Functional Reconstruction of the Diabetic Foot

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ABSTRACT

The diabetic triad of neuropathy, vasculopathy, and foot deformity can be surgically challenging to the reconstructive surgeon. Soft tissue deficits must be closed to protect underlying structures from infection and to provide a stable environment for healing. It is critical to have adequate blood flow and to debride the wound to clean healthy tissue before considering reconstruction. Surgical options commonly used include healing by secondary intention, local flap closure, skin grafts, pedicled flaps, and free tissue transfer. Despite a surgeon's best operative efforts, these strategies may fail perioperatively due to postoperative shear forces created by premature joint motion and/or pressure (either weight bearing or decubitus). In the properly selected patient population, external fixators serve as an indispensable adjunct to wound healing in the Charcot foot by providing temporary but reliable offloading and/or immobilization of joints. Using a team approach is critical to the success of diabetic limb reconstruction.

KEYWORDS: Diabetes, reconstructive surgery, neuropathy, lower-extremity flaps

Progression from the diabetic triad of neuropathy, vasculopathy, and foot deformity to amputation is neither inevitable nor acceptable—contrary to current health-care trends—and may be avoided by maintaining a strict adherence to prevention and by using recent advances in diabetic surgical care. Diabetic foot wounds lead to ~71,000 diabetic limb amputations annually in the United States, a dismal statistic for the 23 million Americans living with diabetes or the 57 million pre-diabetic Americans.^{1,2} Diabetic foot and ankle wounds occur after acute or repetitive trauma to an insensate and biomechanically unstable foot. Infection, inadequate blood flow, ineffective immune system, high serum glucose levels, or poor nutrition need to be adequately addressed or will risk converting an acute wound into a chronic wound. A team approach is essential to achieving a healthy healing wound and includes establishing a

correct diagnosis, ensuring a good local blood supply, optimizing the patient's medical condition, debriding the wound to a clean base, correcting the biomechanical abnormality, and nurturing the wound until it shows signs of healing. Surgical reconstructions of the diabetic foot are then accomplished by simple techniques 90% of the time and by complex flap reconstruction in 10% of cases.

ASSESSING THE DIABETIC FOOT AND PROPER DIAGNOSIS

In diabetics, healing is seriously affected by poorly controlled blood glucose levels. Glucose levels above 300 mg/dl inhibit the white blood cells from effectively destroying invading bacteria and directing the wound-healing effort. Elevated blood glucose concentrations

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have been associated with 4-fold increases in operative complications.^{3,4} High glucose levels glycosylate the red blood cell wall thus stiffening it and leading to an increase in blood viscosity. High glucose levels also contribute to the stiffening of joints and tendons by glycosylating collagen and thereby decreasing their flexibility. The most obvious manifestation of this occurs in the Achilles' tendon resulting in loss of foot dorsiflexion. This then places excessive pressure on the skeletal arch of the foot as well as the plantar forefoot during gait and contributes to Charcot collapse and forefoot ulceration.

More than 60% of diabetic ulcers have insufficient blood flow due to peripheral vascular disease. The atherosclerotic disease is usually manifest below the popliteal artery and involves one or more of the three lower-leg arteries: the anterior tibial artery, the posterior tibial artery, and/or the peroneal artery. Correction depends on the vascular surgeon's ability to correct the defect using endovascular techniques as well as more traditional distal bypasses. The ability to directly revascularize the angiosome where the ulcer is further increases the chances for healing.⁵

During a thorough patient history, the origin (usually traumatic) and age of the wound should be determined. The trauma is usually related to biomechanical abnormalities causing excessive local pressure during gait, changes in shoe wear, penetrating trauma, or excessive heat (hot-water foot baths). It is important to ask what previous topical therapy was applied to the wound because certain topical agents can contribute to the wound's chronicity⁶ (e.g., caustic agents such as hydrogen peroxide, 10% iodine, Dakin's solution, etc.). A careful medical history should reveal possible manifestations of atherosclerotic disease to the heart, nervous system, kidneys, eyes, and lower extremity. The extent of the neuropathy present is then explored: loss of sensation, muscle weakness, loss of ability to sweat, and so forth. Note that a diabetic patient's lack of compliance (as high as 71%) is a major reason for postoperative wound complications.⁷

The wound is assessed carefully by measuring its size and depth and is then photographed. A metallic probe is used to assist in the evaluation of the depth of the wound. If the probe touches bone, there is an 85% chance that osteomyelitis⁸ is present. If tendon is involved, the infection is very likely to have tracked proximally or distally. Note the presence and extent of cellulitis and differentiate from dependent rubor. Antibiotic therapy should be initiated after deep cultures are performed. The blood flow to the area is then evaluated by palpation and/or hand-held Doppler.⁹ If the flow is inadequate, the patient should then be referred to a vascular surgeon who *specializes* in distal revascularizations, as bypass surgery is clinically still superior to endovascular revascularization.¹⁰ After successful bypass surgery, it then takes 4 to 10 days to maximize the

periwound T_{CO_2} tissue oxygen level.¹¹ If angioplasty is performed, then it may take up to 30 days for T_{CO_2} to reach maximal levels. Definitive debridement and aggressive wound care should follow as soon as signs of revascularization occur, such as new granulation, and so forth. Wet gangrene, ascending cellulitis, and necrotizing fasciitis demand immediate debridement of involved tissues before revascularization.^{12,13}

The biomechanical abnormality can result from motor neuropathy and is most often seen in the intrinsic muscles of the foot with resultant hammer toe formation. Skeletal abnormalities can include a prominent metatarsal head, Charcot collapse, and so forth. The elasticity of the Achilles' tendon is affected by diabetes, and open or percutaneous release of the tendon¹⁴ may be indicated to decrease forefoot pressure in the equinovarus foot during gait to allow for the rapid healing of plantar forefoot ulcers (Fig. 1). The release results in a permanent decrease in push-off forces, which has been shown to decrease ulcer recurrence rates by 75% at 7 months and 54% at 25 months out from surgery.^{15,16} Unless correction of the underlying biomechanical

Figure 1 (A) A 29-year-old, obese diabetic patient had bilateral lateral plantar metatarsal ulcers secondary to equinovarus deformity. Both the gastrocnemius and soleus portions of the Achilles' tendon were tight. (B) The left Achilles' tendon was released percutaneously, and the left foot healed in 6 weeks with conservative wound care of the ulcer. The right forefoot ulcer healed similarly within 5 weeks after Achilles' tendon release. Correcting the biomechanical abnormality was all that was required for healing.

abnormality is part of the entire treatment plan, debridement and good wound care may prove futile.

ACHIEVING ADEQUATE DEBRIDEMENT

Debriding a wound of all necrotic tissue, foreign material, and infecting bacteria is essential as these ultimately impede the body's attempt to heal by producing or stimulating the production of proteases, collagenases, and elastases that overwhelm the local wound-healing process.¹⁷ Bacteria produce their own wound-inhibiting enzymes as well and consume many of the scarce local resources (oxygen, nutrition, and building blocks) that are necessary for wound healing. Within this state, bacteria reside in a self-produced matrix of extracellular polymeric substances where they can protect themselves from antibiotics and macrophages by secreting a protective glycocalyx biofilm.¹⁸ Steed et al reviewed data regarding the effect of platelet-derived growth factor on the healing of chronic diabetic wounds¹⁹ and established level 1 evidence that wounds healed almost 3-fold more rapidly when the wound debridement was performed weekly rather than more sporadically.

The principal debriding technique consists of removing the grossly contaminated or necrotic tissue en masse while leaving a maximum amount of viable tissue behind; one should avoid traumatizing techniques such as crushing the skin edges with forceps or clamps, burning tissue with electrocautery, or tying off large clumps of tissue with sutures.²⁰ Surgical tools include a scalpel blade, Mayo scissors, curettes, and rongeurs as well as power tools including a sagittal saw, a power burr, and a hydro surgical debrider (Versa-Jet; Smith & Nephew, Hull, UK). It is critical not to harm the underlying tissue as it will serve as the source for future wound healing. One has to use gentle wound-handling techniques such as sharp dissection instead of cautery resection, skin hooks to retract the tissue, pinpoint use of the cautery to minimize the amount of charred tissue left behind, and so forth.¹⁴ Debridement is performed as often as necessary until the wound is deemed clean and ready for reconstruction.

To be sure that the entire wound has been adequately debrided, three useful adjuncts are helpful. The first is to debride the wound until only normal tissue colors of red, white, and yellow remain at the base of the wound. The second is to first paint the wound base with blue dye using a cotton swab²¹ (Fig. 2). After adequate debridement, there should be no blue dye left in the wound base. Finally, the edges of chronic wounds contain senescent fibroblast cells,²² and an area of 3 to 5 mm of the tissues at the wound edge must be excised during the initial debridement to halt wound chronicity.

In between debridements, topical antibiotics can help reduce the bacterial load: silver-impregnated dressings or silver-sulfadiazine works well for all wounds.

Bactroban (mupirocin, GlaxoSmithKline, Research Triangle Park, NC) is useful for methicillin-resistant *Staphylococcus aureus*, -strength acetic acid or gentamicin ointment for *Pseudomonas* infections, and bacitracin for minimally infected wounds. Once the wound is *clean* and *adequately vascularized*, then it can be covered with a negative-pressure wound therapy (NPWT) device. NPWT applies subatmospheric pressure to a wound via a closed suction mechanism.^{15,23,24} This speeds up the formation of granulation, decreases the bacterial count, and reduces tissue edema. Because this dressing is changed every 48 to 72 hours, the wound is less susceptible to cross-contamination compared with normal bacterial regeneration when the dressing is changed every 8 hours.

ILIZAROV IMMOBILIZATION

A significant portion of wounds will break down or fail to heal in the postoperative period because of repetitive vertical shear and torque forces around a mobile Charcot foot, decubitus pressure, or premature ambulatory pressure in a noncompliant patient. The detrimental effect of motion on wound healing is well established.^{25,26} External fixation has an established role for the treatment of fracture and osteomyelitis.²⁷⁻²⁹ The ability to immobilize the skeletal framework without using internal fixation has led to the salvage of limbs that required amputation in the past.³⁰ The fixator maintains bone and joint alignment through rigid external fixation despite often significant bone resection for osteomyelitis and/or skeletal realignment. It does that without having to resort to internal fixation with plates and screws that are contraindicated in infected wounds. The Ilizarov fixator has been shown to shorten healing time, decrease pin-track infections, and can allow early weight bearing by the addition of a protective plantar foot plate.³¹

In terms of soft tissue reconstruction, the disadvantages associated with the external fixation (Ilizarov more so than external monoplanar fixator) include cost and a cumbersome construct. Applying a fixator carries a relatively high cost ranging from \$2000 to \$10,000. Note that careful preoperative planning is important so that pin placement does not compromise soft tissue reconstructive options, flap dissection, or a microsurgical anastomosis.

Motion along a joint underlying a soft tissue reconstruction often leads to dehiscence or breakdown. We reviewed our experience over a 6-year period and identified 24 consecutive patients in whom external fixation was used solely to protect soft tissue reconstruction and found an overall limb salvage rate of 83% in a multiplanar external fixator group and 73% limb salvage in a monoplanar group.³² Soft tissue reconstructions included primary closure, split-thickness skin grafts, and local flaps. For larger wounds involving a free flap or sural artery flap reconstruction, an Ilizarov frame is ideal because it not only suspends the heel and foot with

Figure 2 (A–C) To remove all traces of a biofilm, a wound may be painted with methylene blue using a cotton swab prior to debridement. Note after debridement, (D) there may still be small crevices remaining that harbor bacteria. Debridement is only finished once no blue dye is left in the wound base.

good skeletal alignment but also protects the flap from any potential trauma. Patient compliance with alternative offloading schemes has proved to be the undoing of many reconstructions and averages more than 20% in our institution. Sagebien et al have recently reported the successful use of external fixation to protect free-flap soft tissue reconstruction of the lower extremity.³³ Similarly, others have previously reported the use of a fixed frame for joint immobilization to support free-flap reconstruction.³⁴ In this complex population with multiple comorbidities, close follow-up and vigilance is critical to prevent wound recurrence.

SOFT TISSUE RECONSTRUCTION

The wound is ready to close when all the abnormal parameters surrounding the wound have been corrected and when all signs of inflammation have disappeared. It can then be allowed to heal by secondary intention, closed by delayed primary closure, skin grafted, or covered with a flap. The wound itself should have no surrounding erythema and minimal induration. Resolution of pain, wrinkled skin edges, fresh granulation tissue, and neo-epithelialization are all signs that a wound is sufficiently ready for reconstruction.

Closure techniques include allowing the wound to heal by secondary intention or by closing it with (1)

delayed primary closure, (2) skin graft, (3) local flap(s), (4) pedicled flap(s), (5) free flap or any combination thereof. If surgical closure is chosen, there should be two setups of instruments in the operating room. The first set of instruments is used to debride the wound. Culturing those instruments postdebridement yields a quantitative culture of up to 10^3 bacteria. Reusing those same instruments during the closure would needlessly recontaminate the freshly debrided wound and increase the risk for postoperative infection. The debrided wound should be cleansed using pulsed lavage and then redraped, the surgeon should change gloves, and a new set of uncontaminated instruments should be used. These steps ensure that the wound remains as clean as possible prior to closure thereby decreasing the risk of subsequent infection and tissue necrosis. Wounds can be allowed to heal by secondary intention by applying daily dressing changes, using wound-healing adjuncts such as growth factors, cultured skin, hyperbaric oxygen, and/or by application of NPWT.

Delayed primary closure is easier to accomplish when the edema and induration of the wound edges has resolved. NPWT devices can be very helpful here. After primary closure, one should always check that relevant arterial pulses have not diminished because the closure was too tight. Adequate soft tissue envelope can also be created by removing bone (e.g., distal fibula in a

tibial-talar fusion) or by converting a large wound to a partial foot amputation and using the resultant soft tissue envelopes for closure (Fig. 3). Technically, deep sutures should be avoided as they could potentiate reinfection. Simple vertical mattress sutures with monofilament are the least likely to facilitate reinfection. Finally, if the closure is too tight, the wound can be partially closed and allowed to granulate in using NPWT.

Skin grafting requires a healthy granulating bed (Fig. 4), which can be achieved by using any one of the following: NPWT, cultured skin derivatives, growth factor, and hyperbaric oxygen. In addition, a healthy neodermis can be built up over inhospitable wounds such as bone or tendon by applying a collagen lattice framework such as Integra (Integra Lifesciences, Plainsboro, NJ) and waiting for it to revascularize before skin grafting. Skin graft recipient bed preparation is optimized by shaving off the superficial granulation tissue prior to skin grafting to remove any residual bacteria that may still reside in the interstices of the granulation buds. Painting the wound base with the blue dye technique described earlier is the key to removing residual biofilm. The wound is then pulse lavaged, and new instruments are used to avoid recontaminating the wound base. The skin graft is meshed or pie crusted to prevent build up of seroma or hematoma that could prevent graft revascularization. Use of NPWT on low continuous suction as a temporary dressing for the first 3 to 5 days will ensure the highest possible skin-graft take rate.

Use of any flap requires an accurate assessment of the blood flow. For local flaps, there should be a Dopplerable perforator close to the base of the flap. For pedicled flaps, the dominant pedicle to the flap should be open. For free flaps, there should be an adequate recipient artery and vein(s). If there is any question, either a duplex scan, magnetic resonance angiogram, or normal angiogram should be obtained.

Local flaps are flaps with unidentified blood supply adjacent to a given defect that are either rotated on a pivot point or advanced forward to cover the defect. They come in various shapes (square, rectangular, rhomboid, semicircular, or bilobed). They usually consist of skin and the underlying fat or skin, fat, and the underlying fascia. It is important to carefully preplan the flap by first accurately determining the size of the defect that needs to be covered after debridement. Only exposed bone, tendon, nerve, or joint need flap coverage, and the rest of the wound can be skin grafted. This combination of limited local flap and skin graft frequently obviates the need of larger pedicled or free flaps. With good preplanning, a local flap can also improve the surgical exposure of the underlying tissue if corrective bone surgery has to be performed.³⁵ The harvesting of an appropriately designed flap allows for better exposure of joints, bone, or tendons so that an extra incision can be avoided.

Equally important, local flaps are a very useful mode of reconstruction when trying to close a wound within the tight confines of an external fixator.

The ratio of length to width is critical for the survival of the tip of the flap.³⁶ Because the blood flow to the skin in the foot and ankle is not as developed as in the face, the length-to-width ratio should not exceed a 1:1 or 1:1.5 ratio. The length-to-width ratio of a flap can be increased when one can Doppler out a cutaneous perforator at the base of the planned flap. To ensure adequate tension-free coverage, the flap should be designed in the area where the tissue is the most mobile and a slightly larger pattern should be used than what would anatomically be necessary. A force of 25 mm Hg causes enough venous congestion for flap necrosis unless the tension is released within 4 hours. If the flap remains pale when inset, it should be rotated back into its bed and delayed for 4 to 7 days while it develops more robust blood supply. During that time, the NPWT can be placed on the open wound to ensure that no edema develops and the tissue edges remain malleable. Finally, we have found that placement of NPWT on the local flaps as well as the skin-grafted recipient site helps ensure flap viability.

The *transposition flap* is the most frequently used flap to cover the malleoli or exposed tibial-talar fusion around an Ilizarov frame (Fig. 5). The flap is designed to cover exposed bone, joint, or tendon only and not the entire defect. The flap is taken from the side that is the most mobile and that has a definable perforator at its base. That means that four possible designs exist: proximally or distally based and medial or lateral to the wound. Sometimes the defect is large enough that a flap from both sides is necessary with one being proximally based and the other distally based. What the flaps do not cover is then skin grafted. NPWT can be placed on the entire construct for 3 to 5 days to ensure good skin graft take and to optimize flap survival.

The *rotation flap* is useful on the plantar aspect of the foot where the flap is elevated off the plantar fascia and rotated into position (Fig. 6). It can also be used over the plantar forefoot, at both malleoli and on the dorsum of the foot. If vascular anatomic considerations dictate, the flap can also include underlying fascia and/or muscle.

The advancement *V-Y flap* is especially useful for defects on the sole of the foot (Fig. 7). It depends on direct underlying perforators to stay alive and hence is dissected out by simply cutting perpendicularly through skin, fat, and fascia without any undermining. Its advancement is limited to 1 to 2 cm so that if the defect is 3 to 4 cm wide, double opposing V-Y flaps should be considered.

PEDICLED FLAPS OF THE LOWER LEG

The lower-leg muscles are poor candidates for pedicled flaps because most of them have segmental

Figure 3 (A, B) This patient had a collapsed Charcot midfoot with osteomyelitis and a methylmethacrylate block in the midfoot. (C) With resection of the midfoot and use of the resultant soft tissue envelopes, he underwent successful Chopart's amputation after closure, Achilles' tenectomy, and (D) immobilization with an external fixator until (E, F) he healed. (G) He has since been able to walk for several years with the aid of a patellar weight-bearing brace.

minor pedicles as their blood supply (type IV), and therefore only a small portion of the muscle can safely be transferred. The distal portion of some of these muscles can be used to cover small defects around the ankle medially, anteriorly, and laterally.³⁷ For small defects, the muscle flap can usually be separated

from its distal tendon to minimize the loss of function.

The extensor hallucis longus muscle (*anterior tibial artery*) can cover small defects that are as distal as 2 cm above the medial malleolus. The extensor digitorum longus muscle and peroneus tertius muscle (*anterior*

Figure 4 A patient presented with a gangrenous Achilles' tendon. (A) The necrotic tendon was sharply debrided to shiny underlying tendon. (B) Hyperbaric oxygen and topical growth factor therapies were started, and (C, D) the tendon began to granulate. (E) The Achilles' tendon was then successfully skin grafted and healed completely after 4 weeks.

tibial artery) are used for small defects as distal as 2.1 cm above the medial malleolus. The peroneus brevis muscle (*peroneal artery*) can be used for small defects as distal as 4 cm above the medial malleolus. The flexor digitorum longus muscle (*posterior tibial artery*) can be used for small defects as distal as 6 cm above the medial malleolus. The soleus muscle (*popliteal, peroneal, and posterior tibial artery*) is the only type 2 muscle in the distal lower leg where the minor distal pedicles can be safely detached and the muscle with its intact proximal major pedicles can be rotated to cover large (10 cm × 8 cm) anterior lower-leg defects as distal as 6.6 cm above the medial malleolus. It can be harvested as a hemisoleus for small defects³⁸ and as an entire soleus for larger defects. All the just-described muscles usually have to be skin grafted for complete coverage. In addition, the ankle has to be immobilized to avoid dehiscence and ensure adequate skin-graft take.

If a larger flap or wider angle of rotation is needed, one of the three major lower-leg arteries with the relevant minor perforators has to be taken with the muscle flap.

The sacrifice of a major artery should only be considered if all three arteries are open and there is excellent retrograde flow. These flaps are usually harvested distally, and therefore the accompanying artery depends on retrograde flow. Because these flaps are larger, the tendon is often taken with the muscle. If so, it is important to tenodesse the distal portion of the severed tendon to the tendon of a similar muscle so that the function is not lost. For example, if the distal extensor hallucis longus muscle is harvested, the extensor hallucis longus tendon distal to the harvest should be tenodesed to the extensor digitorum longus so that the hallux maintains its position during gait. Because the loss of the anterior tibial tendon is so debilitating, the distal muscle should not be harvested unless the ankle has been or is being fused.

Fasciocutaneous flaps are useful for reconstruction of the lower extremity although the donor site usually has to be skin grafted.³⁹ The retrograde peroneal flap (*retrograde peroneal artery*)⁴⁰ is useful for ankle, heel, and proximal dorsal foot defects. Its blood

Figure 5 (A) This patient developed an infected Charcot ankle joint and was debrided and immobilized with an external fixator. (B) The exposed joint was covered with a muscle flap and large transposition flap. (C) The donor site was skin grafted as was (D) the anterior portion of the wound. (E) The transposition flap only needs to be long and wide enough to cover exposed joint or bone, and the rest can be skin grafted.

flow is retrograde and depends on an intact distal peroneal arterial-arterial anastomosis with either or both the anterior tibial artery and/or posterior tibial artery. The dissection is tedious, and it does sacrifice one of the three major arteries of the leg. A similar retrograde anterior tibial artery flap⁴¹ fasciocutaneous flap (*retrograde anterior tibial artery*) has been described for coverage in young patients with traumatic wounds over the same areas. Because the anterior compartment is the only compartment of the leg whose muscle depends solely on the anterior tibial artery, only the lower half of the artery can be safely harvested as a vascular leash. The retrograde sural nerve flap⁴² (*retrograde sural artery*) is a versatile neuro-fasciocutaneous flap that is useful for ankle and heel defects. The sural artery travels with the sural nerve and receives retrograde flow from a peroneal perforator 5 cm above the

lateral malleolus. The artery first courses above the fascia and then goes deep to the fascia at midcalf while the accompanying lesser saphenous vein remains above the fascia. The venous congestion often seen with this flap can be minimized if the pedicle is harvested with 3 cm of tissue on either side of the pedicle and with the overlying skin intact.⁴³ Problems with the venous drainage can be further helped by delaying the flap 4 to 10 days earlier and tying off the proximal lesser saphenous vein and sural artery.^{44,45} The inset of the flap is critical to avoid kinking of the pedicle. The major donor deficit of the flap is the loss of sensibility along the lateral aspect of the foot and a skin-grafted depression at the posterior calf donor site that may pose a problem if the patient later has to undergo a below-the-knee amputation. The supramalleolar flap (*superior cutaneous branch of the anterior perforating branch of the*

Figure 6 (A–D) A double rotation flap can be used to cover a plantar defect from a collapsed Charcot midfoot.

peroneal artery) can be used for lateral malleolar and heel defects as well as for dorsal foot defects.⁴⁶ It can be harvested either with the overlying skin or as a fascial layer that can then be skin grafted. When harvested as a fascial layer only, the donor site can be closed primarily. Small fasciocutaneous flaps based on individual perforators can also be designed over the row of perforators originating from the posterior tibial artery medially and the peroneal artery laterally.⁴⁷ Although the reach and size of the flap is limited, it can be expanded by applying the delay principle. These local flaps have proved to be extremely useful in the closure of soft tissue defects around the ankle in patients in an Ilizarov frame because accessibility to the normal flaps or recipient vessels is always a problem.

PEDICLED FLAPS OF THE FOOT AND ANKLE

The muscle flaps in the foot have a type II vascular pattern with a proximal dominant pedicle, and several distal minor pedicles are useful to cover relatively small local defects.^{48,49} The abductor digiti minimi muscle (*lateral plantar artery*) is very useful for coverage of small mid and posterior lateral defects of the sole of the foot and lateral distal ankle. Its dominant pedicle is just distal and medial to its origin off of the calcaneus, and it has a thin distal muscular bulk.⁵⁰ The abductor hallucis brevis muscle (*medial plantar artery*) is larger and can be used to cover medial defects of the mid and hind foot as well as the medial distal ankle. Its dominant pedicle is at the take-off of the medial plantar artery, and its relatively

Figure 7 (A–D) A “V to Y” flap was used to close an ulcer formed by the collapse of a Charcot midfoot.

thin distal muscular bulk can be difficult to dissect off the flexor hallucis brevis muscle. The extensor digitorum brevis muscle (*lateral tarsal artery*) has disappointingly little bulk but can be used for local defects over the sinus tarsi or lateral calcaneus.⁵¹ The muscle can be rotated in a limited fashion either on its dominant pedicle, the lateral tarsal artery, or in a wider arc if harvested with the entire dorsalis pedis artery. The flexor digitorum brevis muscle (*type 2, lateral plantar artery*) can be used to cover plantar heel defects.⁵² Because the muscle bulk is small, it works best if it is used to fill a defect that can be covered with plantar tissue.

The most versatile fasciocutaneous flap of the foot is the medial plantar flap, which is the ideal tissue for the

coverage of plantar defects.^{53–55} It can also reach medial ankle defects. It can be harvested to a size as large as 6×10 cm, has sensibility, and has a wide arc of rotation if it is taken with the proximal part of the medial plantar artery. It can be harvested on the superficial medial plantar artery (*cutaneous branch of the medial plantar artery*) or on the deep medial plantar artery (*deep branch of the medial plantar artery*). It is preferable to harvest the flap with the superficial branch if the artery can be Dopplered because it will minimally disrupt the existing foot vascular blood supply. However, if it is to be harvested with retrograde flow, the flap should be harvested with the deep branch of the medial plantar artery. The lateral calcaneal flap (*calcaneal branch of the*

Figure 8 (A) This ulcer over a stable collapsed Charcot midfoot was (B) debrided, and a Serratus muscle free flap was used to close the wound. (C, D) The muscle was skin grafted, and the patient was able to use his foot up to 7 years of follow-up.

peroneal artery) is useful for posterior calcaneal and distal Achilles' defects.⁵⁶ Its length can be increased by harvesting it as an "L" shape posterior to and below the lateral malleolus.⁵⁷ It is harvested with the lesser saphenous vein and sural nerve. Because the calcaneal branch of the peroneal artery lies directly on top of periosteum, there is a great danger of damaging or cutting it during harvest. The dorsalis pedis flap (*dorsalis pedis and its continuation, the first dorsal metatarsal artery*) can be either proximally or distally based for coverage of ankle

and dorsal foot defects.⁵⁸ A flap wider than 4 cm usually requires skin grafting on top of extensor tendon paratenon, which leaves the dorsum of the foot with less than ideal coverage. The loss of the dorsalis pedis can pose problems unless the collateral circulation is intact. Because the donor site is vulnerable both from a vascular and a tissue breakdown perspective, this flap is now rarely used. The file of toe flap (*digital artery*) is useful for small forefoot web-space ulcers and distal forefoot problems although the reach of the flap is always less than

expected.⁵⁹ The technique involves removal of the nail bed, phalangeal bones, extensor tendons, flexor tendons, and volar plates while leaving the two digital arteries intact. A variation of this is the very elegant toe island flap where a part of the toe pulp is raised directly over the ipsilateral digital neurovascular bundle.^{60,61} The flap is then elevated with its long vascular leash to cover a distal defect. The leash is buried under the intervening tissue.

FREE TISSUE TRANSFER

Free flaps in the foot and ankle carry the highest failure rate in the microsurgical literature and should be planned carefully. One of the reasons for this is that complications arise when the vascular anastomosis is performed at or near the zone of injury. In addition, in the elderly, diabetics, and chronic renal failure patients, the arteries are often calcified, and special hardened microneedles are often required. Anastomoses should be performed away from the zone of injury. They can be done proximal or distal to the zone of injury providing that the neurovascular bundle is intact. An end to side anastomosis to the recipient artery should be done whenever possible. Two veins should be connected whenever possible to minimize postoperative swelling. Use of an anastomotic device for veins speeds up the procedure.

The choice of which free flap to choose depends in large part on the length of pedicle needed. For long pedicles, the serratus muscle, the vastus lateralis muscle, or the rectus femoris muscles are excellent. Because the patient may eventually rely on crutch walking or use of a wheelchair, we prefer to avoid harvesting the latissimus muscle as it would weaken one side of the patient's upper body. It is important to remember that a longer pedicle can be obtained when the pedicle dissection is continued within the muscle belly. For the dorsum of the foot and ankle, thin fasciocutaneous or cutaneous flaps work best. For the plantar foot, muscle flaps and skin graft seem to hold up better in the long run⁶² (Fig. 8).

COMPLICATIONS ASSOCIATED WITH PEDICLED FLAPS

Once the flap has successfully been raised, it can still fail because of tension at the inseting (see earlier discussion on local flaps, p. 47), inadequate evaluation of the blood flow, twisting of the pedicle, hematoma, and/or infection. Proper flap design and donor-site selection should prevent this from occurring. Hematoma creates pressure on the flap, which can limit venous return and can eventually lead to flap necrosis. If the flap was closed with interrupted sutures, removal of one or two stitches allows for evacuation of the hematoma without risking the disruption of the whole repair.

Infection can damage or destroy a flap by increasing the metabolic demand of the flap so that it

outstrips existing blood supply and by occluding the capillary bed, which short-circuits vascular flow and leads to arterial occlusion. It is therefore important not to plan a reconstruction before all signs of infection are gone.

CONCLUSION

Treating diabetic foot ulcers and gangrene can only be done effectively by using a team approach that at the minimum includes a wound-care team, a vascular surgeon, a foot and ankle surgeon, an infectious disease specialist, an endocrinologist, and a prosthetist. The wound needs to be accurately assessed, debrided, and cultured. The repair is then dictated by how much of the foot remains postdebridement and how the foot can be closed in the most biomechanically stable construct possible. This may involve skeletal manipulation, tendon lengthening, and or partial foot amputations. Soft tissue reconstruction can be as simple as allowing the wound to heal by secondary intention or achieved by use of microsurgical free flaps. More than 90% of the wound can be closed using simple methods from healing by secondary intention to skin grafting. During the reconstruction, special attention should be paid to the local blood flow and to biomechanics to prevent future breakdown.

REFERENCES

1. National Institute of Diabetes, Digestive and Kidney Disease. National Diabetes Statistics 2007. Available at: <http://diabetes.niddk.nih.gov/>. Accessed September 2009
2. American Diabetes Association. American Diabetes Association Statistics 2007. Available at: <http://www.diabetes.org/diabetes-statistics/prevalence.jsp>. Accessed September 2009
3. Barie PS, Eachempati SR. Surgical site infections. *Surg Clin North Am* 2005;85:1115-1135, viii-ix
4. McAlister FA, Man J, Bistriz L, Amad H, Tandon P. Diabetes and coronary artery bypass surgery: an examination of perioperative glycemic control and outcomes. *Diabetes Care* 2003;26:1518-1524
5. Neville RF, Attinger CE, Bulan EJ, Ducic I, Thomassen M, Sidawy AN. Revascularization of a specific angiosome for limb salvage: does the target artery matter? *Ann Vasc Surg* 2009;23:367-373
6. Rodeheaver GT. Wound cleansing, wound irrigation, wound disinfection. In: Krasner D, Kane D, eds. *Chronic Wound Care*. 2nd ed. Wayne, PA: Health Management Publication Inc; 1997:97-108
7. Armstrong DG, Lavery LA, Kimbriel HR, Nixon BP, Boulton AJM. Activity patterns of patients with diabetic foot ulceration: patients with active ulceration may not adhere to a standard pressure off-loading regimen. *Diabetes Care* 2003;26:2595-2597
8. Grayson ML, Gibbons GW, Balogh K, Levin E, Karchmer AW. Probing to bone in infected pedal ulcers. A clinical sign of underlying osteomyelitis in diabetic patients. *JAMA* 1995;273:721-723

9. Attinger C, Cooper P, Blume P, Bulan E. The safest surgical incisions and amputations applying the angiosome principles and using the Doppler to assess the arterial-arterial connections of the foot and ankle. *Foot Ankle Clin* 2001;6: 745–799
10. Adam DJ, Beard JD, Cleveland T, et al. Bypass versus angioplasty in severe ischaemia of the leg (BASIL): multicentre, randomised controlled trial. *Lancet* 2005;366:1925–1934
11. Rhodes GR, King TA. Delayed skin oxygenation following distal tibial revascularization (DTR). Implications for wound healing in late amputations. *Am Surg* 1986;52:519–525
12. Wolff H, Hansson C. Larval therapy—an effective method of ulcer debridement. *Clin Exp Dermatol* 2003;28:134–137
13. Sherman RA, Sherman J, Gilead L, Lipo M, Mumcuoglu KY. Maggot débridement therapy in outpatients. *Arch Phys Med Rehabil* 2001;82:1226–1229
14. Armstrong DG, Stacpoole-Shea S, Nguyen H, Harkless LB. Lengthening of the Achilles tendon in diabetic patients who are at high risk for ulceration of the foot. *J Bone Joint Surg Am* 1999;81:535–538
15. Mueller MJ, Sinacore DR, Hastings MK, Strube MJ, Johnson JE. Effect of Achilles tendon lengthening on neuropathic plantar ulcers. A randomized clinical trial. *J Bone Joint Surg Am* 2003;85:1436–1445
16. Maluf KS, Mueller MJ, Strube MJ, Engsborg JR, Johnson JE. Tendon Achilles lengthening for the treatment of neuropathic ulcers causes a temporary reduction in forefoot pressure associated with changes in plantar flexor power rather than ankle motion during gait. *J Biomech* 2004;37: 897–906
17. Falanga V. Growth factors and chronic wounds: the need to understand the microenvironment. *J Dermatol* 1992;19:667–672
18. Edwards R, Harding KG. Bacteria and wound healing. *Curr Opin Infect Dis* 2004;17:91–96
19. Steed DL, Donohoe D, Webster MW, Lindsley L; Diabetic Ulcer Study Group. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. *J Am Coll Surg* 1996;183:61–64
20. Edgerton MT. *The Art of Surgical Technique*. Baltimore, MD: Williams and Wilkins; 1988
21. Granick MS, Jacoby M, Norruthrun S. Hydrosurgical debridement on chronic wounds: discussion. *Wounds* 2006; 18:35–39
22. Harding KG, Moore K, Phillips TJ. Wound chronicity and fibroblast senescence—implications for treatment. *Int Wound J* 2005;2:364–368
23. Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. *Ann Plast Surg* 1997;38:563–576, discussion 577
24. Armstrong DG, Lavery LA Diabetic Foot Study Consortium. Negative pressure wound therapy after partial diabetic foot amputation: a multicentre, randomised controlled trial. *Lancet* 2005;366:1704–1710
25. Wray JB. Factors in the pathogenesis of nonunion. *J Bone Joint Surg Am* 1965;47:168–73
26. Mooney V, Ferguson AB Jr. The influence of immobilization and motion on the formation of fibrocartilage in the repair granuloma after joint resection in the rabbit. *J Bone Joint Surg Am* 1966;48:1145–1155
27. Dendrinos GK, Kontos S, Lyritis E. Use of the Ilizarov technique for treatment of non-union of the tibia associated with infection. *J Bone Joint Surg Am* 1995;77:835–846
28. Ghoneem HF, Wright JG, Cole WG, Rang M. The Ilizarov method for correction of complex deformities. Psychological and functional outcomes. *J Bone Joint Surg Am* 1996; 78:1480–1485
29. Agarwal S, Agarwal R, Jain UK, Chandra R. Management of soft-tissue problems in leg trauma in conjunction with application of the Ilizarov fixator assembly. *Plast Reconstr Surg* 2001;107:1732–1738
30. Vasconez HC, Nicholls PJ. Management of extremity injuries with external fixator or Ilizarov devices. Cooperative effort between orthopedic and plastic surgeons. *Clin Plast Surg* 1991;18:505–513
31. McKee MD, Yoo D, Schemitsch EH. Health status after Ilizarov reconstruction of post-traumatic lower-limb deformity. *J Bone Joint Surg Br* 1998;80:360–364
32. Parikh PM, Hall MM, Attinger CE, Masden DL, Steinberg JS. External fixation: indications in lower extremity reconstruction and limb salvage. *Plast Reconstr Surg* 2009;123: 160e–161e
33. Sagebien CA, Rodriguez ED, Turen CH. The soft-tissue frame. *Plast Reconstr Surg* 2007;119:2137–2140
34. Buford GA, Trzeciak MA. A novel method for lower-extremity immobilization after free-flap reconstruction of posterior heel defects. *Plast Reconstr Surg* 2003;111:821–824
35. Attinger CE, Cooper P, Blume P, Bulan E. The safest surgical incisions and amputations applying the angiosome principles and using the Doppler to assess the arterial-arterial connections of the foot and ankle. *Foot Ankle Clin* 2001;6:745–799
36. Hallock GG. Distal lower leg local random fasciocutaneous flaps. *Plast Reconstr Surg* 1990;86:304–311
37. Hughes LA, Mahoney JL. Anatomic basis of local muscle flaps in the distal third of the leg. *Plast Reconstr Surg* 1993;92:1144–1154
38. Tobin GR. Hemisoleus and reversed hemi-soleus flaps. *Plast Reconstr Surg* 1987;79:407
39. Cormack GC, Lamberty BGH. *The Arterial Anatomy of Skin Flaps*. 2nd ed. London, UK: Churchill Livingstone; 1994
40. Yoshimura M, Imura S, Shimamura K, Yamauchi S, Nomura S. Peroneal flap for reconstruction in the extremity: preliminary report. *Plast Reconstr Surg* 1984;74:402–409
41. Dang JS, Peng YP, Zhang YX, Lim BH, Pho RW. Reverse anterior tibial flap for reconstruction of foot donor sites. *Plast Reconstr Surg* 2003;116:1604
42. Hasegawa M, Torii S, Katoh H, Esaki S. The distally based superficial sural artery flap. *Plast Reconstr Surg* 1994;93: 1012–1020
43. Baumeister SP, Spierer R, Erdmann D, Sweis R, Levin LS, Germann GK. A realistic complication analysis of 70 sural artery flaps in a multimorbid patient group. *Plast Reconstr Surg* 2003;112:129–140; discussion 141–142
44. Follmar KE, Baccarani A, Baumeister SP, Levin LS, Erdmann D. The distally based sural flap. *Plast Reconstr Surg* 2007;119:138e–148e
45. Erdmann D, Levin S. Delayed reverse sural flap for staged reconstruction of the foot and lower leg. *Plast Reconstr Surg* 2006;118:571–572
46. Masquelet AC, Beveridge J, Romana C, Gerber C. The lateral supramalleolar flap. *Plast Reconstr Surg* 1988;81:74–81
47. Hallock GG. Distal lower leg local random fasciocutaneous flaps. *Plast Reconstr Surg* 1990;86:304–311
48. Ger R. The management of chronic ulcers of the dorsum of the foot by muscle transposition and free skin grafting. *Br J Plast Surg* 1976;29:199–204

49. Attinger CE, Ducic I, Cooper P, Zelen CM. The role of intrinsic muscle flaps of the foot for bone coverage in foot and ankle defects in diabetic and nondiabetic patients. *Plast Reconstr Surg* 2002;110:1047-1054; discussion 1055-1057
50. Attinger CE, Cooper P. Soft tissue reconstruction for calcaneal fractures or osteomyelitis. *Orthop Clin North Am* 2001;32:135-170
51. Leitner DW, Gordon L, Buncke HJ. The extensor digitorum brevis as a muscle island flap. *Plast Reconstr Surg* 1985;76:777-780
52. Hartrampf CR Jr, Scheffan M, Bostwick J III. The flexor digitorum brevis muscle island pedicle flap: a new dimension in heel reconstruction. *Plast Reconstr Surg* 1980;66:264-270
53. Morrison WA, Crabb DM, O'Brien BM, Jenkins A. The instep of the foot as a fasciocutaneous island and as a free flap for heel defects. *Plast Reconstr Surg* 1983;72:56-65
54. Harrison DH, Morgan BDG. The instep island flap to resurface plantar defects. *Br J Plast Surg* 1981;34:315-318
55. Cohen BK, Zabel DD, Newton ED, Catanzariti AR. Soft-tissue reconstruction for recalcitrant diabetic foot wounds. *J Foot Ankle Surg* 1999;38:388-393
56. Grabb WC, Argenta LC. The lateral calcaneal artery skin flap (the lateral calcaneal artery, lesser saphenous vein, and sural nerve skin flap). *Plast Reconstr Surg* 1981;68:723-730
57. Yanai A, Park S, Iwao T, Nakamura N. Reconstruction of a skin defect of the posterior heel by a lateral calcaneal flap. *Plast Reconstr Surg* 1985;75:642-647
58. McCraw JB, Furlow LT Jr. The dorsalis pedis arterialized flap. A clinical study. *Plast Reconstr Surg* 1975;55:177-185
59. Emmett AJ. The filleted toe flap. *Br J Plast Surg* 1976;29:19-21
60. Snyder GB, Edgerton MT Jr. The principle of the island neurovascular flap in the management of ulcerated anesthetic weightbearing areas of the lower extremity. *Plast Reconstr Surg* 1965;36:518-528
61. Kaplan I. Neurovascular island flap in the treatment of trophic ulceration of the heel. *Br J Plast Surg* 1969;22:143-148
62. May JW Jr, Halls MJ, Simon SR. Free microvascular muscle flaps with skin graft reconstruction of extensive defects of the foot: a clinical and gait analysis study. *Plast Reconstr Surg* 1985;75:627-641