutrition. According to a descriptive analysis by Duncan³⁵ and a review by Lyder and Ayello,³⁶ immobility, inadequate nutrition, sensory deficiency, multiple comorbid conditions, circulatory abnormalities, dehydration, age, and incontinence are a few of the more than 100 factors identified as placing adults at risk for developing pressure ulcers. Of all the risk factors, malnutrition contributed the most significantly to pressure ulcer prevalence in the current study (11.5%, RR = 8.45, CI: 8.41-8.5, $P < 0.001$). Compromised nutritional status such as unintentional weight loss, undernutrition, protein energy malnutrition, and dehydration are known risk factors for pressure ulcer development.³⁷ As shown in a pilot study³⁸ and a retrospective cohort study,³⁹ additional nutrition-related risk factors associated with increased risk of pressure ulcers include low body mass index, reduced food intake, and impaired ability to eat independently. These factors might reflect poor health and self-care that is associated with higher incidence of pressure ulcers.

Tissue perfusion. Reduced tissue perfusion is known to play an important role in the development and chronicity of pressure ulcers. Mechanical loading may be significant enough to compromise the capillary circulation, causing ischemia and cell death in areas of pressure and, subsequently, ulcer development. Hypotension and PVD play a similar role. In a retrospective cohort study by Man et al,⁴⁰ hypotension was found to be an important risk factor for pressure ulcer development in the geriatric population. In the current study, rate of pressure ulcers was high in patients with hypotension (5.5%; RR = 3.56, CI: 3.53-3.38, $P < 0.001$) and PVD (5.1%; RR = 3.22, CI: 3.19-3.24, $P < 0.001$).

pH. The pH of normal skin (pH 5.4–5.9) has a bactericidal effect and limits the growth of pathogenic organisms. In the event of urinary incontinence, due to reasons such as CVD, urinary urea decomposes on the skin to form ammonium hydroxide, which raises the skin pH and favors bacterial proliferation. A descriptive study by Leveen et al⁴¹ also shows high pH also negatively impacts the delivery of oxygen to damaged tissue, making wound healing even more challenging. Fecal incontinence also can cause skin irritation and breakdown. The current study showed patients with CVD had a pressure ulcer rate of 3.8% (RR = 2.46, CI 2.44-2.48, $P < 0.001$), and the rate was 5.4% rate (RR = 3.16, CI: 3.12-3.21) among persons with incontinence.

Diabetes and fractures. Other factors that may contribute to pressure ulcer development include diabetes and fractures. Blood sugar control is known to play an important role in wound healing. Persons with diabetes are at risk for developing both pressure ulcers and diabetic foot ulcers, owing to the neuropathy and tendency for unnoticed trauma. In the current study, the rate of pressure ulcers among patients with diabetes was 3.2% (RR = 2.38, CI 2.37-2.39, $P < 0.001$). A retrospective review of data⁴² found fractures that result in immobilization, such as hip⁴² and femur²⁰ fractures, predispose to the development of pressure ulcers. In the current study, patients with fractures had a pressure ulcer rate of 2.7% (RR = 1.56, CI 1.54-1.58, $P < 0.001$).

Ulcer quantity, site, and stage. Documenting the number, site, and stage of ulcers is crucial in pressure ulcer management. Among the 676 435 patients studied in this NIS database, 540 073 (79.8%) had 1 ulcer recorded, 105 383 (15.6%) had 2 pressure ulcers, and 30 979 (4.6%) had more than 2 ulcers. Bony prominences are more susceptible to pressure ulcers as a result of deformation of deep tissues and muscle atrophy. A review of the literature⁴³ found shear force and friction injury to skin surfaces create a process in which the epidermal and dermal layers adhere to bed surfaces causing destructive events to the underlying areas of the skin. The most vulnerable pressure points depend on the position in which most of the patient's time is spent. The current data showed a majority (47%) of the pressure ulcers were located on the patients' lower backs (sacrum). Excessive moisture results in hyperhydration of the skin, rendering it more vulnerable to dermal erosion.

The high percentage of lower back pressure ulcers could be an amalgamation of the influence of shear force, friction, and moisture in patients who are bedbound. Patients also had pressure ulcers on the heel (14%), hip (5%), and other locations (9%). The ankle, upper back, elbow, and locations not otherwise specified each accounted for <5%. A systematic review of the literature⁴⁴ (including 3 RCTs and 1 economic study [N = 502]) recommends manual repositioning of these patients for both treatment and prevention of pressure ulcers. However, the ideal repositioning regimen and frequency has yet to be determined, per a systematic review by Moore and Cowman.⁴⁵

Pressure ulcer staging affects both treatment and prognosis. The NPUAP⁴⁶ redefined the stages of pressure ulcers in 2007 to include the original 4 stages plus 2 additional stages (deep tissue injury and unstageable). The reported incidence of pressure ulcers Stage 2 or greater is between 8.1% and 12.9%.^{47,48} The current data analysis showed both male and female patients had a median pressure ulcer Stage 2. Among Caucasian and other populations, the median stage of pressure ulcers was Stage 2, whereas the median stage for the African American population was Stage 3. The advanced stages seen in African Americans may be attributed to poor general condition or due to difficulty in detecting early pressure ulcers stages due to the clinical difficulty in observing erythema and blanching response in dark skin. The current authors observed patients who suffer from malnutrition had a median ulcer stage of 3, and patients with diabetes, CVD, incontinence, PVD, and hypotension had a median ulcer stage of 2. This could be attributed to frequent surveillance for pressure ulcers in these high-risk patients in nursing homes and hospitals.

For Stage 1 and Stage 2 pressure ulcers, wound care typically does not involve surgery. Management at these stages may involve topical therapy, offloading, and optimization of nutrition/moisture management, as well as proper management of the underlying cause. For Stage 3 and Stage 4 ulcers, surgical intervention may be required, although a review⁴⁹ has shown some of these lesions might be treated conservatively due to coexisting medical problems. Generally, excisional surgical debridement is the standard care for higher-stage pressure ulcers because these often present with necrosis. In the current data, a total of 71 418 excisional debridement procedures were performed in 65 582 patients; of those, 5462 patients required multiple debridement procedures.

LOS. According to a limited literature review,⁵⁰ prolonged LOS is a significant predictor of functional decline in elderly individuals during hospitalization. A 9-year, prospective observational registry study of 275 pressure ulcers by Lardenoye et al⁵¹ showed 5.5% of all pressure ulcers resulted in prolonged hospitalization and found a strong correlation among pressure ulcer development, reason for hospital admission, gender, and age. In a cross-sectional, observational study (N = 2000), Graves et al⁵² concluded the presence of

pressure ulcers was a significant independent contribution to excess length of hospitalization. LOS has been shown to be prolonged an average range of 4 to 6 days, including in the descriptive, comparative study of 2 cross-sectional pressure ulcer surveys^{52,53} by Gunningberg and Stotts.⁵⁴ The current study shows the median LOS for individuals with at least 1 pressure ulcer was 7 days (mean 11.1 ± 15) compared to a median of 3 days (mean 4.6 ± 6.8) in patients without pressure ulcers. Patients with significant weight loss, paralysis, coagulopathy, CHF, fluid and electrolyte disorders, and pulmonary and circulation diseases with concurrent diagnosis of at least 1 pressure ulcer were more likely to have a longer LOS.

Cost. Prolonged LOS not only affects the morbidity and mortality of patients, but it also has a significant impact on hospital charges. The current study estimated the median TC to be \$17 200 (mean $\$32\,200 \pm \$57\,500$) in patients without pressure ulcers. In contrast, the median hospital charges for patients with pressure ulcers were significantly higher at \$36 500 (mean $\$72\,000 \pm \$122\,900$, $P < 0.001$). Along with pressure ulcers, increased LOS (coef = 8613), male gender (coef = ,464), African American race (coef = 3483), private insurance beneficiaries (coef = 7643), and Medicaid beneficiaries (coef = 3729) significantly impacted total hospital charges. Other conditions that contributed to increased hospital charges were pulmonary/circulation disease (coef = 7062), PVD (coef = 5887), obesity (coef = 4229), hypotension (coef = 2530), and fluid and electrolyte disorders (coef = 3971).

Mortality. Pressure ulcers play a significant role in influencing the mortality rate among hospitalized patients and patients in nursing facilities. A retrospective study by Lyder et al⁵⁵ showed that of 3000 individuals who entered the hospital with a pressure ulcer, 16.7% developed at least 1 new pressure ulcer during their stay. The odds of any patient dying in the hospital were 2.8 times higher if the patient had a pressure ulcer. In the current study, the mortality rate in patients with pressure ulcers was significantly higher than in patients without pressure ulcers (9.1% versus 1.8%, OR = 5.08, CI: 5.03-5.1, $P < 0.001$). The current data analysis also showed 72.5% of patients without pressure ulcers were discharged home compared to 13.4% patients with pressure ulcers (OR 5.42, CI: 5.39 – 5.45, $P < 0.001$). Furthermore, 76.9% patients with pressure ulcers were transferred to a skilled nursing facility or intermediate care facility or required home health care compared to 24.7% patients without pressure ulcers (OR = 3.116, CI: 3.112-3.121, $P < 0.001$). This observation is consistent with previous reviews.^{56,57} The additional fees for skilled nursing facilities add to the increase in health care expenditure on treatment and management of this preventable health issue.

Limitations

The results of this study are limited by the inherent limitations of retrospective analysis of administrative data, which includes the risk of erroneous coding or missing data. Given

that ICD-9 codes were used, some error in pressure ulcer diagnosis and description of coding can be assumed. On the other hand, the large sample size is expected to minimize errors associated with data recording and should not interfere with the general conclusion. Also, the authors could not differentiate between pressure ulcers that were hospital-acquired and present on admission; although the aim of the study was not to delineate between them, knowledge about their onset would have enhanced overall conclusions, especially in terms of deep tissue injury and its sometimes slow clinical appearance. Readmission rates, which were not available for this investigation, also may have inflated the number of patients with pressure ulcers. Lastly, while the data facilitated the categorization of patients without pressure ulcers, this may minimally weight the data, because patients at risk are more likely to develop more than 1 pressure ulcer.

Conclusion

The results of this study show the rate of pressure ulcers among patients in US acute care hospitals was relatively stable from 2008 until 2012 (average 1.5%). Patients who had a pressure ulcer had a significantly longer LOS, higher in-hospital mortality rate, and higher TC than patients without a pressure ulcer. Patients with pressure ulcers were also more likely to be discharged to a skilled nursing facility, while those individuals without pressure ulcers were more likely to be discharged home. The ICD-9 code associated with a significantly higher risk of having a pressure ulcer was for malnutrition. Pressure ulcers have been recognized as being a public health issue in the US that contributes greatly to national health care expenditures. Early treatment and a reduction of pressure ulcer rates have been set as goals by the CMS. The Institute of Healthcare Improvement has created the 5 Million Lives Campaign; 1 of the main goals is to use science-based guidelines for prevention. It is crucial that the severity of this issue be recognized and that health care centers develop an interdisciplinary approach to the delivery of coordinated, high-quality care to patients with, or at risk for, developing pressure ulcers. ■

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Skin tears: Prediction, prevention, assessment and management

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Abstract

Skin tears are a significant problem for patients and the nurses who treat them. Estimates of their prevalence differs around the world, but there is strong evidence to suggest that they occur more frequently than pressure ulcers. In the past few years there has been an increased focus and research into skin tears, and the International Skin Tear Advisory Panel has developed internationally recognised best practice recommendations in this important field for the global wound care community. This article will review the most current research and best practice recommendations for the prediction, prevention, assessment and treatment of skin tears.

Key words: Skin tears; skin tear assessment; skin tear management; skin tear prevention

Skin tears are a common, but largely unrecognised acute wound that if left untreated can become chronic, particularly if they occur on the lower limb (Baronoski et al, 2011; Stephen-Haynes and Carville, 2011). In individuals who are acutely ill or who have several chronic diseases, skin tears can become both chronic and complex and can result in misdiagnoses and mismanagement leading to complications such as pain, infection, delayed wound healing and increase costs to the healthcare system.

According to the International Skin Tear Advisory Panel (ISTAP) (LeBlanc et al, 2011:1):

'A skin tear is a wound caused by shear, friction, and/or blunt force resulting in separation of skin layers. A skin tear can be: partial-thickness (separation of the epidermis from the dermis) or full-thickness (separation of both the epidermis and dermis from underlying structures).'

There is now an increasing body of evidence to guide the prevention, assessment and treatment of skin tears. This has been led mainly by ISTAP, which was formed to raise international awareness of the prediction, assessment, prevention, and management of skin tears. ISTAP includes a broad range of health professionals representing: North America, South America, Europe (including the UK), Asia, the Middle East, Australia/New Zealand, and Africa (<http://www.skintears.org/>). The purpose of this article is to present the existing national and international literature relating to the prediction, prevention, assessment and management of skin tears, which can be used to by clinicians to inform clinical practice.

Prevalence of skin tears

The prevalence of skin tears prevalence in long-term care facilities has been identified at 10–54% (Everett and Powell, 1994; LeBlanc et al, 2013a; Carville and Smith, 2004; McErlean et al, 2004; Woo and LeBlanc, 2014), and 4.5–19.5% in all age groups in the community (Carville and Smith, 2004; LeBlanc et al, 2008; Strazzieri-Pulido et al, 2015). While in acute care prevalence ranges from 3.3–22% (Amaral et al, 2012;

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Strazziari-Pulido et al, 2015), and 30% in palliative care settings (Maida et al, 2012). In the paediatric acute care setting, one study reported a skin tear prevalence of 17% (McLane et al, 2004). A recent audit of in-patients in acute hospitals across Wales identified a prevalence of 2.57% (Clark et al, 2017).

Predisposing risk factors

Skin tears are more prevalent with, but not limited to, the extremes of age, for example the physiological characteristics of neonatal/infant skin may affect the skin's ability to resist shear, friction and/or blunt force (LeBlanc et al, 2011). Infant skin is not fully matured and remains at high risk of skin tears until 3 years of age. Furthermore, skin changes related to extremes of age and those who are critically or chronically ill put these patients at higher risk (LeBlanc et al, 2011). Dry skin (xerosis) in combination with advancing age

Table 1. Modifiable and non-modifiable risk factors for skin tears

| Modifiable risk factors | Non-modifiable risk factors |
|--|--|
| Xerosis Pruritus Types of medical adhesives used Care during activities of daily living Falls risk Medications Nutritional status Trauma Health professionals' approach to managing individuals with aggressive behaviour/cognitive impairment | Photo-ageing Skin changes with ageing including dermatoporosis Critical and chronic illness Dementia/cognitive impairment Visual/auditory/sensory impairment Aggressive behaviour Requiring assistance with activities of daily living |

From: LeBlanc et al (2013b)

Table 2. ISTAP guide for the prevention of skin tears

| Risk factor | Individual | Caregiver/provider |
|----------------|--|--|
| General health | <ul style="list-style-type: none"> • Self-management approach (if cognitive function not impaired) • Educate patient on skin tear prevention and promote active involvement in treatment decisions (if cognitive function is not impaired) • Optimise nutrition and hydration • Educate patients on medicine-induced skin fragility (e.g. topical and system steroids) | <ul style="list-style-type: none"> • Safe patient environment • Educate client and/or circle of caregivers • Protect from self-harm • Dietary consult if indicated • Extra caution with extremes of body mass index (<20 or >30kg/m²) • Review polypharmacy for medication reduction/optimisation • Medical review of comorbidities for improved management • Educate caregivers on gentle patient handling • Educate caregivers on skin fragility with extremes of age • Educate caregivers on medicine-induced skin fragility (e.g. topical and system steroids) |
| Mobility | <ul style="list-style-type: none"> • Encourage active involvement if physical function not impaired • Appropriate selection and use of assistive devices | <ul style="list-style-type: none"> • Daily skin assessment and monitor for skin tears • Ensure safe patient handling techniques/equipment and environment (trauma, activities of daily living, self-injury) • Physical therapy consult to assess and improve mobility and assist with safe transfers • Proper transferring/ repositioning • Initiate fall assessment and prevention program • Remove clutter • Eliminate scatter rugs • Ensure proper lighting • Pad equipment/furniture (bed rails, wheel chair, etc.) • Assess footwear • Educate caregivers to lift rather than pull • Use protective clothing/devices, e.g. stockinette, long sleeves, shin guards |
| Skin | <ul style="list-style-type: none"> • Awareness of medication-induced skin fragility (e.g. topical and system steroids) • Wear protective clothing (shin guards, long sleeves, etc) • Moisturise skin (lubrication and hydration) twice a day • Keep fingernails short | <ul style="list-style-type: none"> • Individualise skin hygiene (warm, tepid, not hot, water; soapless or pH-neutral cleansers; moisturise skin twice a day) • Avoid strong adhesives, dressings, tapes • Avoid sharp fingernails/jewelry with patient contact • Use room humidifier if air dry • Maintain a room temperature that is not too hot • Control oedema |

From: LeBlanc et al (2013b)

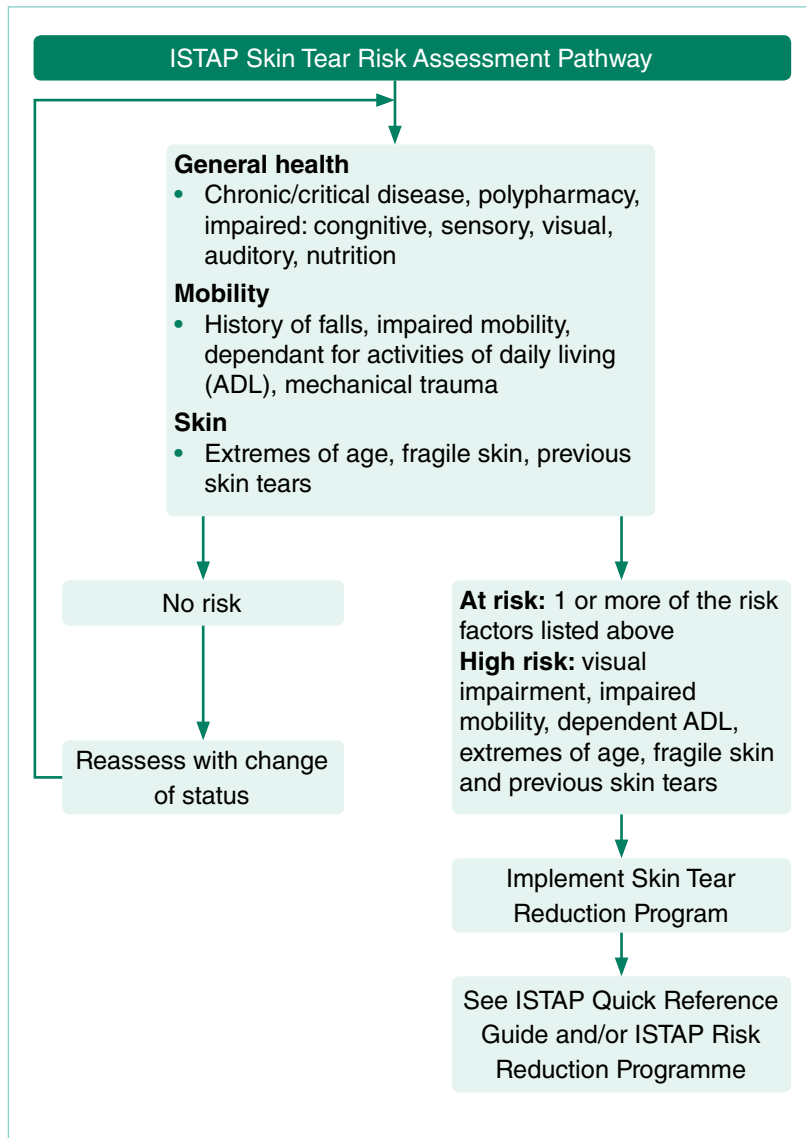


Figure 1. ISTAP Skin Tear Risk Assessment Pathway (LeBlanc et al, 2013b)

results in pruritus (Carville et al, 2014), which leads to itching due a decrease in skin moisture. This type of mechanical trauma can predispose the patient to skin tears (White et al, 1994). Changes with ageing can also decrease sensation and lead to increased risk of mechanical trauma, this in combination with associated comorbidities may result in delayed wound healing (McGough-Csarny and Kopac, 1998). Dermatoporosis is a term that has been developed to group the common skin changes that occur with advanced ageing (Kaya and Saurat, 2007; 2013). These include ecchymosis, senile purpura, haematoma and stellate pseudoscars. In clinical practice it can be difficult to differentiate between these skin changes.

LeBlanc et al (2013b) have identified modifiable and non-modifiable risk factors that can help to prevent the risk of skin tears (Table 1). These

risk factors need to be addressed in any plan for preventing skin tears (All Wales Tissue Viability Forum, 2015).

Skin tear prevention

Prevention and/or reduction of the incidence of skin tears is possible, but it requires the involvement of both the individual patient and caregivers to increase the likelihood of success. ISTAP has developed a table as a quick reference guide for reducing the risk and preventing skin tears (Table 2). While a number of intrinsic factors contribute to an increased risk of skin tears (e.g. dry, thin, inelastic tissue; impaired cognition; agitation; impaired nutrition), there are also extrinsic factors (Table 3) that can be managed to reduce the risk and prevent skin tears (Stephen-Haynes and Carville, 2011). ISTAP has also developed a ISTAP Skin Tear Risk Assessment Pathway, which identifies those at risk and link the patients at risk to a prevention programme (Figure 1.) Education of the patient and caregivers is important as is management of the environment to enhance patient safety (Stephen-Haynes and Carville, 2011).

Skin tear assessment

Reliable and accurate wound descriptions and documentation are essential components of any wound assessment (Stephen-Haynes and Carville, 2011). As part of the daily assessment of an individual, nurses should look for the presence of any lesions including skin tears. One resource that nurses can use is the Data Collection Tool developed by ISTAP, which is part of the Tool Kit (LeBlanc et al, 2013b). This tool has seven components that need to be assessed for each skin tear.

In addition, the measurement of each skin tear, drainage amount and colour should be documented (Stephen-Haynes, 2013). Each healthcare setting and agency needs to develop a protocol that all health professionals follow to ensure consistency in the assessment of skin tears. Assessment data can provide the healthcare provider a mechanism to communicate, improve continuity among disciplines and establish appropriate treatment modalities.

Skin tear management

Skins tears should be managed with the same principles as other wounds (Baranoski et al, 2016), using a systematic and holistic approach (Clothier, 2014). The key areas include:

- Primary prevention is considered the key to management of skin tears. Introduce and document a prevention plan to prevent further trauma. Educate the patient and circle of care in prevention and management of skin tears
- Identify and treat the cause. Remove or minimise the cause of the skin tear (Le Blanc et al, 2011; Baranoski et al, 2011). For example, if the cause is secondary

to falls, a falls prevention programme needs to be implemented

- Address patient and family centred concerns. Control pain by offering analgesia (Beldon, 2008). Acknowledge and address the patient and family beliefs, cultural and psychological variables while formulating a management plan (Le Blanc et al, 2011). Minimise the negative influence of the skin tear on the individual and their care givers to improve activities of daily living and quality of life
- Determine the healing potential of the skin tear. When the skin tear occurs on the lower limb, it is essential to take a comprehensive clinical history and vascular assessment, such as using a Doppler ultrasound to obtain the ankle brachial pressure index, or viewing the wave form to rule out any significant peripheral vascular disease; before applying compression therapy to manage any peripheral oedema in either arms or legs (Erwin-Toth and Stenger, 2007)
- Local wound care. Remove any existing dressing without damaging the peri-wound and interrupting the healing process. Control bleeding by applying pressure and elevating the limb if applicable. An initial dressing selection may be one that promotes clotting, such as a calcium alginate. Cleanse the wound with a non-cytotoxic solution, e.g. normal saline or potable water, irrigating the wound at a pressure less than 8psi to remove blood clots and debris from the skin flap (LeBlanc et al, 2008). ISTAP also suggest that the principles of Wound Bed Preparation and TIME be used to guide wound assessment (Sibbald et al, 2011; Schultz et al, 2003). These principles include:

Tissue/debridement

Debride non-viable skin flaps. Realign the viable skin flap (the pedicle), approximating the wound edges, to the extent possible. *Figures 2–7* show the procedure for cleansing the wound and re-approximating the flap edges using a moistened swab.

Skin tears are considered to be acute wounds that have the potential to be closed by primary intention (LeBlanc et al, 2016). Sutures and staples are not recommended due to the fragility of elderly skin (Le Blanc et al, 2011). Traditional adhesive strips are no longer advised due to their adhesive nature increasing risk of skin injury (LeBlanc et al, 2016; Stephen-Haynes and Callaghan, 2017). Therefore, other methods of wound closure may need to be considered such as topical skin glue i.e. cyanoacrylates (Le Blanc et al, 2016).

Infection

Skin tears do not generally need to be debrided. However, the wound bed needs to be thoroughly cleaned. Covert and overt infection should be managed with a topical antimicrobial and spreading and systemic

Table 3. Skin tear assessment

| | |
|--|---|
| Location | Hands Arms Legs Feet Head/face Trunk Abdomen Buttocks Other |
| Type (using the ISTAP Classification System) | Type 1 – no skin loss Type 2 – partial flap loss Type 3 – total flap loss |
| Cause | During activities of daily living Blunt force trauma From a fall Adhesive/tape injury Resisting care/agitation Unknown |
| Where did the skin tear occur? | Critical care Acute care Long-term care Home care Rehab Palliative care Other |
| Facility acquired | Yes No |
| Intrinsic factors | Senile purpura Echymosis Haematoma Presence of oedema Inability to reposition independently Topical steroid use Systemic or long-term steroid use Anticoagulants Chemotherapy agents Co-existing pressure ulcer Fecal or urinary incontinence |
| Extrinsic factors | Removal of tape or stockings Inadequate nutrition Polypharmacy Using assistive devices Blood draws Transfers and/or falls Prosthetic devices Skin cleansers |

From: LeBlanc et al (2013b)

infection with systemic and topical antimicrobials (International Wound Infection Institute, 2016). Tetanus immunoglobulin should be administered if the patient has not been vaccinated with tetanus toxoid in the past 10 years, before debridement to prevent the potential release of exotoxin (Carden and Tintinalli, 2004).

Moisture

It is important to promote moist wound healing, ensuring moisture balance and preventing peri-wound skin maceration. The peri-wound should be protected with a skin barrier product.

Epidermal margins

Skin tears should typically follow acute wound healing trajectory of 7–14 days (Le Blanc et al, 2008). If healing takes longer, re-evaluate. Refer to a wound care specialist when the skin tear is infected or extensive (Le Blanc et al, 2016).

Dressing selection

Following a systematic review by ISTAP and a wider consultation of an international group of health professionals, recommendations were suggested for the selection of dressings (*Appendix 1*) (Le Blanc et al, 2016). From the consensus process dressings such as films and hydrocolloids were not included in the recommendations due to their adhesive properties. Iodine dressings were also omitted due to their drying nature. Furthermore, honey dressings were left out due to the high risk of peri-wound maceration, although more recent evidence has suggested that leptospermum honey-based dressings have been reported to be effective without causing maceration (Johnston and Katzman, 2015).

Reassessment of the skin tear should take place approximately every 3-7 days and careful consideration given to whether the dressing needs changing. If a patient has very fragile skin it is preferable to leave the dressing in place for up to 5 days to avoid further trauma to the skin flap (Stephen-Haynes, 2013). Where a change of dressing is required, and to promote flap viability, the dressing should be removed in the direction of the pedicle, rather than against it.

Specifying the skin tear classification (*Figure 8*), size and shape and the direction for dressing removal on the dressing can be a useful visual means of conveying this information (Holloway and Le Blanc, 2017).

Conclusion

Skin tears are largely unrecognised acute wounds, that can become chronic and complex if not assessed and treated. ISTAP recommend the use of guiding principles such as WBP and TIME to guide assessment and management. This article reviewed the prediction, prevention, assessment and management of skin tears. There are still gaps in the evidence with regards to the prevalence, incidence, population specific risk factors and prevention strategies for skin tears. Currently, treatment recommendations are largely based on expert opinion; therefore, more research is needed to identify how treatment options impact on the healing of skin tears.



Figure 2. Cleanse the wound



Figure 3. Use two moistened cotton tipped swabs to begin to re-approximate the edges



Figure 4. Slowly re-approximate the edges



Figure 5. Slowly re-approximate the edges



Figure 6. Once the edges are re-approximated pat the skin flap on the wound bed to ensure adhesion



Figure 7. Final outcome of the process of re-approximating the edges

Key Points

- Skin tears are a common, but largely unrecognised acute wound that if left untreated can become chronic and complex
- Healthcare facilities should implement a skin tear prevention programme
- Educate both healthcare providers and patients/families on the prevention and treatment of skin tears
- Use the ISTAP website for the most up-to-date information on skin tears skintears.org

CPD reflective questions

- What is the prevalence of skin tears in the palliative care setting?
- Identify five modifiable risk factors for skin tears?
- Define what a type 2 skin tear is? How would you deal with this in practice?

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ISTAP skin tear classification



Type 1: no skin loss

Linear or flap tear which can be repositioned to cover the wound bed

Type 2: partial flap loss

Partial flap loss which cannot be repositioned to cover the wound bed

Type 3: total flap loss

Total flap loss exposing entire wound bed

Figure 8. ISTAP skin tear classification (LeBlanc et al, 2011) (Used with permission)

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Appendix 1. ISTAP Skin tear product selection recommendations

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| Product categories | Indications | Skin tear type | Considerations |
|--|---|----------------|---|
| Hydrogels | Donates moisture for dry wounds | 2, 3 | <ul style="list-style-type: none"> ● Caution: may result in peri wound maceration if wound is exudative ● For autolytic debridement in wounds with low exudate ● Secondary cover dressing required |
| Foam dressing | Moderate exudate Longer wear time (2-7 days depending on exudate levels) | 2, 3 | <ul style="list-style-type: none"> ● Caution with adhesive border foams, use non-adhesive versions, when possible, to avoid peri-wound trauma |
| Non-adherent mesh dressing | Dry or exudative wound | 1, 2, 3 | <ul style="list-style-type: none"> ● Maintains moisture balance for multiple levels of wound exudate ● Atraumatic removal ● May need secondary cover dressing |
| 2-octyl cyanoacrylate topical bandage (skin glue) | To approximate wound edges | 1 | <ul style="list-style-type: none"> ● Use in a similar fashion as sutures within first 24 hours post injury, relatively expensive, medical directive/protocol may be required |
| Acrylic dressing | Mild to moderate exudate without any evidence of bleeding, may remain in place for an extended period of time | 1, 2, 3 | <ul style="list-style-type: none"> ● Care on removal ● Should only be used as directed and left on for extended wear time |

Appendix 1 continued. ISTAP Skin tear product selection recommendations © ISTAP 2015

| Product categories | Indications | Skin tear type | Considerations |
|--|--|----------------|--|
| Calcium alginates | Moderate to heavy exudate hemostatic | 1, 2, 3 | <ul style="list-style-type: none"> ● May dry out wound bed if inadequate exudate ● Secondary cover dressing required |
| Hydrofibre | Moderate to heavy exudate | 2, 3 | <ul style="list-style-type: none"> ● No hemostatic properties ● May dry out wound bed if inadequate exudate ● Secondary cover dressing required |
| Special consideration for infected skin tears | | | |
| Ionic silver dressings | Effective broad spectrum antimicrobial action including antibiotic-resistant organisms | 1, 2, 3 | <ul style="list-style-type: none"> ● Should not be used indefinitely ● Contraindicated in patients with silver allergy ● Use when local or deep infection is suspected or confirmed ● Use non-adherent products whenever possible to minimise risk of further trauma |
| Methylene blue and gentian violet dressings | Effective broad spectrum antimicrobial action including antibiotic-resistant organisms | 1, 2, 3 | <ul style="list-style-type: none"> ● Non-traumatic to wound bed ● Use when local or deep tissue infection is suspected or confirmed ● Secondary dressing required |

This produce list is not all-inclusive; there may be additional products applicable for the treatment of skin tears.
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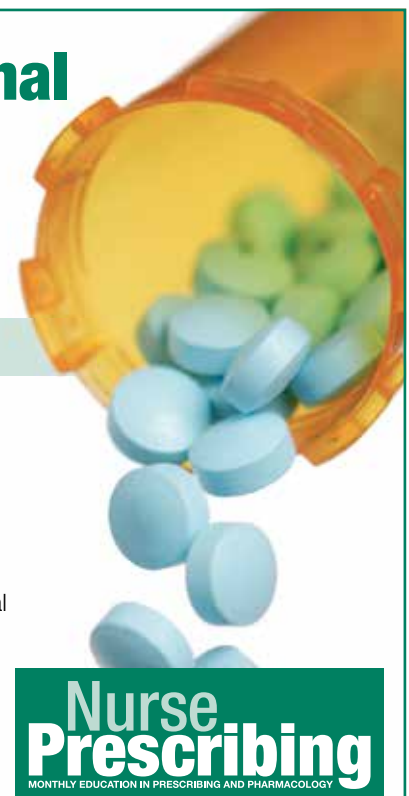
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Venous Ulcers

Jane E. Carmelt¹**OBJECTIVES**

1. Discuss venous ulcers in terms of etiologic factors, risk factors, assessment, diagnostic criteria, pathophysiology, typical presentation, and principles of management.
2. Describe Laplace's law in predicting sub-bandage pressure and the level of compression applied to the lower leg.
3. Explain the mechanism of action underlying effective compression therapy for the individual with chronic venous insufficiency.
4. Discuss considerations for use of inelastic compression, short-stretch bandages, long-stretch bandages, compression stockings, and intermittent pneumatic compression.
5. Identify adjunctive therapies that may be of benefit to the patient with a venous ulcer.
6. List three key points to teach a patient being treated with a multilayer compression wrap.

Chronic venous disorders include a wide range of morphologic and functional abnormalities of the venous system, that includes mild conditions (e.g., uncomplicated telangiectasias and varicose veins) to complex conditions (e.g., deep vein thrombosis, and venous ulcers). The majority of chronic venous disorders exist in the healthy patient population. The term chronic venous disease is used when referring to the subset of chronic venous disorders that are more complicated; those that are associated with signs and symptoms significant enough to require medical care, such as deep vein thrombosis, chronic venous insufficiency, and venous ulcers. Lower extremity venous disease (LEVD) is synonymous with chronic venous disease. Chronic venous insufficiency (CVI) refers to leg-related manifestations of venous hypertension and functional abnormalities of the venous system (edema, skin changes, ulceration) (Meissner, 2009). Venous ulcers are the most common lower extremity ulcers, accounting for 70% to 90% of all leg ulcers (WOCN Society, 2005). These lesions develop as a result of skin and tissue changes caused by CVI and the associated ambulatory venous hypertension.

Management of patients with venous ulcers must include measures to optimize wound healing through reduction of edema, prevention of complications, and

appropriate topical therapy to promote healing (de Araujo et al, 2003; Moffat et al, 2007; Robson et al, 2006). Once the ulcer is healed, the emphasis shifts to long-term disease management and prevention of recurrence.

EPIDEMIOLOGY

The exact prevalence of venous ulcers is not known, although prevalence estimates in developed countries range from less than 1% to greater than 3% of the population (Bolton, 2008; Kerstein, 2003). Venous disease and venous ulcers occur in individuals as young as 20 years. "Peak" incidence occurs between the ages of 60 and 80 years (de Araujo et al, 2003). Although no racial predilection is apparent, most studies report female gender is a risk factor (de Araujo et al, 2003; Kalra and Gloviczki, 2003). In addition, an increased incidence of obesity is seen in 25% of patients with venous ulcers (Benigni et al, 2006).

LEVD affects approximately six to seven million individuals in the United States, and approximately one million of these persons will develop ulcerations (WOCN Society, 2005). The impact of venous disease is tremendous as it relates to the individual and costs to the health care system and society. Individuals with venous disease report pain, itching, anxiety, social isolation, and reduced ability to perform usual activities as their areas of greatest concern (de Araujo et al, 2003). In contrast, nurses caring for these patients rated pain control as a less important aspect of care than wound healing and limb preservation, which indicates the need for increased awareness and focus on quality-of-life issues on the part of health care providers (Ryan et al, 2003).

¹The author and editors acknowledge Rhonda Holbrook and Dorothy Doughty for their work on developing content of this chapter in the third edition of *Acute & Chronic Wounds: Nursing Management*. Many of the concepts and comments they developed in the previous edition are reflected in this chapter. Their significant contribution is well appreciated.

Approximately \$2.5 to \$3.5 billion is spent annually on management of venous ulcers (Bolton, 2008). The average lifetime cost of care for an individual with LEVD can exceed \$40,000 (Weingarten, 2001). Cost per occurrence in the home care or wound clinic setting is estimated at \$1,621 to \$3,279 without calculating lost wage time (Bolton et al 2006; Korn et al, 2002; McGucklin et al, 2002).

The negative impact of venous ulcers is compounded by recurrence rates of 26% to 28% in the first year and is as high as 76% within 3 to 5 years, which reflects the chronicity of the underlying condition (Bolton et al, 2006; Castonguay, 2008) Frequent recurrence is attributed to a failure to adequately address the primary problems of venous insufficiency and venous hypertension (WOCN Society, 2005).

VENOUS STRUCTURE AND FUNCTION

The veins of the lower extremity venous system include deep veins, superficial veins, and perforator veins. The deep veins include the posterior and anterior tibial and the peroneal veins; these veins are located in the deep tissue adjacent to the calf muscle. The superficial venous system is also known as the *saphenous system* because the two major vessels are the greater saphenous vein and lesser saphenous vein. These two vessels are located just below the superficial fascia and have multiple tributaries located in the superficial tissues (Figure 12-1) (Kalra and Głowiczki, 2003). The perforator veins “connect” the two systems, transporting blood from the superficial system into the deep system, from which point the blood is propelled back to the heart. The number of perforator veins per leg can vary greatly; the typical patient can have 200 perforator veins below the knee and 20 above the knee (Hussein, 2008).

Veins fill normally via slow capillary inflow, which takes more than approximately 20 seconds. All veins are equipped with one-way valves that support a unidirectional flow of blood toward the heart. Because these valves prevent reflux of blood from the high-pressure deep venous system to the low-pressure superficial venous system, they play an essential role in normal venous function. Further protection is provided by the fact that the perforator veins follow an oblique course through the fascia and muscle layers, which provides additional support for the connecting veins and their valves. The closed valves in the perforator veins prevent transmission of the high resting pressures back into the superficial system, so long as the valves remain competent (Kalra and Głowiczki, 2003). Approximately 50% to 60% of patients with venous ulcers have incompetent superficial and perforator vein valves (Agren and Gottrup, 2007).

Returning blood from the feet and legs to the heart is a major physiologic challenge because the blood must flow “uphill” against the forces of gravity. When an individual is standing upright, the gravitational force

creates a column of hydrostatic pressure of approximately 90 mm Hg at the ankle. The primary mechanisms by which venous blood is returned to the heart are the smooth muscle tone within the venous walls, the contraction of the calf muscles (gastrocnemius and soleus), and the negative intrathoracic pressure created during inspiration. Of these three mechanisms, contraction of the calf muscle pump is by far the most essential (Meissner, 2009):

The calf muscle pump and one-way valves normally work together to propel venous blood back toward the heart. Calf muscle contraction forces the blood out of the deep veins and into the central circulation. While blood is being pumped from the deep veins, the one-way valves in the perforator system are closed to prevent backflow of the venous blood into the superficial veins. As the calf muscle relaxes, the valves in the perforator veins open to permit the blood in the superficial system to flow into the deep veins. At the onset of calf muscle contraction, the pressures within the deep venous system peak at 120 to 300 mm Hg. These pressures then fall rapidly as the veins empty and the calf muscle relaxes (Figure 12-2). Thus high resting (filling) pressures, but low walking (emptying) pressures, characterize normal venous function (Figure 12-3).

CHRONIC VENOUS INSUFFICIENCY

The three elements most essential to normal venous function are competent valves, the physical properties of the venous wall, and the normally functioning calf muscle pump. With the loss of valvular competence, veins no longer fill normally via slow capillary inflow alone. Retrograde flow (reflux or back flow) of venous blood occurs during calf muscle pump relaxation preventing a reduction in the venous pressure and a rapid (<20 second) venous refill time. This sustained high pressure or failure to lower venous pressure in the deep veins via the action of the calf pump muscle can be transmitted into the perforator veins and into the normally low-pressure superficial venous system. Incompetent valves subsequently contribute to venous hypertension and the condition known as CVI. Conditions that cause or contribute to valvular incompetence include those that cause direct damage to the valve leaflets and those that cause venous distention. Distention contributes to valve dysfunction by causing mechanical stretch that results in loss of coaptation of the valve leaflets.

Valve failure changes the normal unidirectional flow of blood into a “bidirectional” flow. As a result, blood refluxes back into the superficial system, causing distention and congestion of the superficial veins and capillaries, which manifest clinically as edema. The deep veins are incompletely emptied, causing increased pressures within the deep system, which create resistance to blood draining from the superficial veins. Resistance to flow creates congestion and distention of the superficial

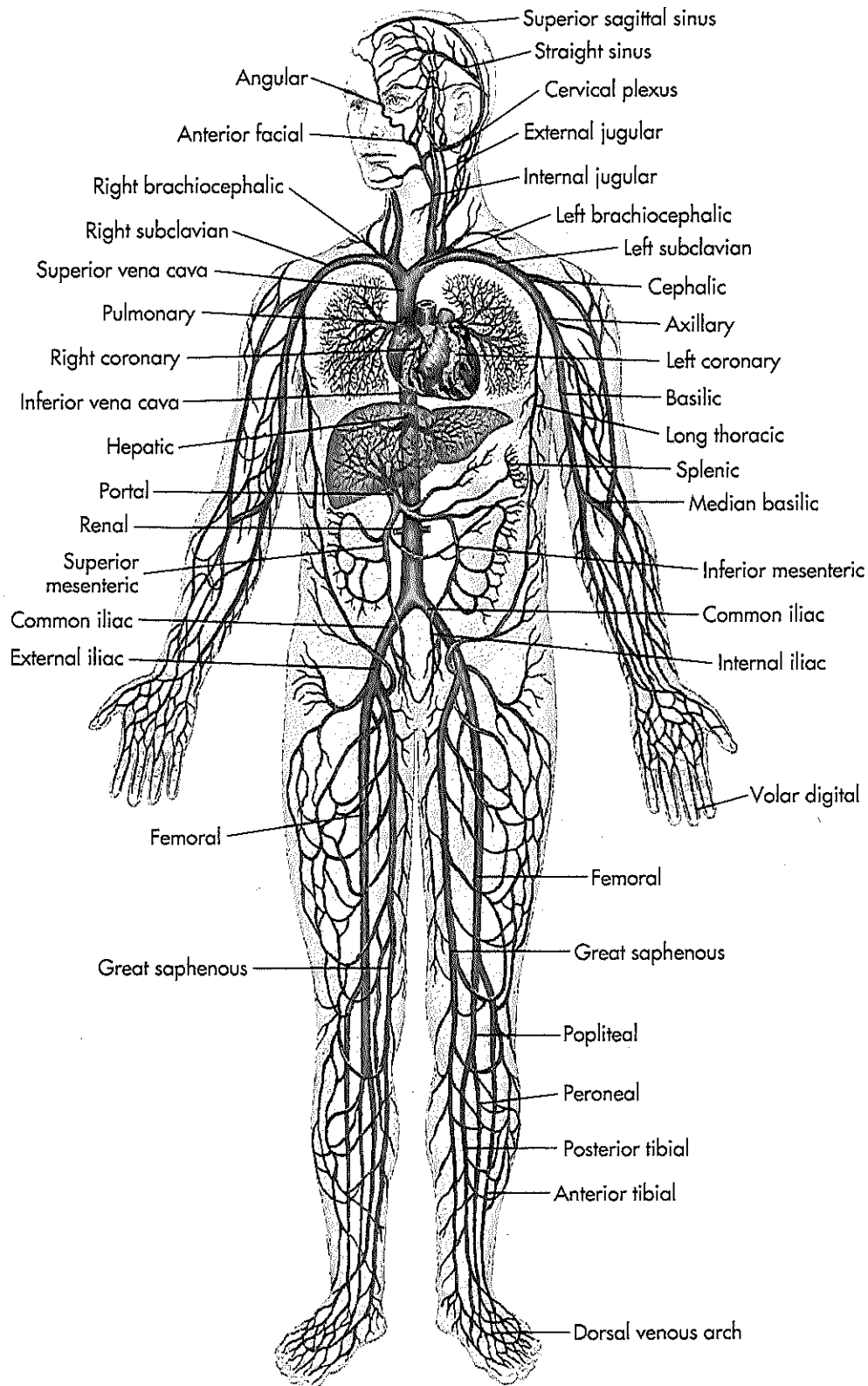


FIGURE 12-1 Systemic circulation: veins. (From Seidel HM et al: *Mosby's guide to physical examination*, ed 6, St. Louis, 2006, Mosby.)

and perforator veins, which cause loss of valve coaptation. The incompetent valves then permit backward transmission of the high pressures in the deep system (Meissner, 2009). The failure to adequately lower venous pressure with the pumping of the calf muscle or by the incompetence of the valves creates ambulatory

venous hypertension. Venous ulceration is a direct result of ambulatory venous hypertension from CVI. A clear understanding of the anatomy and physiology of the lower extremity venous system provides the framework for determining the pathology of LEVD, ambulatory venous hypertension, and venous ulceration.

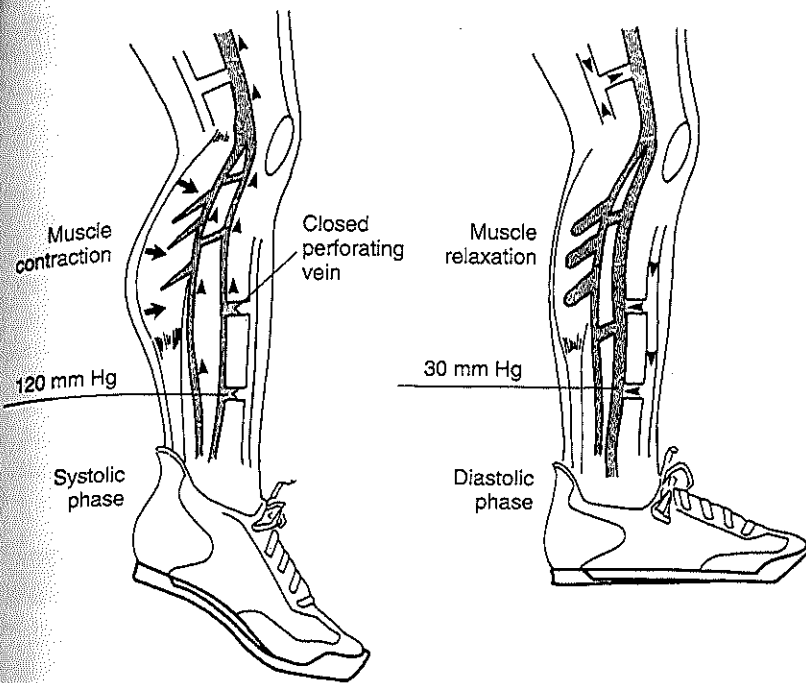


FIGURE 12-2 Anatomy of the perforating (communicating) veins. During the systolic phase of calf muscle contraction, the one-way valves of the perforating veins are closed, which prevents deep-to-superficial blood flow. During the diastolic phase the valves of the perforating veins are open, allowing superficial-to-deep blood flow to refill the deep veins. (From O'Donnell TF Jr, Shepard AD: *Chronic venous insufficiency*. In Jarrett F, Hirsch SA, editors: *Vascular surgery of the lower extremities*, St. Louis, 1996, Mosby.)

The majority of patients have multisystem valvular incompetence (i.e., incompetent valves in at least two of the three venous systems) (Meissner, 2009). Perforator valve incompetence is particularly common and clinically significant. At least two thirds of patients with venous hypertension and venous ulcers have incompetent perforator valves, which can result in supramalleolar pressures well above 100 mm Hg and a “reflux rate” greater than 60 ml/min. When multiple valves become incompetent, the effect is magnified and clinically evident disease becomes much more likely (Kalra and Glociczki, 2003; Meissner, 2009).

Of the three leg muscle pumps responsible for venous return in lower extremities (foot, calf, and thigh), the calf muscle pump is of greatest importance and generates the highest pressure. Among the venous pumps, the ejection fraction of the calf muscle pump is 65% compared to only 15% from the thigh muscle. In the limb with

active ulceration, the ejection fraction can decrease to 35% (Meissner, 2009).

Ultimately, the end result of prolonged ambulatory venous hypertension is damage to the skin and soft tissues that renders these structures vulnerable to minor trauma and susceptible to spontaneous ulceration. Venous ulcers are caused primarily by chronic valvular disease of the deep venous system and perforators (Hussein, 2008). In the past, the cutaneous inflammation observed with venous insufficiency was believed to be the result of blood pooling (thus the term *stasis*) with low oxygen tension in the superficial veins, which precipitated hypoxic damage to the overlying skin. Today, no evidence supports the theories of stasis or hypoxia, prompting discontinuation of the terms *stasis dermatitis* and *stasis ulcers* (Flugman and Clark, 2009).

Classification

CVI is classified according to: Clinical indicators, Etiologic factors, Anatomic location of the dysfunctional venous structures, and specific Pathophysiologic processes (CEAP). This system is presented in Table 12-1 (Agren and Gottrup, 2007; WOCN Society, 2005).

Risk Factors

Risk factors for CVI include a history of major leg trauma, hip or knee surgery, and vein stripping. However, factors that lead to valvular or calf muscle dysfunction are the most common risk factors; they are listed in Table 12-2 along with other key elements of the medical history that must be obtained for the patient with a venous ulcer.

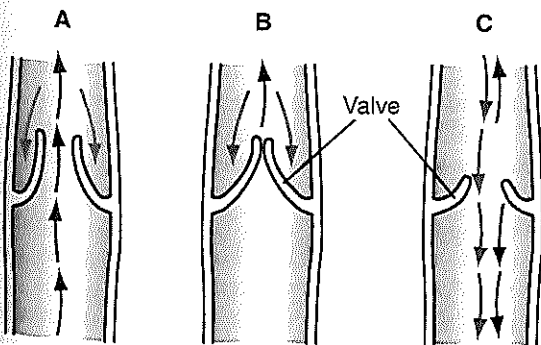


FIGURE 12-3 Venous valves. **A**, Open valves allow forward blood flow. **B**, Closed valves prevent back flow. **C**, Incompetent valves unable to fully close, causing blood to flow backward and producing venous insufficiency.

TABLE 12-1 CEAP Classification for Lower Extremity Venous Disease

| Clinical Classification | Etiologic Classification | Anatomical Classification | Pathophysiologic Classification |
|--|---|---------------------------------------|---|
| C ₀ : No visible or palpable indicators of venous disease | E _c : Congenital | A _s : Superficial veins | P _r : Reflux |
| C ₁ : Telangiectases | E _p : Primary | A _d : Deep system | P _o : Obstruction |
| C ₂ : Varicosities | E _s : Secondary (post-thrombosis) | A _p : Perforator system | P _{ro} : Combination of reflux and obstruction |
| C ₃ : Edema | E _n : No venous cause identified | A _n : No venous identified | |
| C ₄ : Venous skin changes (hemosiderosis, dermatitis, lipodermatosclerosis) | | | |
| C ₅ : Venous skin changes <i>plus</i> healed ulceration | | | |
| C ₆ : Venous skin changes <i>plus</i> active ulceration | | | |
| | S: Symptomatic (aches, pain tightness, skin irritation) | | |
| | A: Asymptomatic | | |

An example of using CEAP classification is C_{6s}; E_p; A_{pd}; P_r.

TABLE 12-2 Key Elements of History for Patients with Venous Ulcers

| Element | Description |
|---|--|
| Risk factors for valvular dysfunction | Obesity, pregnancy, thrombophlebitis, leg trauma (e.g., fracture), thrombophilic conditions |
| Risk factors for muscle dysfunction | Sedentary lifestyle, prolonged standing, advanced age, altered or "shuffling" gait, musculoskeletal conditions and surgeries that compromise calf muscle function (e.g., paralysis, arthritis) |
| Factors that impede healing | Diabetes, tobacco, malnutrition, unplanned weight loss, medications |
| Factors that impede treatment | Limited activity and mobility, cardiac disease, heart failure (clinically significant heart failure is contraindication to compression) |
| Ulcer history | Previous ulcers, onset, duration, precipitating event (duration >6 months is negative predictor for wound healing) |
| History of prior treatment | Surgical, pharmacologic, compression (venous ulcers that consistently fail to respond to treatment should be evaluated for misdiagnosis, malignant or mixed disease) |
| Patient concerns and anticipated barriers | Pain, itching, anxiety, anticipated barriers, transportation, ability to apply compression, job/financial limitations, impact on activities of daily living, treatment goals and priorities |

Valvular Dysfunction. Numerous risk factors for valvular dysfunction have been identified and include the following (Burrows et al, 2007; WOCN Society, 2005):

- Obesity, which creates resistance to venous return due to pressure on pelvic veins
- Pregnancy, especially multiple pregnancies or pregnancies that are close together, because of increased pressure against pelvic veins and compromised venous return
- Thrombophlebitis (e.g., deep vein thrombosis, pulmonary embolism), which triggers an inflammatory response that can cause direct damage to the valve leaflets, or chronic partial deep vein obstruction due to incomplete recanalization of the vein, which in turn causes venous distention and valvular compromise
- Leg trauma (e.g., fracture), which suggests undiagnosed damage to the vessel walls and valves
- Thrombophilic conditions (e.g., protein S deficiency, protein C deficiency, factor V [Leiden mutation]), which increase the coagulability of venous blood, thus increasing the risk of deep vein thrombosis and microvascular thrombosis; thrombophilic conditions have been identified in as many as 50% of patients with venous ulcers

Calf Muscle Dysfunction. The dynamics of the calf muscle pump can be adversely affected by changes that accompany major injuries, neurologic disease, and bone or joint pain. The calf muscle becomes weak with disuse and gait changes can exacerbate venous hypertension and calf muscle atrophy (Burrows et al, 2007). Risk factors for compromised calf muscle function include the following:

- Sedentary lifestyle
- Occupations that require prolonged standing
- Musculoskeletal conditions that compromise calf muscle function (e.g., paralysis, arthritis)
- Advanced age, which is associated with decreased elasticity of the calf muscle tendon
- Reduced mobility
- Altered or "shuffling" gait that fails to induce calf muscle contraction; reduced mobility and gait do not relate to calf muscle dysfunction
- Arthroscopic surgery, which could cause fixation of the hip, knee, or ankle, leading to loss of calf muscle pump
- Injection drug use due to progressive deterioration of the venous function of the legs (Pieper et al 2008)

Pathology of Venous Ulceration

Whereas CVI is clearly precipitated by ambulatory venous hypertension, the reason for venous ulceration as a consequence of venous hypertension is not well understood. This mystery is compounded by the fact that only a minority of patients with chronic venous disease actually progress to ulceration.

Fibrin Cuff Theory. Browse and Burnand (1982) initially postulated that capillary bed distention permitted leakage of large molecules such as fibrinogen into the dermal tissue, and that the fibrinogen then polymerized to form a thick perivascular cuff composed of fibrin, fibronectin, laminin, tenascin, and collagen. These cuffs do not pose any barrier to the diffusion of oxygen and nutrients into the tissues; therefore skin hypoxia cannot be the ultimate factor in the pathogenesis of venous ulcerations (Meissner, 2009).

White Blood Cell Activation and Trapping Theory. The trap hypothesis suggests that venous hypertension reduces the velocity of blood flow in the postcapillary bed. When blood flow becomes sluggish, leukocytes begin to adhere to each other (leukocyte aggregation) and/or to the capillary walls (leukocyte margination). This triggers the release of toxic oxygen metabolites, proteolytic enzymes, and cytokines causing tissue inflammation and dermal fibrosis (Meissner, 2009). Dermal capillary loops also become plugged with leukocytes so that fibrin and other macromolecules leak out of the permeable capillary beds into the dermis, further aggravating inflammatory and fibrotic changes in the subcutaneous tissues and rendering them very susceptible to ulceration, which can occur spontaneously or as a result of minor trauma. Leukocyte migration and activation and the interaction of leukocytes with the endothelium in the presence of venous hypertension play considerable roles in the pathophysiology of venous ulcerations (Agren and Gottrup, 2007; Kalra and Gloviczki, 2003; WOCN Society, 2005).

ASSESSMENT

Key assessment parameters for patients with a leg ulcer include medical history, ulcer history, previous treatments, clinical examination, inspection of the ulcer, and Doppler assessment of pulses (Nelzen, 2007). Assessment of Doppler pulses are discussed in Chapters 10 and 11 and illustrated in Figure 11-1.

Patient History

Risk factors for CVI are identified through the patient history. Of particular importance are risk factors that differentiate venous insufficiency from arterial disease and other pathologies that may cause ulceration in the lower extremity (see Table 12-2). Because of the contraindications to sustained compression, pretreatment

evaluation must include a cardiac history and any indicators of uncompensated heart failure.

Lower Extremity Assessment

Both legs need to be examined by the clinician to ascertain if the manifestations are bilateral or more severe on one extremity. Findings unique to LEVD include edema, hemosiderosis, dermatitis, lipodermatosclerosis, atrophie blanche, varicose veins, ankle flaring, and scarring from previous ulcers (WOCN Society, 2005). Checklist 12-1 gives a list of assessment findings unique to the leg of a patient with CVI.

Arterial Perfusion. Concomitant arterial disease occurs in as many as 25% of patients with venous ulcers (de Araujo et al, 2003; Ryan et al, 2003). Therefore an ankle-brachial index (ABI) should be obtained to determine if some degree of arterial insufficiency is present. An ABI of 1.0 indicates a “pure” venous ulcer (no coexisting arterial insufficiency). An ABI of 0.9 or less indicates arterial insufficiency is present; these ulcers are referred to as *mixed arterial/venous ulcers*. This is an important initial assessment because compression (critical for treatment of venous ulcers) must be modified or, in some cases, omitted. Wounds that begin specifically as a venous ulcer can develop an arterial component, so monitoring for signs of arterial disease at regular intervals is necessary (WOCN Society, 2008).

Edema. Edema is a classic indicator of venous insufficiency because of the combination of capillary bed distention and elevated intracapillary pressures. As described in Chapter 10, the severity of edema varies among patients and from time to time throughout the day. The classic pattern is pitting edema (see Figure 10-1) that worsens with dependency and improves with elevation. Box 10-1 describes the assessment of pitting edema. With prolonged disease and gradual fibrosis of the soft tissues, edema may become “brawny,” that is, nonpitting. Thus

CHECKLIST 12-1

Lower Extremity Assessment for Chronic Venous Insufficiency

- ✓ General Appearance
 - Trophic changes: lipodermatosclerosis
 - Edema: present from ankle to knee, often pitting
 - Color: hemosiderin staining, atrophie blanche
 - Dermatitis or varicosities may be present
- ✓ Pain
 - Dull aching (see Table 10-2)
 - Exacerbates with dependency, improves with compression
- ✓ Wound Characteristics
 - Gaiter area (see Table 10-3)
 - Exudative and shallow
- ✓ Perfusion
 - Diminished only with coexisting arterial disease
 - Diagnostic evaluation: duplex ultrasound

the characteristics of the edema are a clue to the duration of the underlying disease process.

The distribution of edema is also indicative of the underlying process. Venous edema primarily involves the lower leg between the ankle and the knee. In contrast, lymphedema and lipedema involve the entire extremity. Table 10-1 gives a comparison of the edema associated with these three conditions. Measuring the circumference of the calf and gaiter area is another method for assessing edema, especially if it is unilateral. Thus changes in these circumferential measurements provide an indication of the effectiveness of compression therapy (Nelzen, 2007). Circumferential measurements usually are taken weekly when the compression bandage is changed.

Hemosiderin Staining (Hemosiderosis). Another “classic” indicator of venous insufficiency is hemosiderosis, the discoloration of the soft tissue located in the gaiter area that results when extravasated red blood cells break down and release the pigment hemosiderin. The result is a gray-brown pigmentation of the skin known also as *hyperpigmentation* or *tissue staining* (de Araujo, et al, 2003) (see Plate 34). Hemosiderin plays a role in the evolution of skin changes toward lipodermatosclerosis and ulceration (Caggiati et al, 2008).

Lipodermatosclerosis. Lipodermatosclerosis (see Plate 38), a term used to denote fibrosis, or “hardening,” of the soft tissue in the lower leg, is indicative of long-standing venous insufficiency. The fibrotic changes typically are confined to the gaiter, or “sock,” area of the leg, which results in an inverted “champagne bottle” or “apple core” appearance of the affected lower leg. The fibrosis causes abnormal narrowing of the affected area, which contrasts sharply with the normal tissue in the proximal limb, and a “woody,” hard texture when the area is palpated. These fibrotic changes are thought to result from a combination of fibrin deposits, compromised fibrinolysis, and deposition of collagen in response to growth factors produced by activated white blood cells (de Araujo et al, 2003). A body mass index greater than 34 has been found to predispose to lipodermatosclerosis (Bruce et al, 2002).

Varicosities. Varicose veins are swollen and twisted veins that appear blue, are close to the skin’s surface, may bulge or throb, cause the legs to swell, and precipitate a feeling of heaviness. They are most often seen in the back of the calf or the medial aspect of the leg. Varicosities precede valvular incompetence and appear to develop as a consequence of intrinsic structural and biochemical abnormalities of the vein wall (Meissner, 2009). Patients with varicosities should manage their weight and exercise and avoid crossing their legs and wearing constrictive garments (WOCN Society, 2005).

Skin Changes Near the Ankle. *Ankle blowout* has been described as uncommon painful clusters of tiny venous ulcers located near the medial malleolus originating from dilated ruptured vessels (Kunimoto, 2001), but

no further discussion related to ankle blowout has been noted in recent literature or evidence-based guidelines. *Malleolar flare* has been described in recent guidelines as visible capillaries from distention of small veins around the medial malleolus (WOCN Society, 2005).

Atrophie Blanche Lesions. Atrophie blanche lesions (see Plate 35) can be found in as many as one third of patients with LEVD. These lesions are smooth white plaques of thin, “speckled” atrophic tissue with tortuous vessels on the ankle or foot with hemosiderin-pigmented borders. Sometimes mistaken for scars of healed ulcers, this clinical finding actually represents spontaneously developing lesions. Prompt recognition is important so that a plan can be established to protect these high-risk areas from ulceration due to the thin, atrophic epidermis (de Araujo et al, 2003; Ryan et al, 2003; WOCN Society, 2005). Ulcers occurring in this area usually are small, very painful, and hard to heal. Topical steroids should be avoided because they can cause further damage to the very fragile skin.

Venous Dermatitis. Venous dermatitis is a common but distressing inflammation of the epidermis and dermis on the lower extremity of the patient with LEVD (see Plate 38). Often the earliest cutaneous sequelae of venous insufficiency, they most commonly affect middle-aged to elderly patients (Flugman and Clark, 2009). Venous dermatitis is characterized by scaling, crusting, weeping, erythema, erosions, and intense itching; symptoms may be acute or chronic. The cutaneous inflammation of venous dermatitis is often confused with cellulitis. Factors that distinguish between dermatitis and cellulitis are listed in Table 12-3 (WOCN Society, 2005).

Venous dermatitis results from the release of inflammatory mediators from activated leukocytes that are trapped within the fibrin cuffs and surrounding perivascular space (Flugman and Clark, 2009). Dermal fibrosis, a hallmark of venous dermatitis, develops as a result of fibrin cuff formation, decreased fibrinolysis, and release of transforming growth factor- β_1 (a mediator of dermal fibrosis) by the leukocytes. Potent chemoattractants (intercellular adhesion molecule-1 and vascular cell adhesion molecule-1) keep leukocytes active in the perivascular environment and perpetuate cutaneous inflammation with fibrosis. Why venous dermatitis is common among some patients but is rare among others is unclear (Ryan et al, 2003; WOCN Society, 2005).

Venous dermatitis increases the risk of developing contact sensitivity due to the presence of chronic inflammation of the skin (Flugman and Clark, 2009). Exposure to usually benign topical substances (e.g., wound exudate, skin sealants, adhesives, silver sulfadiazine) easily exacerbates venous dermatitis. Frequent contact allergens include lanolin, balsam of Peru, and fragrances (Romanelli and Romanelli, 2007). Patients also can become sensitized to rubber products contained in some compression wraps and stockings. More than 30% of patients with contact dermatitis developed sensitivity to the topical antibiotics

TABLE 12-3 Distinguishing Between Dermatitis and Cellulitis

| | Dermatitis | Cellulitis |
|------------------|--|--|
| Symptoms | Afebrile Itching Varicose veins/deep vein thrombosis | May have fever Painful No relevant history |
| Signs | Normal temperature Erythema, inflammation May be tender Vesicles and crusting Lesions on other parts of the body (e.g., other leg, arms) May be unilateral or bilateral | Elevated temperature Erythema, inflammation Tenderness One or a few bullae No crusting No lesions elsewhere |
| Portals of entry | N/A | Unilateral Usually unknown; breaks in skin, ulcers, trauma, tinea pedis, intertrigo implicated |
| Laboratory | Normal white blood cell count Negative blood cultures Skin swabs (<i>Staphylococcus aureus</i> common) | Leukocytosis Blood cultures usually negative Skin swabs usually negative except for necrotic tissue |

DVT, Deep vein thrombosis; WBC, white blood cell.

From Wound, Ostomy and Continence Nurses Society (WOCN Society): *Guideline for management of patients with lower extremity venous disease*, WOCN clinical practice guideline series #1, Glenview Ill, 2005. Data from Quartey-Papafio CM: Lesson of the week: importance of distinguishing between cellulitis and varicose eczema of the leg, *BMJ* 318(7199):1672-1673, 1999.

neomycin and bacitracin (Alguire and Mathes, 2007). Although less common, sensitization of the skin to topical corticosteroids can develop, triggering an allergic contact dermatitis. When the clinical manifestations of the limb affected with venous dermatitis worsen despite appropriate topical therapy, contact dermatitis should be considered.

To prevent venous dermatitis, product ingredients should be carefully scrutinized before topical therapy is selected, and products containing sensitizers should be avoided (de Araujo et al, 2003). Skin moisturizers such as bland, perfume-free topical emollients and white petrolatum, can be used to maximize epidermal integrity. An essential component of prevention and treatment of venous dermatitis is graduated compression (discussed later in this chapter), which may require considerable patient education and encouragement due to the discomfort associated with an inflamed, edematous limb. Patients need reassurance that the discomfort should decrease as the edema resolves. Exudate absorbers such as alginates and hydrofibers are commonly indicated, often in combination with a secondary foam dressing to adequately absorb and contain exudate.

To reduce inflammation and itching, mild-potency topical corticosteroids (e.g., triamcinolone 0.1% ointment) can be used short term (i.e., 2 weeks) but sparingly because of the risk for skin atrophy. High-potency topical corticosteroids are rarely used because of the risk for skin atrophy as well as systemic absorption through open denuded skin. Systemic corticosteroids are seldom warranted for treatment of venous dermatitis (Flugman and Clark, 2009). Cool compresses with Burow's solution (aluminum acetate) followed by an application of plain petrolatum also can be used to relieve itching. Additional remedies for venous dermatitis include Condy

solution (potassium permanganate), dilute vinegar compresses, and cool tar ointment. Patients with severe or nonresponsive dermatitis should be referred to dermatology for management (Bonham, 2003; Ryan et al, 2003).

Ulcer Characteristics. The classic venous ulcer is located in the gaiter area and around the medial malleolus, due to the greatest hydrostatic pressure at these sites. Typically, the ulcers are shallow, with moderate to high exudate and a dark red "ruddy" wound base or a thin layer of yellow slough. Islands of eschar may be present. Ulcers that are deep and may have exposed tendon are likely not to be of pure venous origin (Nelzen, 2007). Venous ulcers usually have irregular edges and periwound maceration, crusting, scaling, and/or hemosiderin staining (Wipke-Tevis and Sae-Sia, 2004) (see Plates 34 and 38). See Table 10-3 for a comparison of features that distinguish venous, arterial, and neuropathic ulcers.

Diagnostic Evaluation

In order to effectively diagnose and safely manage the patient with a CVI wound, the patient must first be evaluated for the presence of arterial insufficiency because standard therapy for venous ulcers (compression) may be contraindicated or may require modification of therapy in the presence of arterial disease (Robson et al, 2006). Arterial insufficiency is diagnosed with an ABI of 0.9 or less or a toe-brachial index of 0.6 or less (see Table 11-3). Chapter 11 describes diagnostic tests for arterial disease.

CVI is the result of either venous reflux or venous obstruction. Noninvasive vascular tests are used to distinguish between these two conditions. Traditionally,

tourniquet test, photoplethysmography, and venography were used. However, poor reliability and inability to provide visualization of the venous system compromised the utility of the tests. Therefore these tests have been largely replaced by duplex ultrasound imaging (Meissner, 2009; Min et al, 2003). However, Kelechi and Bonham (2008) propose further clinical studies to examine the utility of a hand-held noninvasive photoplethysmography instrument that measures venous filling time to facilitate early diagnosis of chronic venous insufficiency that might otherwise go unrecognized.

Duplex Ultrasound. Duplex ultrasound scanning is now recognized as the gold standard for accurate evaluation of the highest point of valve failure and the extent of reflux (Whiddon, 2007).

Duplex ultrasound imaging is noninvasive and has a high degree of sensitivity. Duplex imaging technology uses two-dimensional ultrasound and Doppler shift to produce images of blood flow through the superficial, deep, and perforating veins, pinpointing the anatomic site of reflux, obstruction such as deep vein thrombus, and abnormal vein walls as well as reflux through delicate venous valves (Kelechi and Bonham, 2008; WOCN Society, 2005). Color duplex ultrasound scanning performed with proximal compression or a Valsalva maneuver helps to confirm a venous etiology and assists the clinician to determine if radiologic (laser ablation) or surgical intervention is warranted, especially in patients with nonhealing or recurrent venous ulceration (Robson et al, 2006).

MANAGEMENT

As outlined in the LEVD guideline (WOCN Society, 2005) and the Wound Healing Society's guidelines for the treatment of venous ulcers (Robson et al, 2006), primary interventions for correcting the underlying cause of venous insufficiency and venous hypertension include lifestyle adaptations and compression therapy; pharmacologic agents and surgical procedures do not play a large role in the management of LEVD.

Limb Elevation

Limb elevation is a simple but effective strategy for improving venous return by making use of gravitational forces. This is an important component of management for any patient with venous insufficiency, but it is an essential element of therapy for patients who are unable to adhere to a compression therapy regimen. Patients should be taught to lie down and elevate the affected leg above the level of the heart for at least 1 to 2 hours twice daily as well as during sleep. This position may be difficult for the obese person to manage comfortably. In addition, patients should be taught to strictly avoid prolonged standing or prolonged sitting with the legs dependent. Periods of standing or sitting must be interspersed with

walking. Having the patient keep a "legs-up" chart can reinforce the importance of leg elevation. This chart should be reviewed with the patient at each visit (Wipke-Tevis and Sae-Sia, 2004).

Exercise

Normal function of the calf muscle pump is essential to venous return, and effective contraction of the calf muscle requires a mobile ankle and routine dorsiflexion beyond 90 degrees. The patient with limited mobility is a challenge for clinicians. A patient with reduced ankle mobility or a "shuffling" gait should undergo a physical therapy evaluation to determine if he or she can benefit from gait retraining and routine exercises to increase ankle strength and range of motion (Burrows et al, 2007). A home-based exercise program that includes isotonic exercise can improve poor calf muscle and calf muscle pump function (WOCN Society, 2005). The Wound Healing Society's Prevention of Venous Ulcers Guidelines recommends calf muscle pump exercises as helpful in long-term maintenance and venous ulcer prevention (Robson, et al, 2006). All patients with venous insufficiency should be encouraged to perform ankle pumps routinely while standing or sitting and to intersperse standing and sitting with walking (Wipke-Tevis and Sae-Sia, 2004). Primarily used to assess the elderly for fall risk, the Tinetti Balance and Gait Scale was found to be reliable and valid in evaluating the walking mobility of patients with venous ulcers using injectable drugs. The wound specialist may find it beneficial to incorporate this scale (available in Appendix B) into the workup of the patient with venous ulcers.

Weight Control

Obesity interferes with venous return, thus increasing the risk for LEVD. Morbid obesity can cause insufficiency in the deep venous system. In addition, significant obesity makes it very difficult for the patient to adhere to compression therapy and to avoid prolonged sitting. With the increase in obesity in younger people, the incidence of venous insufficiency and ulcers will continue to rise. Therefore it is important to educate patients regarding the relationship between weight and venous disease and to strongly encourage patients to reduce their weight to a healthy level. Patients who are morbidly obese should be referred to a bariatric treatment center for evaluation and management (Wipke-Tevis and Sae-Sia, 2004).

Pharmacologic Therapy

Treating venous ulcers with pharmacologic means is based on the hypothesis of venous insufficiency pathogenesis. Inappropriate leukocyte activation, which has been shown to be present in chronic venous disease, can

lead to the development of a venous ulcer (Coleridge-Smith et al, 2005). Diuretics and topical corticosteroids reduce edema and pain in the short term but offer no long-term treatment. Herbal supplements decrease the inflammatory response to venous hypertension but are not licensed by the U.S. Food and Drug Administration (FDA) and can vary in efficacy and safety.

Several pharmacologic agents have demonstrated benefit in the management of venous disease. The three agents with documented efficacy in the management of venous disease are pentoxifylline (Trental), micronized purified flavonoid fraction (Daflon), and horse chestnut seed extract (HCSE).

Pentoxifylline (Trental). In the United States, pentoxifylline (Trental) is the drug most commonly prescribed for venous disease and appears to be an effective adjunct to compression therapy. Its mechanism of action appears to be reduced aggregation of platelets and white blood cells, which reduces capillary plugging, and enhanced blood flow in the microcirculation, which reduces tissue ischemia (Jull et al, 2004, 2007). Dosages of 400 mg orally three times daily can accelerate healing of venous ulcers and should be considered for slow-healing venous ulcers (Jull, 2007; WOCN Society, 2005).

Pentoxifylline may also promote healing even in the absence of compression. However, the beneficial effects of pentoxifylline must be balanced against its potential adverse effects (e.g., diarrhea, nausea) and its cost. Therefore pentoxifylline is generally reserved for patients who do not respond to standard therapy and is not used for routine care (Jull, 2007).

Micronized Purified Flavonoid Fraction (MPFF). Although not available in the United States, the phlebotropic drug known as micronized purified flavonoid fraction (MPFF; Daflon) has been approved by Europe and other countries to improve outcomes for patients with LEVD. The specific mechanisms of its action include the following: (1) enhances venous tone, which promotes venous return; (2) reduces capillary permeability, which reduces edema formation; and (3) reduces expression of endothelial adhesion molecules, which reduces margination, activation, and migration of leukocytes (Robson et al, 2006). These mechanisms reduce the release of inflammatory mediators, which is thought to be the primary pathologic event resulting in dermatitis, lipodermatosclerosis, and ulceration (Coleridge-Smith et al, 2005; Lyseng-Williamson and Perry, 2003; Simka and Majewski, 2003).

The combination of MPFF with standard therapy (compression plus topical therapy) resulted in a statistically significant improvement in healing rates compared to standard therapy alone or to placebo in a double-blind trial, with a side effect profile comparable to that of placebo (Coleridge-Smith et al, 2005). A meta-analysis of MPFF as adjunct therapy for venous ulcers concluded that venous ulcer healing was accelerated and recommended MPFF use for large and long-standing ulcers

(Coleridge-Smith et al, 2005). In addition, cost analysis studies have demonstrated a significant reduction in cost of healing compared to conventional therapy. Finally, studies indicate significant improvement in quality-of-life scores for patients with LEVD treated with MMPF (Coleridge-Smith, 2003; Simka and Majewski, 2003).

Horse Chestnut Seed Extract. The herbal agent HCSE containing aescin is commonly used in Europe as a method for managing CVI (Jull, 2007; Leach et al, 2006). The mechanism of action appears to be an inhibitory effect on the catalytic breakdown of capillary wall proteoglycans (Suter et al, 2006). Several placebo-controlled trials have demonstrated decrease in leg size, pain, pruritus, and tenseness. Preliminary evidence suggests comparable outcomes between HCSE and compression therapy. Available as an oral tincture, topical gel, and tablets (20 mg, 50 mg), the recommended oral dosage is 300 mg every 12 hours for 12 weeks (Pittler, 2006). Severe allergic reactions have been reported when HCSE was given intravenously, and hepatitis has been associated with intramuscular injections. More studies are needed to clearly verify the efficacy of HCSE in treatment, especially in long-term use and as an adjunct to compression therapy.

COMPRESSION THERAPY

Venous insufficiency is associated with increased hydrostatic pressure in veins in the legs. Compression therapy is used to reduce hydrostatic pressure and aid venous return (Vowden & Vowden, 2006). Compression is provided by wraps, bandages, garments or devices. Compression wraps are products that specifically wrap around the extremity. Bandage is the more common term used in European literature in place of wrap or compression dressing, however. Garment refers to compression products that are a clothing item such as compression stockings. The intermittent pneumatic compression device is the only product that is powered. Compression products apply pressure externally from the base of the toes to the knee to support the calf muscle pump during ambulation and dorsiflexion. The increased interstitial tissue pressure serves to oppose leakage of fluid into the tissues and to return interstitial fluid to the blood and lymph vessels, thus eliminating edema. Compression of the superficial veins promotes coaptation and normal function of the valves; it also increases the velocity of blood flow, which reduces the aggregation and extravasation of white blood cells (Weingarten, 2001).

Features

Compression products share many different features and these features guide the selection of the most appropriate compression option for each individual. Compression therapy can be either sustained (i.e., continuous) pressure or intermittent pressure. Products that remain in

place and are removed after several days or only at night provide continuous pressure; the majority of products on the market provide continuous compression (Table 12-4). Products that provide intermittent compression are applied 2-3 times per day for 1- to 2-hour intervals; intermittent compression is particularly beneficial to the patient who cannot tolerate continuous compression or is unable to apply the continuous compression wraps or stockings.

Another feature of compression products is based on the type of material used to deliver the pressure: elastic or inelastic. *Elastic compression adapts to changes in limb volume.* The product exerts external pressure while the leg is at rest as well as when the calf muscle expands during ambulation, thus continuing to provide external

pressure. Elastic compression products are appropriate choices for patients who are relatively sedentary or who have a "shuffling" gait that fails to engage the calf muscle (de Araujo et al, 2003). *Inelastic (or nonelastic) compression will not expand during ambulation.* As the calf muscle expands while walking, pressure is created by the muscle pressing against the "semi-rigid" bandage/dressing. At rest, when there is no calf pump muscle activity, only limited compression occurs. Therefore, inelastic compression products are most appropriate for patients who are actively ambulating.

Compression products can be disposable or reusable. Many compression products are disposable and are used early in treatment when significant edema or an ulcer is present. Reusable compression products can be removed,

TABLE 12-4 Formulary of Compression Therapy Products

| Category | Type | Examples* | Features | | | | | |
|------------------------------|---------------------------|--|----------|------------|------------|----------|----------------------|-------------------|
| | | | Elastic | In-elastic | Disposable | Reusable | Therapeutic pressure | Modified pressure |
| Wraps | Multilayer | Profore/ProGuide (Smith & Nephew) | X | | X | | X | X |
| | | Dyna-Flex (Systagenix Wound Management) | | | | | | |
| | Long stretch | Coban™ 2 Layer (3M™) | | | | | | |
| | | ThreePress (Hartmann-USA Inc.) | | | | | | |
| Garments | Short stretch | SurePress (ConVaTec) | X | | | X | X | X |
| | | Setopress (Mölnlycke Health Care) | | | | | | |
| | Paste | Comprilan (BSN Medical) | | X | | X | | X |
| | | Farrow wrap (Farrow Medical Innovation) | | | | | | |
| Garments | Reusable inelastic device | Viscopaste (Smith & Nephew) | | X | X | | | X |
| | | Unna-Flex (ConvaTec) | | | | | | |
| | Tubular sleeve | Gelocast (BSN Medical) | | | | | | |
| | | Unna Press (Derma Sciences) | | | | | | |
| Intermittent pneumatic Pumps | Stockings | CircAid (CircAid Medical Products) | | X | X | X | | X |
| | | Tubigrip (Mölnlycke Health Care) | X | | | X | | X |
| | Medigrip (Medline) | Jobst | X | | | X | X | X |
| | | Juzo Sigvaris Medi-Strumpf TheraPress DUO Mobility (Derma Science Inc.) | | | | | X | X |

*=Not inclusive.

cleaned, and then reused. The reusable products are non-adhesive wraps or garments. The nonadhesive reusable wraps can be used while edema and/or ulcer are present. The reusable garments are primarily used for "maintenance compression" once the edema has resolved and the ulcer is significantly healed or at least no longer exudative.

A key feature of compression wraps/garments is that they can provide a range of pressures. The amount of compression considered "therapeutic" for venous insufficiency (effective in controlling venous hypertension and preventing edema formation) is 30 to 40 mm Hg at the ankle (de Araujo et al, 2003; O'Meara et al, 2009; Paquette and Falanga, 2002; Vowden & Vowden, 2006). Some clinicians recommend even higher levels of compression (40 to 50 mm Hg) for patients with severe venous insufficiency (Robson et al, 2006). While the evidence is clear that 30 mm Hg or more is considered high compression and is the preferred level of pressure, many patients experience discomfort with high levels of pressure or are unable to physically apply the garment. In these situations, lower levels of pressure are more appropriate (Table 12-4). A general guide to categories of pressure is as follows: high pressure (30-40 mm Hg), medium pressure (20-30 mm Hg), and low pressure (15 mm Hg) (Nelson et al, 2000). Many compression wraps have "indicators" to guide the amount of tension or stretch to use when applying the wraps so that the desired amount of pressure can be attained. Similarly, compression stockings are manufactured to provide a specific range of pressure.

Clues for Compressing Correctly

1. Follow manufacturer's instructions for application technique to assure attaining the appropriate level of compression.
2. Measure the extremity accurately when using compression garments.
3. Assess patient mobility and activity carefully when selecting elastic versus inelastic compression products. Inelastic compression products will not be effective if the patient is unable to perform very frequent calf muscle pumps, such as with ambulation.
4. Compression should be used with caution if the patient has decreased leg sensation, infection in the leg, or allergies to ingredients in the compression materials.
5. All patients with a leg ulcer should be screened for arterial disease using a Doppler measurement of the ABI. Use modified or lower levels of compression (23-30 mm Hg at the ankle) when coexisting arterial disease is present. An ABI greater than 0.5 and less than 0.8 precludes high levels of sustained compression (Kelechi and Bonham, 2008; Cullum et al, 2003; Robson et al, 2006). Patients with venous insufficiency and $ABI \leq 0.5$ who require compression

should be managed with IPC (Kelechi and Bonham, 2008).

6. Monitor the skin around and under the compression wrap or device closely and particularly during dressing changes to prevent pressure ulcers. External compression at high pressures will reduce blood supply to the skin and may lead to pressure damage. Similarly, impaired arterial blood supply to the legs may also result in pressure damage. If the patient expresses complaints about the extremity that is suggestive of device-related pressure damage, the compression may need to be removed so that the extremity can be assessed.
7. Patients with venous hypertension need to understand that "compression is for life!" It is not a treatment that can be discontinued once the ulcer heals or the edema resolves. This ongoing compression is often referred to as "maintenance" compression. When the patient fails to adhere to maintenance compression, a 70% recurrence rate of venous ulcers has been reported (Nelson et al, 2000). Furthermore, a normal ABI can potentially deteriorate over time, thus requiring a modification in the recommended level of compression. Diligent monitoring by the wound specialist is essential for continued follow-through with the plan of care (Robson et al, 2006; Wipke-Tevis and Sae-Sia, 2004; WOCN Society, 2005). The wound specialist is challenged to recommend the most clinically effective and "patient-friendly" system for each patient based on individual assessment, indications, contraindications, advantages and disadvantages, and special considerations of the many available compression products.

Contraindications

All compression is contraindicated in the patient with a coexisting venous thrombosis in the extremity with the ulcer and uncompensated heart failure. When compression of any kind is used with uncompensated (unstable) heart failure, edema fluid can mobilize into the circulatory system, potentially increasing preload volume and precipitating pulmonary edema (de Araujo et al, 2003; Weingarten, 2001). In most cases, sustained compression is contraindicated in the presence of severe peripheral vascular disease (i.e., $ABI \leq 0.6$) because sustained tissue pressure could further compromise tissue perfusion and potentially cause ischemic tissue death (Hopf et al, 2006). In these situations, *intermittent* pneumatic compression is a safe and viable option.

COMPRESSION WRAPS

Wraps are one of the most commonly used compression products, especially during the initial phase of treatment when limb volumes are changing rapidly as a result of

edema reduction. Compression wraps are identified by the number of components in the wrap. Single-layer wraps contain a single component while a multi-layer wrap may have 2, 3, or 4 components that are applied to the extremity. All wraps have a bandage layer; this bandage determines whether the wrap is elastic or inelastic. In addition to the bandage layer, the two-layer wrap also has either an orthopedic wool layer placed against the skin under the bandage wrap or a tubular sleeve over the bandage wrap. The components of a three-layer wrap include the orthopedic wool, a bandage wrap, and a tubular sleeve. The four-layer wrap contains the orthopedic wool, a crepe support bandage, a bandage wrap, and a self-adherent bandage (O'Meara et al, 2010).

Generally, compression wraps are applied by professionals and left in place for 3 to 7 days. Most wraps are capable of providing modified compression. The most commonly used wraps are inelastic paste wraps, multi-layer elastic wraps, and single-layer inelastic (short-stretch) bandages (see Table 12-4). Although individual clinical trials have reported multilayer and single-layer compression systems to be equally effective (Burrows et al, 2007; Castonguay, 2008), a recent systematic review of literature conducted by the Cochran Collaboration reported that multi-component systems achieve better healing outcomes than single-component compression (O'Meara et al, 2009).

Application Technique

All compression wraps are designed based on *Laplace's law of physics*, which states that sub-bandage pressure is directly proportional to the tension and number of bandage layers and inversely proportional to leg circumference and bandage width (Figure 12-4). Laplace's law explains why application of a wrap with constant tension will create graduated pressure. Bandage tension is held constant while the circumference of the leg increases steadily from ankle to knee, and sub-bandage pressure will be highest at the ankle and lowest at the knee (Burrows et al, 2007; Ghosh et al, 2008; Moffat et al, 2007).

Some wraps are designed to be applied with a spiral technique, whereas others require a figure-of-eight

application to achieve optimal results. Application techniques must follow the manufacturer's instructions. Some layers require 100% stretch and others 50% stretch; some may incorporate a visual indicator to achieve the correct level of pressure (Wipke-Tevis and Sae-Sia, 2004).

The skill of the clinician applying the wrap will impact tension (Moore, 2002; O'Meara et al, 2009). Studies indicate that even when nurses are experienced with application, they frequently wrap with insufficient tension to produce therapeutic pressure levels. Training has been shown to significantly improve accuracy, but further studies are needed to quantify the interval at which this training should be repeated (Feben, 2003). An accurate and precise sub-bandage pressure monitor may be an option for assessing the clinician's ability to apply safe, graduated pressure.

The patient with a large calf or uneven contours can experience difficulty in keeping the bandage in place. Slippage of the bandage can lead to a tourniquet effect, which can cause edema above the wrap and injury to the skin. Compression wraps must be replaced when slippage occurs. One technique for preventing slippage is the use of extra padding to recontour the leg to a normal shape (Moffat et al, 2007). It has been found that bandages applied in a figure-of-eight configuration tend to stay in place better, especially for the person with a large leg. However, the manufacturer's instructions for wrapping must be followed to accommodate differences in product materials and layers, all of which impact the level of compression achieved. The best time of day to apply compression is when the least amount of edema is present: first thing in the morning before getting out of bed and before hanging the legs over the side of the bed. Patients who are unable to lift their leg(s) should be wrapped by two clinicians to ensure an even application (Moffatt et al, 2007).

Multi-layer Wraps/Bandages

Multi-layer wraps are disposable, elastic, provide sustained compression, and can be applied to provide either a modified or therapeutic level of pressure. These wraps therefore provide compression when the patient is active as well as when they are at rest (Figure 12-5). Multilayer wraps cannot be reused and should be changed when they begin to loosen, slip, or become saturated (typically in 3–7 days). A key feature of multi-layer wraps is absorption of exudate. In many settings, these devices have become the product of choice for early intervention because of their ability to absorb exudate, adapt to changes in limb size, and provide sustained compression at rest and with activity.

Paste Wraps. Paste wraps are inelastic wraps that cannot be reused and provide sustained compression at a modified level of pressure. Dr. Paul Unna was the first to introduce

$$\text{Sub-bandage pressure} = \frac{\text{Tension} \times \text{Number of layers}}{\text{Leg circumference} \times \text{Width of bandage}}$$

FIGURE 12-4 Laplace's law of physics as it applies to compression bandaging. Laplace's law demonstrates how compression therapy is a function of tension, number of layers, leg circumference, and bandage width. Increases in tension and/or layering increase sub-bandage pressure, whereas increases in leg circumference and/or bandage width decrease sub-bandage pressure.

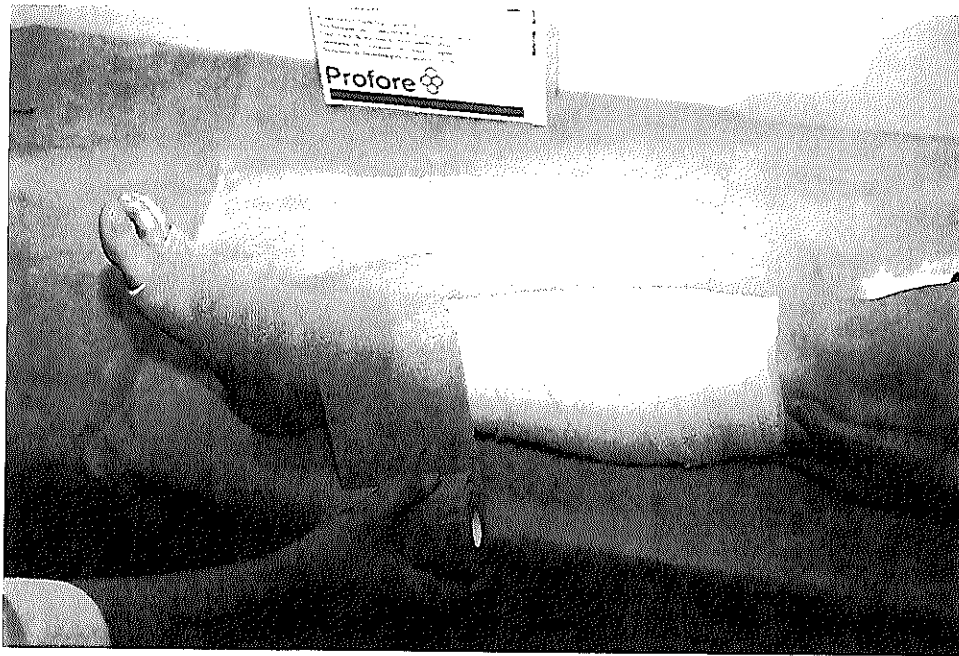


FIGURE 12-5 Compression wraps: layered bandage system.

use of a zinc paste bandage to create a conformable but inelastic “boot” around the leg; thus a paste-type compression wrap is commonly referred to as an Unna’s boot (Weingarten, 2001). Today various inelastic paste wraps exist and are impregnated with any of the following products: zinc, glycerin, gelatin, or calamine (Wipke-Tevis and Sae-Sia, 2004). Thus the paste wraps are not identical and an adverse reaction to one does not predict a reaction to another.

Inelastic paste wraps should be applied without tension, beginning at the base of the toes and extending to the tibial tuberosity below the knee. The patient must be reminded to maintain the foot in a dorsiflexed position while the paste wrap is applied (Box 12-1). Common and appropriate techniques used to ensure a smooth conformable fit include open or closed heel, pleating, reverse folding, and cutting and restarting (Davis and Gray, 2005; Wipke-Tevis and Sae-Sia, 2004). If the paste layer is left open to air, it dries to a “semi-cast” consistency. Often the paste layer is covered with a self-adherent wrap to protect clothing from the paste. Paste bandages should be changed when they begin to loosen, slip, or become saturated (typically in 3–7 days). Problems associated with paste bandages include skin reaction to certain paste ingredients (e.g., calamine), maceration due to lack of an absorptive layer, slippage, poor fit, and inability to bathe (Davis and Gray, 2005). As with all inelastic compression devices, paste bandages are most appropriate for actively ambulating patients.

Short-Stretch (Single-Layer) Reusable Wraps. Short-stretch reusable wraps are inelastic, single-layer wraps that provide sustained compression at a modified or

BOX 12-1 Procedure for Paste Bandage Application

1. Apply gloves after assembling supplies and washing hands.
2. Gently wash and dry extremity. Replace gloves.
3. Place patient in supine position with affected leg elevated and not in dependent position.
4. Foot should be dorsiflexed so foot and leg are at 90-degree angle while applying initial bandage layers around the foot and ankle.
5. Open all paste bandage wrappers and cover wrap. Estimate amount of material based on size of leg(s).
6. Hold paste bandage roll in nondominant hand. Begin to apply bandage at base of toes.
7. If an ulcer is present, apply appropriate topical dressing to ulcer and secondary dressing (if indicated) before applying paste bandage.
8. Treat periwound (if indicated) and moisturize rest of leg.
9. Wrap twice around base of toes without using tension.
10. Continue wrapping bandage around foot, ankle, and heel, using a circular technique, with each strip overlapping previous strip by approximately 50% to 80%. Do not apply tension to the wrap.
11. Smooth paste bandage while applying and remove any wrinkles and folds (may pleat, reverse fold, or cut to ensure smooth bandage).
12. Wrap up to knee and finish smoothing.
13. Remove gloves.
14. Apply cover wrap using recommended amount of tension (e.g., 50% stretch) and 50% overlap.
15. Remove twice weekly or weekly as indicated by leakage, slippage, hygiene, wound care, complaint of numbness, or anticipated decrease in edema.

therapeutic level. Because they are inelastic they are most appropriate for the actively ambulating patient. These short-stretch wraps or single-layer wraps must not be confused with *Ace-type bandages*, which provide low levels of compression and are not considered therapeutic for patients with venous hypertension or insufficiency. *Ace-type bandages* tend to stretch when the calf expands and thus fail to provide calf muscle support during ambulation. In addition, they are very user dependent and are frequently applied incorrectly.

A major advantage of short-stretch reusable wraps is their “wash and reuse” feature. This feature permits more frequent removal of wraps for bathing and dressing changes and contributes to cost-effective care. Many of these wraps incorporate a visual indicator of correct tension, which is advantageous when teaching caregivers how to apply the wrap correctly to achieve the prescribed level of pressure (Wipke-Tevis and Sae-Sia, 2004).

Long-Stretch Reusable Wrap. The long-stretch wrap is also a reusable, single-layer product that provides sustained compression at either a therapeutic or modified level of pressure. However, the distinctive feature of the long-stretch reusable wrap is that it is elastic so it can be used for both the sedentary and the actively ambulating patient. Much like the short-stretch wrap, the long-stretch wrap also can be washed and reused and should not be confused with *Ace-type bandages*.

COMPRESSION GARMENTS

Compression garments are essentially cloth products that can be reused: reusable inelastic device, tubular sleeve, and compression stockings.

Stockings

Compression stockings are reusable, elastic garments that are most commonly used for patients with stable venous insufficiency to prevent ulceration (either initial or recurrent). These garments are appropriate for sedentary as well as actively ambulating patients. **Compression stockings must not be confused with antiembolism stockings, which provide 15 to 17 mm Hg of pressure and are therefore not appropriate for therapeutic compression (WOCN Society, 2005).**

Stockings are generally not a good choice for compression during the initiation of therapy because of the rapid changes in limb circumference associated with edema reduction. They should be used once the edema has been controlled and limb circumference has stabilized. Another relative contraindication for stocking use is severe lipodermatosclerosis because the “inverted champagne bottle” configuration of the leg typical of this condition makes obtaining a good fit difficult. If necessary, however, customized stockings can be made after a referral to a trained “stocking fitter.”

Stockings are available in a variety of colors, styles and sizes. Accurate measurement for safe sizing must be done according to careful review of the manufacturer’s instructions. Stockings may be knee high or thigh high; most patients with venous insufficiency are effectively managed with knee-high stockings. Compression stockings are classified according to the pressure produced at the ankle (Kline et al, 2008): class I (light support), class II (medium support), class III (strong support), and class IV (very strong support). The actual amount of pressure (in mm Hg) appointed to each classification varies by country and manufacturer. Compression stockings are produced as either a circular knit or a flat knit garment. The circular knit stocking is a thin, lightweight material without a seam; it is not available in high levels of compression (Moffatt et al, 2007).

Stockings are the “mainstay” of maintenance compression but are effective only if the patient wears them. Therefore it is critical for the wound specialist to educate and ensure that the patient understands the importance of life-long compression and is able to correctly don the stocking (Moffatt et al, 2007). If barriers are identified, resources and devices to assist with application or other options for compression must be explored. Tips for applying compression stockings (including assist devices) and other key points for patient education are outlined in Box 12-2. One example of a device that facilitates stocking application is shown in Figure 12-6.

Tubular Sleeve

The tubular sleeve is reusable, elastic and, when measured and applied correctly as a double layer, provides sustained compression at a modified level of pressure (O’Meara et al, 2009). A tubular sleeve may be selected when the patient cannot tolerate other types of compression. It also may be used as a temporary intervention while a more permanent solution is pending. Application and removal are easy and require minimal, if any, education; therefore the tubular sleeve is an excellent option when simplicity is the top priority.

Reusable Inelastic Device

Many of the compression garments and devices used for managing lymphedema can be used to manage the edema associated with venous hypertension. For example, the CircAid (CircAid Medical Products, San Diego, CA, USA) is an inelastic reusable compression device that is secured with overlapping bands. By securing the bands according to the pressure indicators, the device can provide therapeutic or modified levels of pressure (Figure 12-7). The ability to easily reapply and readjust the straps helps prevent slippage as edema fluctuates and permits frequent bathing and wound care. Ease of

BOX 12-2 Patient Education: Compression Stockings

Tips for Putting On Stockings

- Don stockings immediately upon awakening, before getting out of bed.
- For easier application of stockings, wear rubber gloves; apply talcum powder (light dusting) first to foot and leg.
- Apply heavy stockings over light silk stocking or silky stocking "liner."
- Use commercial device designed to facilitate stocking application:
 - Stocking butler or donning gloves (Jobst)
 - Easy-slide toe sleeves for open-toe stockings (Jobst, Juzo, Sigvaris)
 - Stocking donner (Beiersdorf-Jobst)
 - Slippie Gator (Juzo)
- Wash new stockings before wearing (follow manufacturer's directions) to reduce stiffness and difficulty in application.
- Use a "layered" approach: either two-piece stockings (TheraPress DUO) or two layers of lower-compression stockings (e.g., two layers of stocking, each of which provides 15 mm Hg compression).
- Turn leg portion of stocking inside-out down to heel. With stocking stretched, slip foot in while pulling stocking by its folded edge over heel. Gently work stocking up leg, gradually turning stocking right-side out.
- Conduct foot exercises with stockings on: move toes in circular motion (make big circles) both clockwise and counterclockwise. Repeat exercise at least 10 times per day.

Care and Management

- Purchase two pairs of stockings to permit laundering.
- Launder with mild detergent and line dry (follow manufacturer's guidelines).
- Replace stockings every 3–4 months to maintain therapeutic efficacy.
- If stockings become too tight or too loose, contact wound care nurse, wound program, or physician for refitting.
- For person having problems with stocking sliding down, use roll-on adhesive applicator (It Stays by Jobst).

removal and application may improve compliance among individuals who are unable to tolerate other forms of compression therapy. The product is washable and reusable, and it comes with a warranty for 3–6 months.

INTERMITTENT PNEUMATIC COMPRESSION (IPC)

IPC (also known as *dynamic compression therapy*) is a reusable compression device that involves the use of an air pump to intermittently inflate a sleeve applied to the lower extremity (Figure 12-8). IPC may be used for patients with LEVD who are mobile or for those who are immobile and need higher levels of compression than can be provided with stockings or wraps. IPC may be used as adjunct therapy to sustained compression, or an alternative for patients unable to tolerate sustained compression or who are too compromised for sustained



FIGURE 12-6 Application of a therapeutic support stocking with a "stocking donner."



FIGURE 12-7 An example of an inelastic reusable device. (Courtesy CircAid® Medical Products, San Diego, CA).

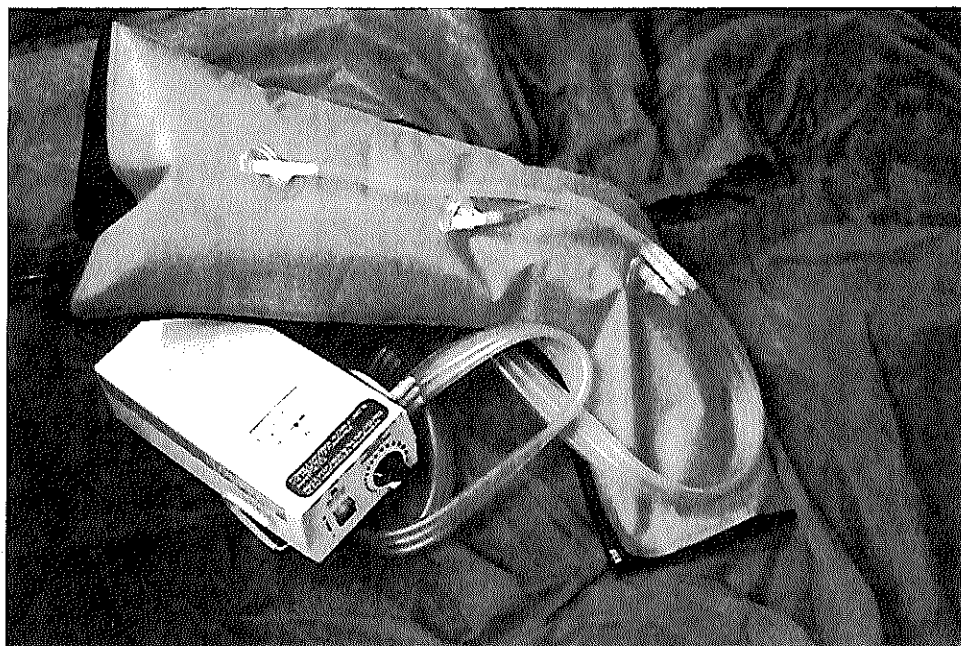


FIGURE 12-8 Dynamic compression device: sequential compression therapy.

compression (Robson et al, 2006). The basic effects of IPC are increase venous velocity, reduce edema, increase popliteal artery blood flow and increase nitric oxide synthase (Comerota, 2009).

Intermittent compression devices vary in terms of the inflation–deflation cycle, amount of pressure exerted against the leg, and number of compartments in the sleeve. Single-compartment sleeves simply inflate and deflate on a cyclic basis, whereas multicompartments sleeves provide sequential compression (i.e., distal to proximal “milking” compression wave). Computer-simulated models suggest that sequential compression devices have the greatest impact on venous return (Chen et al, 2001). Typically, patients are instructed to apply the therapy once or twice daily for 1 to 2 hours each time.

Benefits of IPC include mobilization of interstitial (edema) fluid back into the circulation and enhanced venous return without impairing arterial flow. In fact, IPC may actually improve distal perfusion, making the therapy safe for patients with coexisting arterial disease (Kelechi and Bonham, 2008; Hopf et al, 2006). IPC is thought to exert antithrombotic and vasodilatory effects, possibly as a result of the marked increase in velocity of blood flow and the resultant “shear stress” at the level of the endothelial cells (Chen et al, 2001). IPC therapy may contribute to healing of long-standing venous ulcers that have “failed” standard compression therapy. Studies also report higher levels of patient satisfaction and adherence to IPC therapy (Berliner et al, 2003; Mani et al, 2001).

A disadvantage to most IPC devices is the need for the patient to stay immobile during the therapy.

However, a newer dynamic device (MOBILITY1 Derma Sciences) comes with a small compressor that allows mobility of patients while they are receiving optimal therapy. Medicare and Medicaid reimbursement only covers patients with venous ulcers that have “failed” to heal after a 6-month trial of conservative therapy directed by a physician. The trial of conservative therapy would be expected to include a compression bandage system or garment, an appropriate dressing for the ulcer, exercise, and elevation of the limb(s) (Medicare Determination Manual, 2010).

LOCAL WOUND CARE

As with all wounds, topical therapy for the venous wound is selected based upon wound characteristics (see Chapter 18). Initially, the venous ulcer may present with copious amounts of exudate. Black necrotic tissue is seldom seen in venous ulcers except when infection or trauma is present. Therefore an appropriate rule of thumb is to select dressings that minimize potential allergens while effectively managing the exudate to prevent periwound maceration and control bioburden. Interventions to prevent and manage venous dermatitis were described earlier in the chapter. As edema decreases, the volume of exudate from the venous ulcer also will diminish, and the types of dressings will need to be modified.

When a wound fails to progress, the entire treatment plan must be reevaluated; if the plan remains appropriate and implemented, the diagnosis should be reevaluated. Many other causes of lower extremity ulcers can present as venous ulcers, such as mixed venous/arterial, lymphedema, vasculitis, autoimmune disease, and malignancy.

If a biopsy and differential diagnosis again confirm a venous etiology, failure to heal may be due to the negative cellular environment of a chronic wound. In this case, a product or therapy designed to convert the chronic wound environment into an environment that supports repair should be considered. Skin grafts, bioengineered human skin equivalents, negative pressure wound therapy, electrical stimulation, and selected growth have shown varying degrees of success in the management of refractory venous ulcers if the underlying cause is appropriately addressed. However, laser therapy, phototherapy, and ultrasound therapy have not been shown to statistically improve venous healing (Robson et al, 2006). Biophysical and biological agents are described in detail in Chapters 19 through 24.

SURGICAL INTERVENTIONS

Appropriate topical therapy and compression are not sufficient to heal all venous ulcers (Robson et al, 2006). Factors associated with failure to heal include increased ulcer size (>5 cm²), longer duration (>6 months), and failure to show significant progress toward healing during the first 3 to 4 weeks of compression therapy. Coexisting arterial disease, persistence of fibrin throughout the wound bed, reduced mobility, and history of vein ligation or knee or hip replacement have been reported as negative prognostic indicators (Paquette and Falanga, 2002).

The procedure of choice for patients with significant perforator and/or deep vein incompetence is ligation of the incompetent perforator veins, which acts to prevent transmission of the elevated pressures within the deep system to the vulnerable superficial veins and tissues. This procedure may be combined with superficial vein stripping for patients who also have significant saphenous vein incompetence. In the past, ligation of perforator veins was performed as an open (Linton) procedure, but the classic Linton procedure has fallen out of favor. In its place is an endoscopic procedure—subfascial endoscopic perforator surgery (SEPS). Wide excision of diseased tissue and free flap transfer of healthy tissue with its own microvasculature and uninjured venous valves can benefit the patient with severe lipodermatosclerosis and persistent, recurrent ulcers (Robson et al, 2006). The Wound Healing Society Guidelines recommend the SEPS procedure of choice to address underlining venous etiology of the venous ulcer by preventing backflow from deep to the superficial venous system. The procedure is not effective if the patient has severe deep venous disease with either reflux or obstruction (Robson et al, 2008).

Less extensive procedures are available for the patient who has not responded to conservative treatments. Duplex Doppler studies can demonstrate an intact deep venous system with abnormal perforators or superficial valves (Tenbrook et al, 2004). Procedures such as superficial (saphenous) venous ablation, endovenous laser ablation, and valvuloplasty can help to decrease venous

hypertension and prevent the recurrence of venous ulcers when combined with compression therapy (Robson et al, 2006; Vowden and Vowden, 2006). A new approach of injecting ultrasound-guided foam into refluxing superficial and perforator veins has been reported to be safe and effective (Whiddon, 2007).

FOLLOW-UP AND LIFELONG MAINTENANCE

With recurrence rates ranging between 26% and 69% during the first 12 months following ulcer healing, the emphasis of management must shift to prevention of recurrence once the ulcer is healed (Nelson et al, 2000). Lifelong exercise, weight control, and compression therapy are ongoing challenges. Many patients fail to utilize compression consistently for a variety of reasons (Jull et al, 2004; Nelson et al, 2000). In one study, the two factors that were most predictive of patients' continued use of compression therapy were their perception of the value of compression and their level of comfort/discomfort with the stockings (Jull et al, 2004).

These findings clearly speak to the importance of effectively communicating with the patient, stressing the importance of lifelong compression and identifying barriers and solutions to the issues that impair intervention adaptation (Wipke-Tevis and Sae-Sia, 2004). Solutions may involve placing the patient in a lower level of compression so that he or she is able to apply the garment. Although some evidence suggests that high-level compression stockings are more effective in preventing recurrence, other evidence indicates that medium-level compression stockings are associated with significantly higher compliance rates (Jull et al, 2004; Nelson et al, 2000). The Wound Healing Society's Guidelines recommend the use of compression stockings constantly and forever and attempts must be made to aid the patient's compliance (Robson, 2008). Education that includes the families and caregivers as well as the patient is critical to achieving optimal outcomes (Burrows et al, 2007). Chapter 29 provides more strategies for facilitating the patient's adaptation to therapy rather than simply labeling the patient as "noncompliant."

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