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Operative Debridements of Chronic Wounds

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The term "chronic wound" does not refer to duration over time, but rather describes a wound that is physiologically impaired. All venous, pressure, and diabetic foot ulcers are defined as chronic wounds. Elderly patients are more likely to experience venous and pressure ulcers,[1–4] which lead to more than half of all lower extremity amputations in persons with diabetes.[5] Chronic wounds heal at the same frequency of closure in elderly populations as they do in younger populations, but may heal at a slower rate, primarily because of comorbidities associated with age.[6–9] The comorbidities that delay healing are prevalent among older populations and include venous insufficiency and diabetes. Although there are age-related changes to the skin, it has yet to be shown, clinically, that age alone decreases an elderly person's ability to heal.[7,10,11] A synergistic effect of advanced age and diabetes significantly slows healing.[12]

Chronic wounds in elderly patients heal successfully if the care regimen includes a coordinated effort to treat skin breakdown early and to prevent further ulceration. In the absence of ischemia and osteomyelitis, prompt medical treatment will heal most venous ulcers, diabetic foot ulcers, and stage I, II, and III pressure ulcers (defined below).[6] Delayed wound treatment, combined with multiple comorbidities affecting the elderly population, can lead to amputations, sepsis, and death. The established pathway from untreated chronic wound to death has been used as evidence for the manslaughter convictions of several care providers of the elderly.[13]

Pain is a complex and almost universal complication of this population, and appropriate pain management by an anesthesiologist is becoming increasingly recognized as essential in the optimal treatment of these patients.

The Operating Room

The only universally accepted treatment for chronic wounds is surgical debridement. This is the standard of care for nonviable and infected tissue and for the stimulation of healing.[14] Because these patients usually have multiple comorbidities and American Society of Anesthesiologists (ASA) scores of 3 or 4, we recommend operative debridement under local or regional anesthesia whenever possible. Regional blocks of the sciatic, popliteal, and femoral nerves are ideal. Many patients will require general anesthesia. It is essential that the patient and primary care physician understand this, as well as understand that the risks of not performing the debridement, i.e., leaving necrotic or infected tissue in an elderly person, is greater than the risks of anesthesia itself. For patients with peripheral arterial disease, treatment of underlying ischemia must be achieved before elective debridements unless infection is present.

Chronic Wounds in the Elderly Result in Significant Morbidity and Mortality

Diabetic Foot Ulcers

There are 20.8 million Americans diagnosed with diabetes.[15] The elderly are the largest constituent of this group because diabetes affects more than 18% of Americans greater than 60 years old.[16] In the United States between 1997 and 2004, the number of new cases of diabetes increased 54%.[17] By the year 2030, 366 million persons worldwide are estimated to have diabetes, with 130 million of them over the age of 64.[18] All of these patients are at risk for diabetic complications including retinopathy, nephropathy, neuropathy, and accelerated atherosclerosis. Neuropathy and atherosclerosis are associated with the development of diabetic foot ulcers and impaired healing.[19–21]

In the United States, the elderly account for 53.3% of diabetes-associated amputations per annum.[5] Diabetic foot ulcers are defined as any breakdown of skin on the foot of a diabetic person. Recent prevalence is as high as 12% of all people with diabetes.[22] These ulcers act

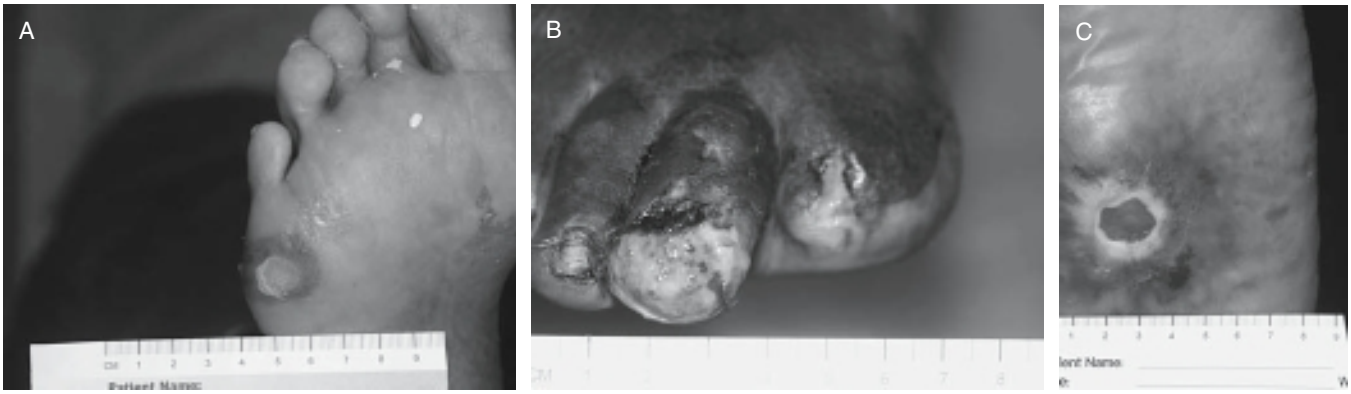


FIGURE 12-1. Examples of diabetic foot ulcers. Note: any break in the epidermis on the foot of a patient with diabetes is considered a diabetic foot ulcer. **A:** Typical-appearing ulcer under the fifth metatarsal head. **B:** A more advanced ulcer on the toe. **C:** A callous surrounded by central ulceration on the plantar aspect of the foot.

as portals for infectious organisms. A large multicenter study recently reported 58% of all patients with ulcers had concomitant foot infection.[23] Half of the patients who develop one foot lesion subsequently develop a contralateral wound.[24] Each wound is considered to be chronic from its inception and should be treated early. Individuals with diabetes have a 30- to 40-fold higher risk of lower limb amputation.[25] See Figure 12-1A–C for examples of the typical locations of diabetic foot ulcers.

Pressure Ulcers

The four stages of pressure ulcers are defined as[26,27]:

Stage I—Observable pressure-related alteration of intact skin whose indicators as compared with the adjacent or opposite area on the body may include changes in one or more of the following: skin temperature (warmth or coolness), tissue consistency (firm or boggy feel), and/or sensation (pain, itching). The ulcer appears as a defined area of persistent redness in lightly pigmented skin, whereas in darker skin tones the ulcer may appear with persistent red, blue, or purple hues. See Figure 12-2A.

Stage II—Partial-thickness skin loss involving epidermis, dermis, or both. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater. See Figure 12-2B.

Stage III—Full-thickness skin loss involving damage to, or necrosis of, subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue. See Figure 12-2C.

Stage IV—Full-thickness skin loss with ulceration extending through the fascia, with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (e.g., tendon, joint, capsule). Undermining and sinus tracts are frequently associated with stage IV pressure ulcers. See Figure 12-2D.

In 2003, 455,000 patients in the United States alone were hospitalized for pressure ulcers, representing a 63% increase from 1993: the most common reason for admission was septicemia.[28] The true incidence and prevalence of pressure ulcers is not known. A recent national study of acute care settings found that the prevalence of pressure ulcers ranged between 14% and 17%, whereas the incidence was between 7% and 9%.[29] At least 10% of hospitalized patients, more than 20% of nursing home patients, and 20%–30% of spinal cord injury patients are affected.[30] In a nursing home study, 6.5%–19.3% of patients developed a new pressure ulcer over a 3- to 21-month period. Patients at highest risk were those who had diabetes or fecal incontinence.[31] Recent studies have demonstrated that both age and immobility are strongly linked to development of pressure ulcers[32,33] as well as cognitive ability.[34]

The presence of a pressure ulcer doubles the risk of mortality in an elderly patient.[35,36] Elderly patients discharged with a hospital-acquired pressure ulcer have a much greater risk of death within a year than patients without a pressure ulcer, indicating that stage IV pressure ulcers should never be ignored.[37] A recent study of more than 100,000 patients revealed an overall age-adjusted mortality rate of 3.79%, for which pressure ulcers were listed as the cause of death in 18.7% of patients.[38] Stage IV pressure ulcers often lead to sepsis, a common cause of death in the elderly.[39,40]

Venous Ulcers

It is estimated that 1.7% of the elderly population is affected by venous ulcers, indicating that they are more prevalent in the elderly than in younger populations.[41–43]

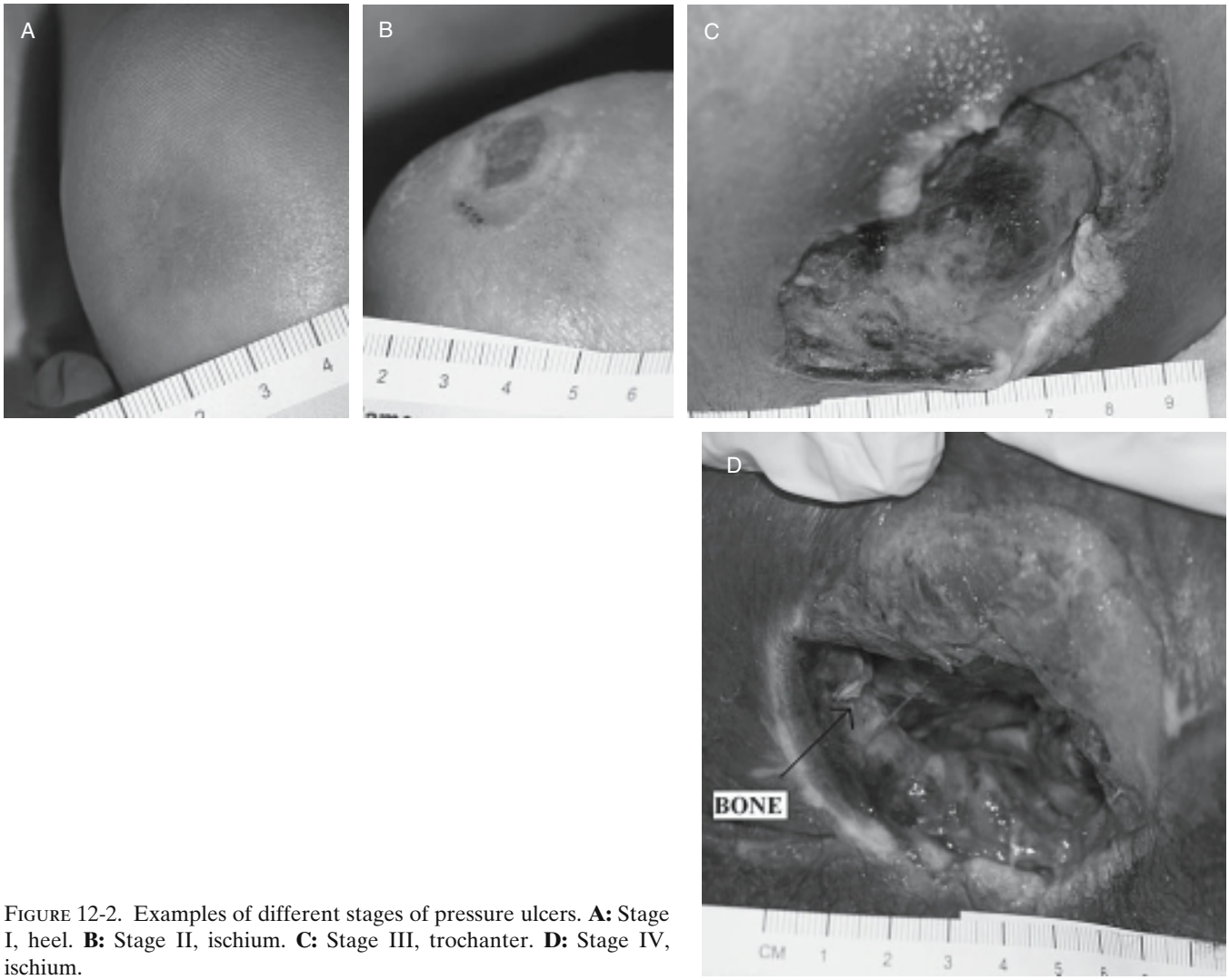


FIGURE 12-2. Examples of different stages of pressure ulcers. **A:** Stage I, heel. **B:** Stage II, ischium. **C:** Stage III, trochanter. **D:** Stage IV, ischium.

Venous ulcers are often misdiagnosed as traumatic injuries and are therefore undertreated.[44] Although these wounds are not frequently associated with osteomyelitis or amputation, when undertreated, they provide a gateway for infection that results in multiple hospitalizations, substantial suffering, and health care costs exceeding $1 billion annually.[45,46]

Venous ulcers are secondary to venous reflux disease, which correlates with increased age.[41,47,48] An example of a typical venous ulcer is shown in Figure 12-3. Venous reflux disease occurs when valvular incompetence prevents normal blood flow from superficial veins to deep veins. The most common etiologies for valvular dysfunction of the deep venous system are advanced age[41,49] and a history of deep vein thrombosis (DVT).[50] Valvular incompetence causes increased venous pressures, leading to venous distention and activation of apoptotic pathways, resulting in ulceration. Another hypothesis is that leukocytes and other large molecules become trapped in the dermis in response to endothelial damage and venous hypertension.[51] It is thought the extravasation of these large molecules, proteins, and leukocytes inhibits growth factors from reaching their targets.[52]

Ischemic Wounds

Peripheral arterial disease is often the primary etiology of an ischemic wound. It can also impair healing in venous, pressure, and diabetic foot ulcers because of a decreased blood flow to the affected area. Age, hypertension, smoking, and diabetes are each independent risk factors for developing peripheral arterial disease.[4,53] Although the incidence of peripheral arterial disease is only 4.3% in patients over 40 years old, the incidence sharply increases to 14.5% in patients over 70.[4] Additionally, a large study of Asian patients with diabetes over the age

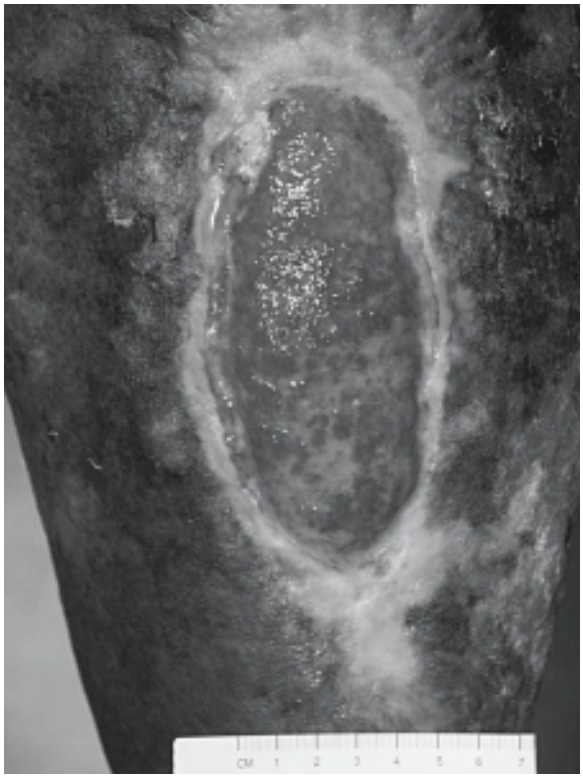


FIGURE 12-3. Example of a typical lower extremity venous ulcer.

of 50 showed the prevalence of peripheral arterial disease to be 17.7%.[54] Many if not most of these patients are asymptomatic.[55,56]

Experimental Evidence of Physiologic Impairments in the Elderly

Clinical and experimental studies have demonstrated that there is a greater frequency of physiologic impairments to wound healing in the elderly population.

Angiogenesis in Wound Healing

Laboratory research has identified more than 30 regulatory mechanisms of angiogenesis that occur during wound healing, including growth factors, growth factor receptors, chemotactic agents, and matrix metalloproteinases. In animal models, decreased angiogenesis significantly inhibits wound healing.[16,57–59] In these models, angiogenic cytokines such as vascular endothelial growth factor are present in smaller concentrations in the wounds of older animals compared with younger animals, resulting in smaller capillary densities within the wound bed.[60–62] It is theorized that the discrepancy is secondary to decreased macrophage function in the older animals.[63–65] The decrease in angiogenesis causes an initial delay in wound healing; yet, despite this, wounds contract with comparable frequency.[66] Therefore, a decreased angiogenic response in older animals contributes to an impaired wound-healing rate.

Decreased Immune Response

Review of experimental and clinical research has shown physiologic impairments in the immune response of the elderly, making them prone to infection.[67] Older animals have a markedly decreased adaptive immune response (B and T cells),[67] a bolstered but possibly dysfunctional regulatory T cell population (CD4+ CD25+),[68,69] a decrease in T cell receptor diversity,[70] and a decrease in toll-like receptors[65] and γδ-T cells.[71] The decreased immune response makes the elderly more susceptible to pathogenic invasion.

A Multidisciplinary Approach for Treating Venous, Pressure, and Diabetic Foot Ulcers

Many wounds that appear minor on initial physical examination signal extensive necrosis beneath the skin's surface and consequently may still be a significant source of sepsis (Figure 12-4). It is therefore critical to treat all wounds early and comprehensively. In the elderly population, patient care frequently focuses on the patient's comorbidities, whereas wounds are simply covered with a bandage and ignored. Too often, untreated wounds lead to preventable complications such as amputation, sepsis, and death. As part of an ongoing multidisciplinary collaboration among many specialists who care for elderly patients with chronic wounds, we have developed protocols to treat venous,[44] pressure, and diabetic foot ulcers.[6,44,72,73] Outlined below are the precepts of our treatment protocol (Table 12-1).

1. Contact the Primary Care Physician:

A strong relationship with the primary care physician is essential to optimize the many comorbidities of the elderly patient. Common medical diseases that must be assessed include: coronary artery disease, diabetes, obesity, hypertension, dyslipidemia, chronic renal insufficiency, malnutrition, muscular atrophy, hepatic disease, chronic obstructive pulmonary disease, coagulopathy, and pain. In the diabetic population, seeking the counsel of a diabetologist may be necessary to maintain proper glucose control. The primary care physician, along with the assistance of a nutritionist, can also be helpful in evaluating the patient's nutritional status. A small number of prospective studies has found that nutritional

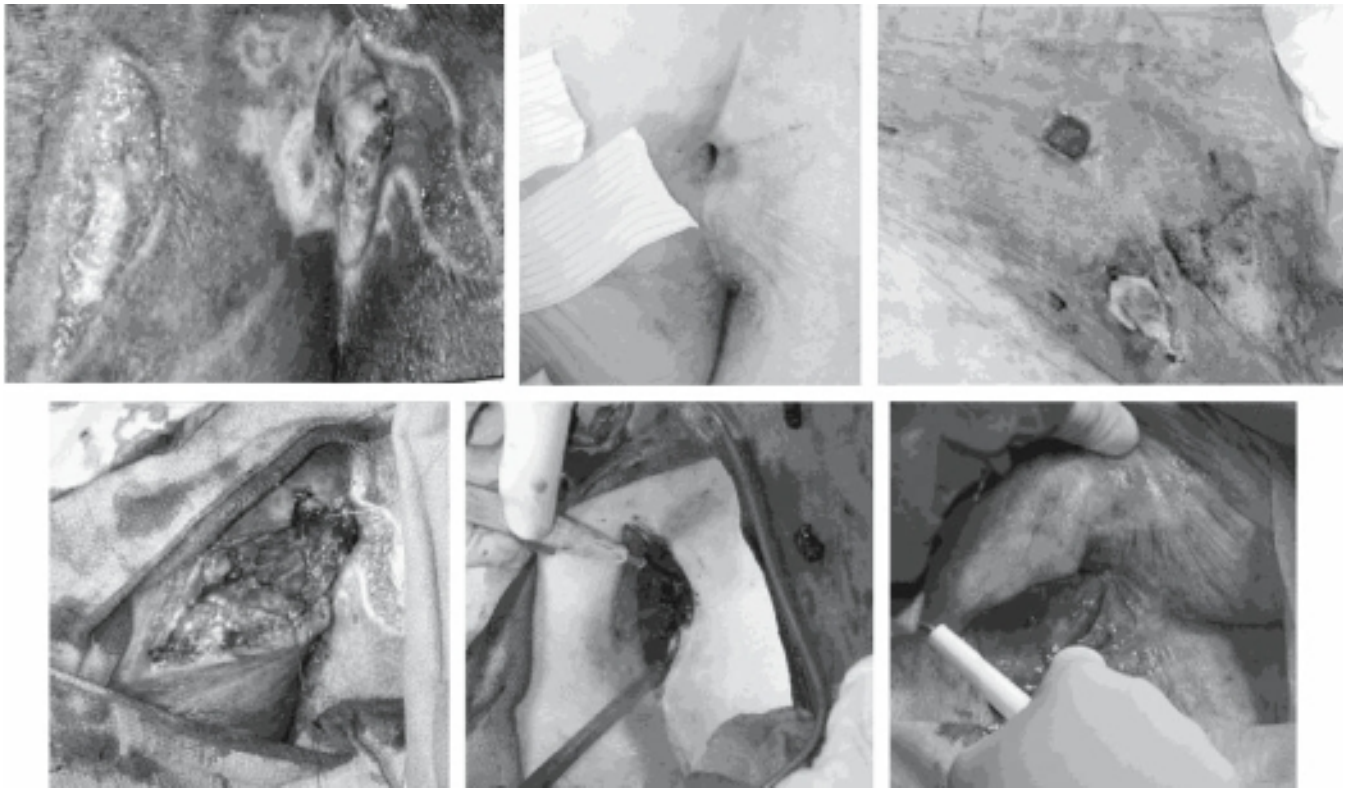


FIGURE 12-4. Pressure ulcers can be deceptively large. Pressure ulcers are often much larger than they appear to be. Wounds that may look quite small can contain extensive undermining and/or tunneling. This figure demonstrates three cases in which, upon debridement, the wound was shown to be substantially larger than it appeared at presentation.

supplements are beneficial in treating chronic wounds in malnourished patients.[74–76]

2. Comprehensive Physical Examination of At-Risk Patients:

TABLE 12-1. Summary of the current standard of care for the healing of venous ulcers, pressure ulcers, and diabetic foot ulcers.

1. Contact the primary care physician
2. Comprehensive physical examination of at-risk patients
3. Prevention of deep vein thrombosis
4. Laboratory and radiologic evaluations
5. Evaluation of blood flow in the lower extremities
6. Objective measurement of every wound weekly with digital photography, planimetry, and documentation of the wound-healing process
7. Elimination of cellulitis, drainage, and infection
8. Local wound care
9. Debridement of nonviable tissue and wound bed preparation
10. Offloading pressure from the wound and compression therapy
11. Growth factor therapies
12. Addressing comorbidities that may affect anesthesia
13. Physical therapy
14. Pain management

Diabetic and bed-bound patients are at high risk for developing diabetic foot and pressure ulcers, respectively. These patients often lack protective sensation. As a consequence, they do not sense skin breakdown and are unable to assess wound progression. All diabetics must have their feet examined daily for evidence of skin breakdown. All bed-bound patients should have their pressure points (heels, ischia, trochanters, and sacrum) examined daily for evidence of pressure ulcer development.

3. Prevention of Deep Vein Thrombosis:

The physical therapy team should ensure that every patient who is able to ambulate is doing so. In nonambulatory patients, we recommend moving from the bed to a chair at least twice a day. Standard deep vein prophylaxis is mandatory, including pneumatic compression boots and subcutaneous heparin or low-molecular-weight heparin.

4. Laboratory and Radiologic Evaluation:

Baseline laboratory tests should be obtained to evaluate the patient's overall health and to potentially detect underlying disease states. We recommend a complete blood count with differential, coagulation profile, creatinine and blood urea nitrogen levels, electrolyte panel,

lipid panel, glycosylated hemoglobin, albumin and prealbumin, erythrocyte sedimentation rate, hepatic panel, and thyroid-stimulating hormone level. Plain films of the affected area are recommended for all leg and foot wounds. Magnetic resonance imaging or bone scan is recommended to assess for osteomyelitis in stage IV pressure ulcers and in diabetic foot ulcers.

5. Evaluation of Blood Flow in the Lower Extremities:

Peripheral arterial disease and venous stasis disease are prevalent in the elderly population. All patients with limb ulcers should undergo arterial testing, regardless of what the primary etiology is thought to be, because the etiology may be multifactorial. Arterial testing is crucial because patients with ischemia should be revascularized before debridement and should not undergo compression therapy. Testing for venous stasis disease should be done in patients who clinically appear to have venous stasis ulcers.

Noninvasive flow studies, which include bilateral ankle brachial indices (comparison of pressures in ankles and arms) and pulse volume recordings (to determine the amount of blood flow when pressures are falsely elevated), are necessary to detect lower extremity ischemia. In particular, recent studies have suggested high sensitivity to detect peripheral arterial disease using the low ankle pressure test defined as the quotient of the lowest ankle artery pressure of two measurements and the highest of two brachial artery pressure measurements.[77,78] Depression of these values is associated with a greater risk of amputation.[79] An ankle brachial index less than 0.9 indicates significant arterial disease that requires referral to a vascular surgeon.[80] An elevated ankle brachial index greater than 1.30 has been found to be predictive of major amputation[81] and, particularly in the context of poor waveforms (i.e., monophasic),[82] may indicate atherosclerosis requiring vascular surgery referral.

When indicated, revascularization should proceed as soon as possible. Bypass grafting procedures are often avoided in elderly patients because their comorbidities make them poor surgical candidates. Alternatively, endovascular revascularization provides a minimally invasive and effective surgical option. Endovascular correction of arterial disease has proven safe and leads to 5-year limb salvage rates of more than 89% in diabetic and elderly populations.[83–87] If wound debridement is necessary, it should be done shortly after revascularization so as to utilize the improved blood supply.[88]

Duplex ultrasonography testing determines the presence and degree of venous insufficiency in patients with venous ulcers. Because venous incompetence is often attributable to a previous DVT, it is possible that the patient may concurrently have a DVT and a venous ulcer. In this scenario, the DVT will be identified by the venous flow studies.

Once venous ulcers have healed, venous insufficiency should be corrected. Minimally invasive techniques such as radiofrequency ablation of the greater saphenous vein, percutaneous vein valve bioprosthesis, and subfascial endoscopic perforator vein surgery are treatment options.[89–91] Correction of the underlying venous disease prevents recurrence of venous ulcers.[92–94]

6. Objective Assessment of Wound Healing:

Weekly wound-healing assessments should be objectively calculated by digital photography and planimetric measurements. Although a simple ruler has been shown to be reliable for predicting wound healing,[95] digital photography and computer-based wound measurements are more accurate for larger wounds and allow for easy transportation of data. Ideally, all data can be compiled into a wound electronic medical record (WEMR), if available, which plots a wound graph, demonstrating the healing curve of the wound based on planimetric measurements and allowing the team to objectively follow the progress of the wound. Serial objective measurements of the wound area allow the treatment team to accurately and rapidly detect a failure to heal and to adjust the treatment plan accordingly. A WEMR can also store the patient's medical history, wound history (wound graph, drainage, pain, associated pathology, surgery history, radiology, and microbiology), laboratory values, antibiotic history, vascular studies, medications, wound picture, and contact information for the patient's primary care doctor, pharmacist, and next of kin. The WEMR provides all of the patient's pertinent data needed for thorough treatment in an easy-to-read format.

7. Elimination of Infection:

Infection substantially impairs chronic wound healing. Drainage, cellulitis, and pain are indicators of infection. Deep tissue cultures reflect the pathogens populating the wound bed and surrounding tissue, and they allow the physician to tailor the patient's antibiotic regimen to cover only the pathogens grown from cultures, thereby helping to prevent drug-resistant organisms.[96–99] Deep tissue cultures are taken when the wound is debrided. If definitive debridement is not immediately planned, then an initial deep culture should be obtained at bedside, in the emergency room, or in the outpatient clinic, at the time when antibiotics are started.

8. Local Wound Care:

Homecare nursing is an integrated element in the wound-healing team. All wounds must be properly cleaned, treated with topical medications, and covered with the appropriate noncompressive or compressive dressing. Cleaning the wound includes washing with antimicrobial soap and water and scrubbing the wound with sterile saline and gauze. The topical therapy, such as cadexomer iodine (Iodosorb or Iodoflex; Smith & Nephew, Largo, FL), Acticoat (Smith &Nephew), and

Collagenase (Healthpoint, Fort Worth, TX), should provide a moist wound-healing environment and prevent bacterial colonization.

9. Debridement of Nonviable Tissue and Wound Bed Preparation:

Debridement of a chronic wound accelerates healing.[88] It is a recommended treatment for diabetic foot ulcers, pressure ulcers, and venous ulcers.[100–103] For ischemic ulcers, debridement should be deferred until after revascularization. Gene expression is altered in the nonhealing edge of a chronic wound, resulting in hyperkeratotic epidermis and up-regulation of the oncogene c-*myc*.[104] Debriding past this edge into healthy tissue stimulates the healthy epithelium to release growth factors and reduces local inflammation. It is thought that debridement encourages new fibroblasts to invade and replace the senescent cells of chronic wound beds, as well as release of various growth factors that stimulate wound healing, although few rigorous studies demonstrate this.[88] Debridement also allows the surgeon to obtain microbiology and pathology samples that tailor antibiotic treatments and guide future debridements. For patients with active infection (such as cellulitis, fever, or elevated white blood cell count), debridement removes infected necrotic tissue that is the source of infection.

Venous ulcers should be debrided deeply enough to remove all underlying scar and infected tissue; only several millimeters of the surrounding epithelium need to be debrided. Pressure ulcers should be debrided to remove all infected, necrotic, and scarred tissue, as well as to allow deep packing during dressing changes. Diabetic foot ulcers often appear as a callus with a central ulceration. We consider the callus as part of the wound, and we recommend debridement of a diabetic foot ulcer 2–3 mm beyond the callus into healthy epithelium (Figure 12-5).

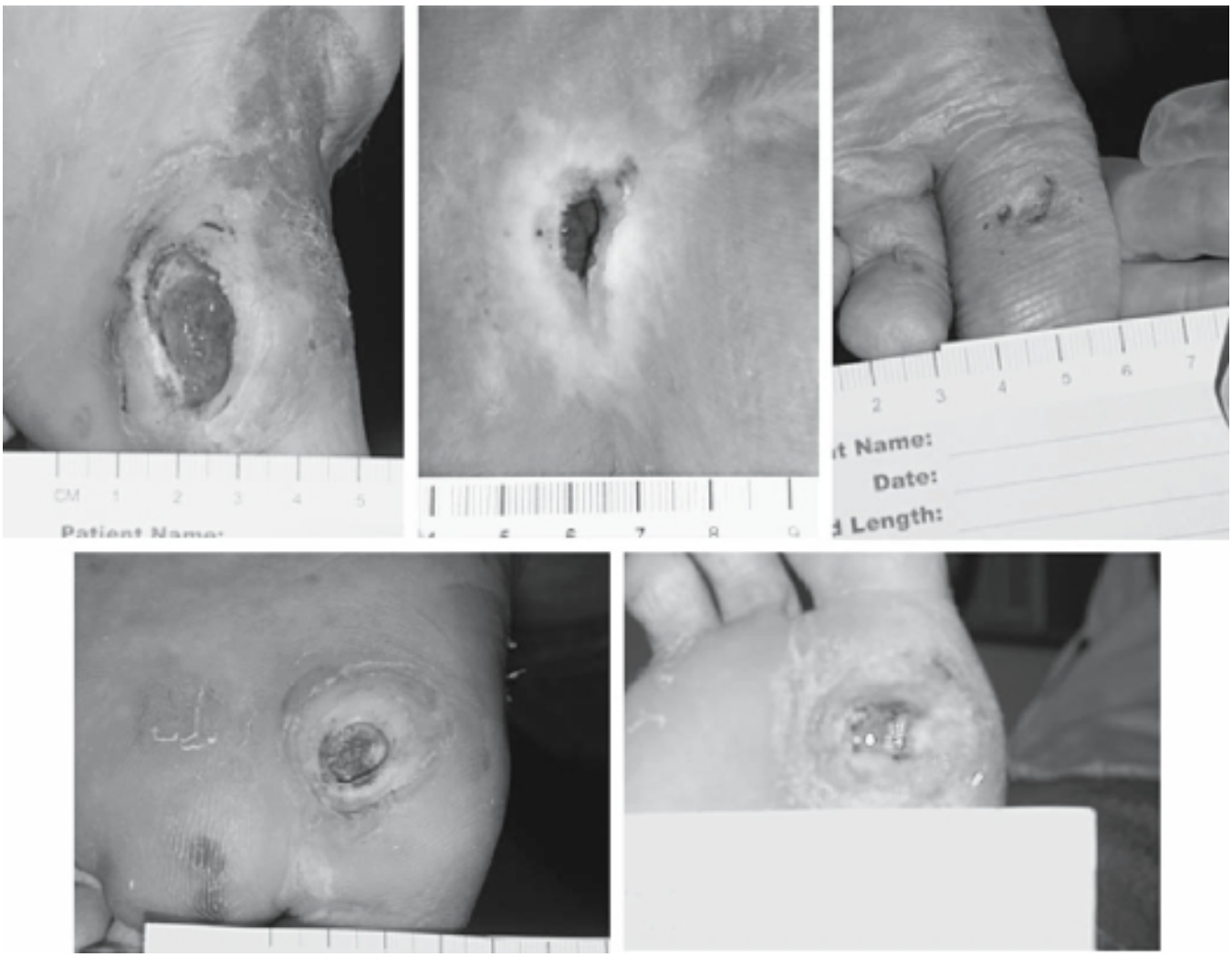


FIGURE 12-5. Proper debridement of diabetic foot ulcers. Diabetic foot ulcers are often associated with a thick hyperkeratotic callus. This callus impedes the healing process and needs to be removed. Debridement of diabetic foot ulcers must extend several millimeters past the nonmigratory edge into healthy epithelium. Debridement will aid in healing by removing nonviable tissue while stimulating reepithelialization with healthy tissue.

The surgeon should take pathology samples from the postoperative wound bed to confirm whether the remaining tissue is healthy. Almost all patients require multiple debridements, and the pathology results will guide the extent of future debridements.

Although it is possible to debride some ulcers at the bedside, many elderly patients are debrided in the operating room because of their significant cardiac and pulmonary comorbidities, the size of their wounds, dementia, pain control, and concerns regarding hemostasis. The personnel involved and close monitoring make the operating room the safest place for most elderly patients who require debridement.

Pressure ulcers often have a significant amount of tunneling and undermining that are revealed only after debridement has begun (Figure 12-4). Bone resection can cause significant bleeding, and most debridements of wounds extending to bone should be considered for the operating room (Figure 12-6). Because of venous reflux disease, venous ulcer debridement often causes significant blood loss from capillary bleeding, varicose veins, and venous perforators. Large venous ulcer debridements should be done in the operating room.

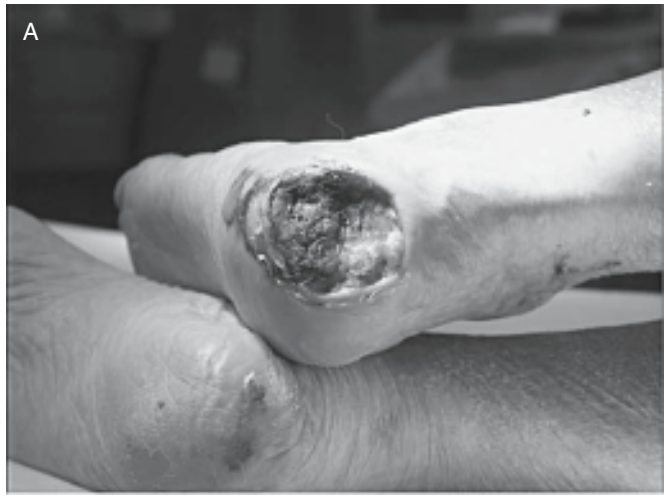
10. Offloading Pressure from the Wound and Compression Therapy:

Pressure is a significant contributor to the development and progression of pressure and diabetic foot ulcers. Pressure can be alleviated in pressure ulcer patients by using specialized air fluidized or alternating air mattresses. Heel pressure ulcer patients should be given a Multi-Podus splint (Restorative Care of America, St. Petersburg, FL) or a foam-based Heelift (DM Systems, Evanston, IL) to relieve pressure. Frequent turning and attentive skin care are mandatory for the treatment of all patients. Many devices have been created to offload pressure from diabetic foot ulcers.[105–109]

For venous ulcers, compression therapy with a measured multilayered bandage such as the Profore system (Smith &Nephew) should be used in conjunction with topical therapies.[110] These bandages decrease superficial vein distention and venous pressure, and they increase the efficacy of venous valves. This promotes the proper flow of blood from superficial to deep veins. High compression is more effective than low compression bandages, but their use is limited to nonischemic patients.[111] It is therefore imperative that all venous ulcer patients be evaluated for arterial insufficiency before compression therapy. Compression therapy should not be applied in the presence of active infection.

11. Growth Factor Therapies:

For the topical treatment of diabetic foot ulcers, the Food and Drug Administration has approved only Dermagraft[112] (Advanced Tissue Sciences, La Jolla, CA), Human Skin Equivalent (HSE), Apligraf,[113] a bilayered, biologically active construct composed of a bovine collagen scaffold seeded with a layer of keratinocytes covering a layer of fibroblasts (Organogenesis Inc., Canton, MA), and Becaplermin (recombinant platelet-derived growth factor)[114,115] for safety and efficacy. Only Apligraf is approved for safety and efficacy in venous ulcers.[116] No treatment is approved for safety and efficacy in pressure ulcers. These agents should not be applied in the presence of active infection (cellulitis or wound drainage) and should be used only after surgical wound debridement.



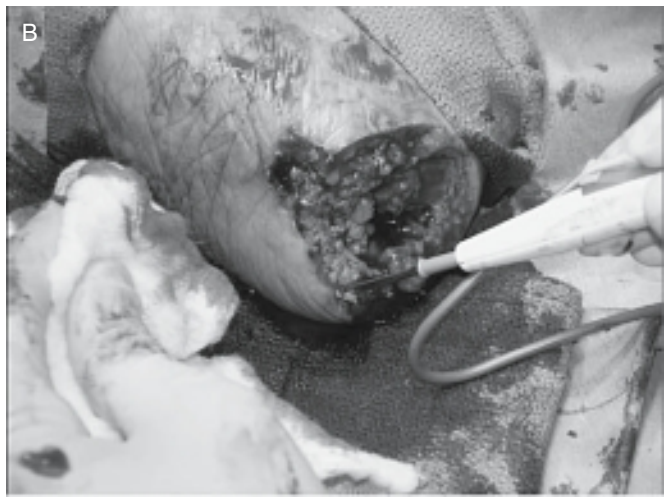


FIGURE 12-6. Proper hemostatic control often necessitates use of the operating room. **A:** Preoperative photo. **B:** Intraoperative debridement. The operating room provides a much higher level of hemostatic control than can be achieved at the bedside. This patient was admitted with a stage IV pressure ulcer. The wound permeated deep into the bone, and because of the large amount of bleeding that can be associated with the debridement of bone in these wounds, this case was safely completed in the operating room.

12. Comorbidities Affecting Anesthesia:

Complicated cardiac conditions are common in the elderly and must be appropriately evaluated before

surgery. If the patient has risk factors for coronary artery disease, an exercise stress test should be considered before surgery. If the patient has a history of coronary atherosclerosis, pacemaker placement, valvular disease, congestive heart failure, or cardiac arrhythmia, cardiac function should optimized.

Nephropathy is a common comorbidity in the elderly. The hypoproteinemia and acidemia seen in end-stage kidney disease patients can significantly affect the pharmacokinetics and pharmacodynamics of certain drugs used in anesthesia.[117] We recommend that end-stage kidney disease patients be dialyzed before surgery and that blood chemistries be closely monitored perioperatively. A venous blood gas is a safe, common approach to rapid evaluation of serum potassium and other electrolytes.

Neuropathy is also a common complication in people with diabetes and most notably in the vast majority of persons with diabetic foot ulcers: in a recent study, 78% of diabetic patients with ulcers also had neuropathy.[118] Because of the decrease in lower extremity sensation, it is rarely necessary to use a stronger anesthetic approach than a regional ankle block or Monitored Anesthesia Care with local anesthetic injections.

Pressure ulcers frequently affect spinal cord injury patients. The level of the spinal cord injury is particularly important. If the patient's injury is above thoracic vertebra 6, then the patient is at risk for autonomic dysreflexia.[119] Autonomic dysreflexia is an abrupt and exaggerated autonomic response to stimuli in patients with spinal cord injuries or dysfunction above the splanchnic sympathetic outflow (T5–6).[120] It is imperative that all patients with a T6 lesion or higher be debrided in the operating room under spinal or general anesthesia and with close blood pressure monitoring.

13. Physical Therapy:

Physical therapy is advised for patients with limited mobility and those with venous ulcers. For patients with limited mobility, physical therapy (1) decreases the incidence of DVT, (2) decreases respiratory complications, (3) increases mental acuity, and (4) decreases the development of contractures.[121] In patients with venous ulcers, musculoskeletal changes attributable to calf pain and venous hypertension dramatically affect the patient's gait. Physical therapy has been shown to improve underlying venous disease and patient ambulation.[122]

14. Pain Management:

Pain is common in patients with ulcers, and effective pain management regimens must be instituted. Pain should be quantified by the patient at each clinic visit. By using a Verbal Analogue Score in conjunction with the wound-healing graph from a wound data sheet, a physician can track the success of pain treatments as they relate to functional outcomes.[123] The end goal is to reduce morbidity by eliminating pain. Pain control also facilitates appropriate cleaning and dressing of the wound, because patients may avoid these tasks if they are very painful. It may be necessary to incorporate a pain specialist into the treatment team.

Each category of chronic wounds presents with a unique type of pain. In venous ulcers, pain is believed to occur as a result of tissue damage, which stimulates the release of inflammatory mediators, sensitizing peripheral somatic pain receptors.[124] Because of intense pain in many venous ulcer patients, we recommend using a tiered system of pain medications based on the World Health Organization analgesic ladder,[125] in which patients are started on nonopioid drugs with or without adjuvant medications, to which increasing strengths of opioid medications are added depending on pain-control needs.[123,124] Patients with multiple comorbidities may have contraindications to common pain medications. In addition, in elderly patients, special attention must be given to the potential side effects of pain medications, such as constipation or mental status changes from narcotics.

Although often associated with neuropathy, diabetic foot ulcers and pressure ulcers can present with substantial pain management challenges. The pain associated with neuropathic diabetic foot ulcers can be treated with tricyclic antidepressants, anticonvulsants, capsaicin, mexiletine, lidocaine patches, *N*-methyl-D-aspartate (NMDA) inhibitors, clonidine, and tramadol.[124] All have been used with varying degrees of success. Spinal cord injury patients with pressure ulcers develop central pain that occurs when there is neuropathy in the area of the wound. Thus, spinal cord patients may also need pain control even though the wounded area is insensate.

Intraoperative Risk and Precautions for Development of Pressure Ulcers

Elderly patients with fragile skin and comorbid conditions are at highest risk for development of intraoperative pressure ulcers. Patients with a high ASA grade may be more likely to develop pressure ulcers.[126] The most comprehensive study was conducted in the United States in 1998 involving 104 hospitals and 1128 surgical patients, all undergoing procedures longer than 3 hours in duration. An overall prevalence of stage I and stage II pressure ulcers was 7.8% when examining patients up to 4 days postoperatively.[127]

Part of the intraoperative risk can be assessed by type of procedure. Vascular surgery, cardiac surgery, and orthopedic surgery are associated with higher risks, although rigorous studies have yet to emerge.[128] The risk may double for operations lasting more than 2.5 hours.[129] In a prospective Dutch study[130] of patients undergoing operations lasting more than 4 hours, 44 patients (21.2%)

developed 70 pressure ulcers in the first 2 days postoperatively. All but three were stage I and II.

Various locations may be at higher risk than others. The same Dutch study found that most heel ulcers were associated with cardiac procedures. They also found head and neck procedures most often associated with sacral ulcers and that use of a semi-Fowler position (elevating both head and lower extremities to 30 degrees) may be beneficial.[130]

Full examination of all areas at risk including the sacrum, heels, ischia, and trochanteric areas with documentation of any skin changes and existing ulcers can help identify postoperative changes. Because the heel has the smallest surface area, this area may be at greatest risk intraoperatively. An ordinary head-pillow placed underneath each heel during the operation and through the time the patient is immobile is likely the most effective pressure-reducing device, followed by a siliconized hollow-fiber–based heel protector.[131] Further studies into a variety of mattress (foam versus gel) and alternative positioning have yet to be evaluated by rigorous randomized controlled trials.

In most cases, if a wound is identified, extra care should be taken to relieve as much pressure from the surface as possible and to provide extra dressings either with 4×4 sterile gauze or circumferential roll gauze to account for extra drainage from the wound during prolonged procedures. After the procedure, the dressings on all chronic wounds should be examined and changed as necessary.

Conclusion

Elderly patients are frequently affected by chronic wounds including pressure, venous, and diabetic foot ulcers. The elderly have the ability to heal from chronic wounds, but they are prone to multiple comorbidities that may slow the rate of healing, and complications from infection pose significant risks to patients who are already fragile. Operative debridement is usually most important to remove the source of infection. Safety of the elderly patient can be optimized by focusing on glycemic control, hydration, and often beta-blockade. If all wounds are treated using the protocol described herein, we postulate that amputations will be decreased, stage IV pressure ulcers nearly eliminated, and morbidity from venous ulcers reduced.

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